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INTERVENTIONAL RADIOLOGY

ORIGINAL ARTICLE

Single-center retrospective comparative study of 40–120 µm versus 100-300 µm trisacryl gelatin microspheres as the main embolic agents for prostatic artery embolization

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PURPOSE

To compare the safety and efficacy of trisacryl gelatin microspheres (TGM) with diameters of 40–120 µm (TGM 40–120) vs. 100–300 µm (TGM 100–300) as the primary embolic agents for prostatic artery (PA) embolization (PAE).

METHODS

This single-center retrospective comparative study included patients with symptomatic benign prostatic hyperplasia treated with PAE using TGM 40–120 (group A) or TGM 100–300 (group B) over a two-year period. Evaluation included International Prostate Symptom Score (IPSS), quality of life score (QoL), prostatic volume (PV), post-void residual (PVR), and percentage of prostatic infarction (pPI) measured by contrast-enhanced ultrasound. Clinical success was defined as post-PAE reduction of IPSS ≤ 15 points with a decrease of at least 25% from the baseline, QoL score ≤ 3 points or a decrease of at least 1 point from baseline, and successful bladder catheter removal (for catheter-dependent patients). Adverse events were recorded and graded using the modified CIRSE classification system for complication reporting.

RESULTS

Most baseline characteristics did not differ significantly between group A (n = 21) and group B (n = 31). At 3 months post-PAE, there were no statistically significant differences between groups in improvement of IPSS (group A: 49.5 ± 26.1%, group B: 51.8 ± 23.3%, $P = 0.403$) or QoL (group A: 54.7 ±3 1.6%, group B: 51.2 ± 14.1%, $P = 0.589$), nor in reduction of PV (group A: 35.5 ± 16.9%, group B: 28.4 ± 11.0%, $P = 0.104$) or PVR (group A: 59.8 ± 23.9%, group B: 60.4 ± 28.5%, $P = 0.934$).

However, pPI (calculated 1–4 days post-PAE) was significantly higher in group A (48.3 ± 16.2% vs. 30.3 ± 11.9%, $P = 0.007$). Mid-term PV reduction was also significantly greater in group A (31.4 ± 21.4% vs. 12.2 ± 10.2%, $P = 0.030$).

There were more complications in group A (7/21 vs. 4/31, $P = 0.095$). Three complications in group A were grade 3a events (bladder wall ischemia, n = 2; persistent severe anal and perineal pain requiring hospitalization and opioids, n = 1). No grade 3 events were observed in group B ($P = 0.060$).

Clinical success rates at 6, 12, 24, and 36 months post-PAE were 76.2%, 76.2%, 71.1%, and 71.1%, respectively, for group A, and 87.1%, 83.9%, 79.9%, and 79.9%, respectively, for group B ($P = 0.451$).

CONCLUSION

Compared with TGM 100–300, PAE with TGM 40–120 appears to provide similar clinical benefit but results in significantly higher pPI and greater mid-term prostate shrinkage. Ischemic complications may be more frequent and more severe with the smaller microspheres.

CLINICAL SIGNIFICANCE

This is the first systematic comparison between the smallest and the second smallest commercially available TGM sizes in the clinical context of PAE. These findings may help guide ongoing research to identify the most appropriate embolic material for PAE.

KEYWORDS

Prostatic artery embolization, trisacryl gelatin microspheres, prostatic infarction, complications

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Although liquid embolic agents have recently emerged as a promising material for prostatic artery embolization (PAE) in symptomatic benign prostatic hyperplasia (BPH),[1] the vast majority of procedures are still performed with polyvinyl alcohol (PVA) particles or microspheres.[2] Among the latter, trisacryl gelatin microspheres (TGM) with diameters of 100–300 µm and 300–500 µm (Embosphere, Merit Medical Systems Inc., South Jordan, UT, USA), or a combination of these sizes, have been widely used for PAE worldwide since the early applications of this procedure,[3] and several studies have demonstrated their satisfactory safety and efficacy profiles.[4-6]

Currently, the smallest commercially available diameter of TGM is 40–120 µm (TGM 40–120), and experience with this very small TGM in PAE remains limited.[7] Experimental studies have demonstrated that TGM 40–120 can cause complete occlusion of arteries with a mean diameter as small as 137.8 µm,[8] and that TGM can cause significantly larger infarction than other types of microspheres.[9] A human cadaveric study[10] found that arteries surrounding the hyperplasic adenomatous prostatic nodules (perinodal arteries) had a mean diameter of 150 µm (range: 59–266 µm), whereas arteries entering the prostatic nodules (terminal, intranodal arteries) had a mean diameter of 56 µm (range: 24–104 µm).

It may be hypothesized that TGM 40–120 could maximize the efficacy of PAE through more distal penetration and tighter packing within perinodal and even some intranodal arteries, resulting in more complete and irreversible ischemia. Notably, ischemic necrosis of hyperplastic prostatic tissue induced by embolization is considered one of the proposed mechanisms of therapeutic action of PAE.[11] However, in addition to these potential benefits, the intense ischemia caused by very small microspheres may also be associated with a higher prevalence and greater severity of ischemic complications compared with larger microspheres.

Main points

- Microspheres measuring 40–120 µm represent the smallest commercially available trisacryl gelatin embolic microspheres (Embospheres), and their use in prostatic artery embolization remains limited.
- Compared with the second-smallest size (100–300 µm), 40–120 µm trisacryl gelatin microspheres cause significantly more extensive prostatic necrosis and greater mid-term prostate shrinkage; however, these differences are not clearly reflected in the clinical efficacy of PAE.
- Microspheres measuring 40–120 may cause more frequent and more severe ischemic complications than larger microspheres.

To evaluate these hypotheses regarding the safety and efficacy of PAE using the smallest commercially available TGM, a retrospective comparative study was conducted in patients with BPH who underwent PAE with TGM 40–120 and another group treated with the more commonly used TGM 100–300.

Methods

Patients

A retrospective review was conducted of patients treated with PAE for symptomatic BPH at a single tertiary center during a 2-year period (April 2021–April 2023). Inclusion criteria were largely consistent with CIRSE standards of practice for PAE:[12] moderate-to-severe lower urinary tract symptoms [International Prostate Symptom Score (IPSS) ≥ 8]; quality of life (QoL) ≥ 3; failure of medical treatment (at least 6 months of administration of 5α-reductase inhibitors or selective α1 blockers) or urinary retention managed with an indwelling bladder catheter (IBC), with at least three failed catheter removal attempts prior to PAE; and a prostate volume > 40 mL measured by ultrasound.

Exclusion criteria were previous surgical or interventional prostate treatment, urinary tract infection, prostate or bladder cancer, neurogenic bladder, large (> 2 cm) bladder diverticula or bladder stones, contraindications to angiographic procedures, and vascular pathologies that precluded safe arterial access. A minimum follow-up period of three months was also required.

All patients had been informed in detail regarding the procedure and potential benefits and risks (including complications specifically associated with very small microspheres) and had provided their written informed consent. The study was approved by the Scientific and Ethics Committee of General Hospital Tzanio (decision number:17; date: October 10th, 2024). Patients treated with TGM 40–120 comprised "group A," and those treated with TGM 100–300 comprised "group B". Figure 1 shows how the two study groups were formed.

Baseline evaluation and treatment planning

All patients underwent clinical evaluation by the referring urologist prior to PAE, and the IPSS and QoL scores were recorded. A transabdominal ultrasound was performed a few days before PAE to measure baseline prostatic volume (PV) and post-void residual (PVR). Contrast-enhanced ultrasonography (CEUS) was performed at the same session to assess prostatic parenchymal enhancement. Computed tomographic angiography (CTA) of the pelvic arteries was performed 5–10 days prior to PAE for vascular planning in all patients.

PAE procedure

Thirty minutes before the procedure, the patients received an intravenous antibiotic (cefradine, 2 g), analgesic (parecoxib, 40 mg), and gastric protection. Vascular access was obtained via the right or left common femoral artery using the Seldinger technique, and a 5 French (Fr) reverse-curve angiographic catheter (usually the MOT catheter, Merit Medical Systems Inc., South Jordan, UT, USA) was introduced into the ipsilateral internal iliac artery. An ipsilateral oblique angiogram was then performed to clearly demonstrate the origin of the PA.

The PA was catheterized with a 1.98 Fr microcatheter (PARKWAY SOFT, Asahi Intecc Co.) and a double-angled 0.016" microguidewire (ASAHI Meister). If catheterization failed, the microcatheter was exchanged for a steerable one (2.4 Fr, SwiftNINJA, Merit Medical Systems). Successful catheterization was confirmed with a superselective angiogram using manual contrast injection through the microcatheter.

Embolization commenced with the microcatheter tip positioned in the extraprostatic segment of the PA. Syringes containing 2.0 ml of microspheres were used for both TGM sizes. Microspheres were diluted according to the manufacturer's instructions, with a total volume of 22 mL of diluted microspheres prepared per syringe. Slow, flow-directed manual injection was performed under fluoroscopy until complete flow stasis was observed in the targeted PA.

Distal advancement of the microcatheter and additional embolization of intraprostatic branches were then attempted according to the "PErFecTED" technique.[13] Finally, the microcatheter was withdrawn to the proximal PA, and a minimal amount of diluted PVA particles (Bearing, 150–250 µm, Merit Medical) was injected to enhance proximal flow stasis in all the patients. The contralateral PA was then embolized after a crossover maneuver.

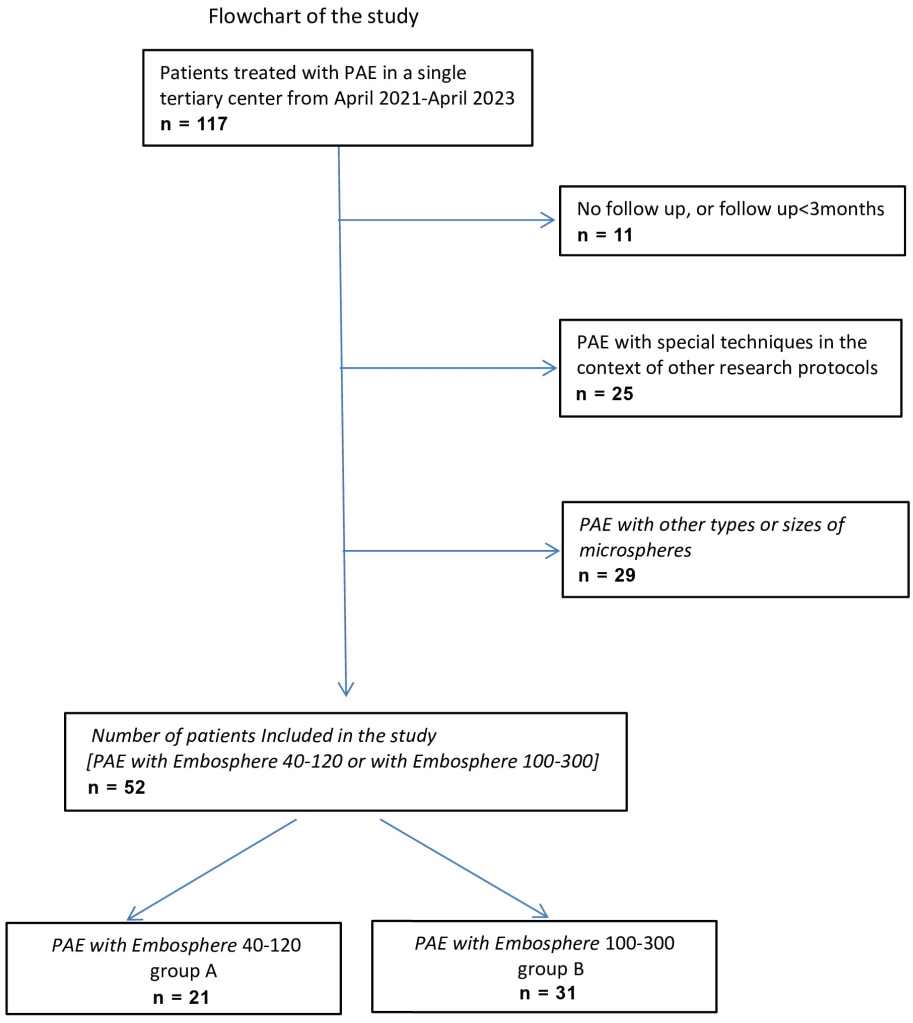

Figure 1. Flowchart of the study population. PAE, prostatic artery embolization.

All PAE procedures were performed using a flat-panel angiographic unit (Axiom Artis Zee, Siemens Healthineers, Erlangen, Germany) by the same operator, who had 6 years of prior experience performing PAE with TGM. Intraprocedural cone-beam computed tomography (CBCT), unenhanced ultrasound, and CEUS were available but used only when deemed necessary by the operator for detailed vascular assessment or monitoring of embolic agent distribution.

Clinical assessment and diagnosis of clinical success and of adverse events

Patients were clinically evaluated within the first 4 days after PAE, at 1 and 3 months post-PAE, and then every 3 months; IPSS and QoL scores were updated at each visit. After the first 6 months, some clinical visits could be replaced by telephone or online communication if all the required information could be consistently and reliably provided by the patient.

Clinical success post-PAE was diagnosed if all the following criteria were met: (a) IPSS ≤ 15 points with a decrease of at least 25% from baseline; (b) QoL score ≤ 3 points or a decrease of at least 1 point from baseline; (c) no need for additional medical or surgical treatment. For patients with urinary retention and IBC, clinical success also required successful and permanent catheter removal with spontaneous micturition and PVR < 100 mL.

Complications were recorded and graded according to the modified CIRSE classification system.[14] Post-embolization syndrome (PES) was defined as dysuria, urgency, urinary frequency, nausea, fever (without evidence of sepsis), pelvic or prostatic pain, or any combination of these symptoms within the first 7 days following PAE.[12,15] Because PES was considered an adverse event, rather than a complication, it was recorded separately. Rectal bleeding, hematuria, and hemospermia were also considered as adverse events (not true complications) and components of PES if mild and self-limiting.[12,14]

Intra- or post-procedural pain was not systematically recorded. However, cases of significant pain affecting technical or clinical outcomes, or pain associated with a complication, were documented, and pain intensity in these cases was assessed using the visual analogue scale (VAS).

Imaging follow-up

Unenhanced ultrasound was performed at the first post-procedure visit (within 4 days) and at 3 months post-PAE. PV was calculated using the ellipsoid formula based on transabdominal ultrasound measurements, and prostate shrinkage was determined by comparing baseline PV with PV at 3 months post-PAE.

CEUS was also performed at the first post-procedure visit to assess prostatic infarcts. Infarcts were defined as newly appear-

ing non-enhancing areas within the prostatic parenchyma. The volume of each infarct was calculated using the ellipsoid formula, and the volumes of all infarcts within the treated prostate were summed. The percentage of prostatic infarction (pPI) was calculated by dividing total infarct volume by PV, consistent with previous work.[16]

Additional imaging was not routinely scheduled during follow-up. However, if PV measurements were obtained during later visits (more than 3 months post-PAE), they were recorded. Imaging performed for other reasons (e.g., pelvic CT for complication assessment or unrelated imaging months or years after PAE) was also considered if they yielded findings relevant to the purposes of this work.

Statistical analysis

Descriptive statistics were used to present baseline characteristics and imaging and clinical outcomes. The Shapiro–Wilk test was used to assess the normality of the data distribution. Comparisons between group A and group B were performed for baseline and outcome parameters. Depending on parameter type and distribution, appropriate statistical tests were applied (Fisher's exact test, Welch's t-test, Mann–Whitney U test).

The Kaplan–Meier method was used to calculate the clinical success rates of PAE over time, and the log-rank test was used to evaluate differences between the groups. Statistical significance was defined as $P < 0.05$. Statistical analyses were performed using the Statistical Package for Social Sciences (Version 26.0, SPSS Inc., Chicago, Illinois, USA).

Results

General and clinical outcomes

Fifty-two patients were included in the study: 21 in group A and 31 in group B. Apart from age, baseline clinical features were comparable between the groups (Table 1). Most anatomic features did not differ significantly between the two groups, either; however, there were significantly more patients with mild prostatic hyperplasia (PV ≤ 50 mL) in group A (Supplementary Tables 1 and 2). Most technical aspects of PAE were also comparable between the two groups (Supplementary material Table 3). Notably, a significantly larger volume of diluted TGM suspension was injected in group A compared with group B (25.1 ± 6.9 mL vs. 20.0 ± 5.7 mL, $P = 0.008$). The volume of diluted PVA injected at the end of embolization of each pelvic side did not exceed 2 mL in any case.

Follow-up ranged from 3 to 48 months [mean ± standard deviation (SD): 23.4 ± 15.5 months] for group A and from 3 to 45 months (mean ± SD: 23.3 ± 13.4 months) for group B. At 3 months post-PAE, there were no statistically significant differences in early clinical improvement (expressed by reduction of IPSS and QoL) between the two groups (Table 2).

Clinical success rates at 6, 12, 24, and 36 months post-PAE were: 76.2%, 76.2%, 71.1%, and 71.1%, respectively, for group A and 87.1%, 83.9%, 79.9%, and 79.9%, respectively, for group B; these differences were not statistically significant ($P = 0.451$, Figure 2). Among patients with mild prostatic hyperplasia (n = 5, all in group A), three experienced early clinical failure; in two of these, further investigation revealed pathologies other than BPH as potential causes of LUTS. The remaining two patients with mild prostatic hyperplasia reported only moderate symptomatic improvement, with IPSS reductions of 25% and 27%, respectively.

Table 1. Demographic and clinical features of the two patient groups at baseline

Variable	Group A (n = 21)	Group B (n = 31)	Significance of differences (*P*)
Age (years, mean ± SD)	75.3 ± 7.0	68.9 ± 10.1	0.024
PV (mL, mean ± SD)	95.3 ± 55.8	107.6 ± 43.4	0.472
PVR	184 ± 162	118 ± 84	0.097
IPSS	26.2 ± 5.8	26.5 ± 4.0	0.984
QoL	4.2 ± 0.8	4.0 ± 0.6	0.284
Indication for PAE (proportion of patients)			
-Moderate LUTS	2/21	3/31	0.984
-Severe LUTS	10/21	19/31	0.332
-IBC	9/21	9/31	0.303

For moderate LUTS, IPSS = 8–19; for severe LUTS, IPSS = 20–35. Welch's t-test was used for comparisons of age, PV, and PVR. Mann–Whitney U test was used for comparisons of IPSS and QoL. Fisher's exact test was used for comparisons of proportions of patients with moderate/severe LUTS and IBC.
SD, standard deviation; PV, prostate volume; PVR, post-void residual; IPSS, International Prostate Symptom Score; QoL, quality of life; PAE, prostatic artery embolization; LUTS, lower urinary tract symptoms; IBC, indwelling bladder catheter.

Table 2. Comparison of short-term outcome parameters for the two patient groups

Variable	Group A (n = 21)	Group B (n = 31)	Significance of differences (*P*)
pPI[a] (mean ± SD, %)	48.3 ± 16.2	30.3 ± 11.9	0.007
PV reduction[b] (mean ± SD, %)	35.5 ± 16.9	28.4 ± 11.0	0.104
PVR reduction[b] (mean ± SD, %)	59.8 ± 23.9	60.4 ± 28.5	0.934
IPSS reduction[b] (mean ± SD, %)	49.5 ± 26.1	51.8 ± 23.3	0.403
QoL improvement[b] (mean ± SD, %)	54.7 ± 31.6	51.2 ± 14.1	0.589

[a]Calculated 1–4 days post-PAE; [b]Calculated 3 months post-PAE and compared with respective baseline measurements.
All values for outcome parameters represent percentages (%), not absolute values. Welch's t-test was used for comparisons.
pPI, percentage of prostatic infarction; SD, standard deviation; PV, prostate volume; PVR, post-void residual; IPSS, International Prostate Symptom Score; QoL, quality of life.

Imaging outcomes

Early CEUS (1–4 days post-PAE) revealed almost 60% greater pPI in group A. At 3 months post-PAE, prostate shrinkage was also greater in group A, although the difference did not reach statistical significance. Reduction in PVR was similar between the two groups. Detailed comparisons of the early outcomes are provided in Table 2.

Mid-term PV measurements (12–48 months post-PAE) were available for 9 patients in group A (these 9 patients formed subgroup A) and for 11 patients in group B (subgroup B). In this subset, prostate shrinkage was significantly greater in subgroup A (Table 3, Figure 3). Almost complete prostate regrowth (0%–5% difference compared with baseline PV) occurred in 1 of 9 patients in subgroup A and 4 of 11 patients in subgroup B. Interestingly, two of the four subgroup B patients with almost complete prostate regrowth reported sustained symptomatic improvement. Sustained symptomatic improvement was also reported by all patients with documented prostate shrinkage on mid-term PV measurements.

Adverse events

Post-procedure PES was by far the most common adverse event in both groups (Table 4).

Two patients in group A experienced significant intraprocedural pain (VAS scores 8 and 7), occurring approximately 5 minutes after completion of embolization on one pelvic side. In the first patient, the pain (VAS 8) persisted despite additional intravenous analgesia and intra-arterial lidocaine and, combined with patient anxiety and discomfort, led to early termination of the procedure before embolization of the contralateral side. According to the updated CIRSE classification system,[14] this event was recorded as a grade 1b complication.

Additionally, three grade 2 complications occurred in group A (acute urinary retention) and four in group B (acute urinary retention, n = 3; small ischemic penile ulcers with moderate pain, n = 1). All grade 2 complications resolved completely with conservative treatment (and/or Foley catheter placement for less than 7 days).

Three grade 3a complications occurred in group A. Two patients (both with type I PA origin, or "common vesicoprostatic trunk") experienced hematuria and moderate pain (VAS 5) 2–4 days post-PAE. In both patients, CT revealed bladder wall ischemia with no signs of bladder perforation, and cystoscopy was performed to assess the extent of injury and remove superficial necrotic bladder wall tissue. In one of these patients, a necrotic portion of the protruding median lobe was also cystoscopically removed (Figure 4). Both patients recovered with no permanent sequelae.

A third patient in group A (with a history of sigmoidectomy for cancer 4 years earlier) developed severe anal and perineal pain (VAS 8) beginning the day after PAE. The patient required hospitalization for 3 days, and the pain persisted for several months despite oral opioid analgesia. CT performed 2 days post-PAE indicated inadvertent embolization

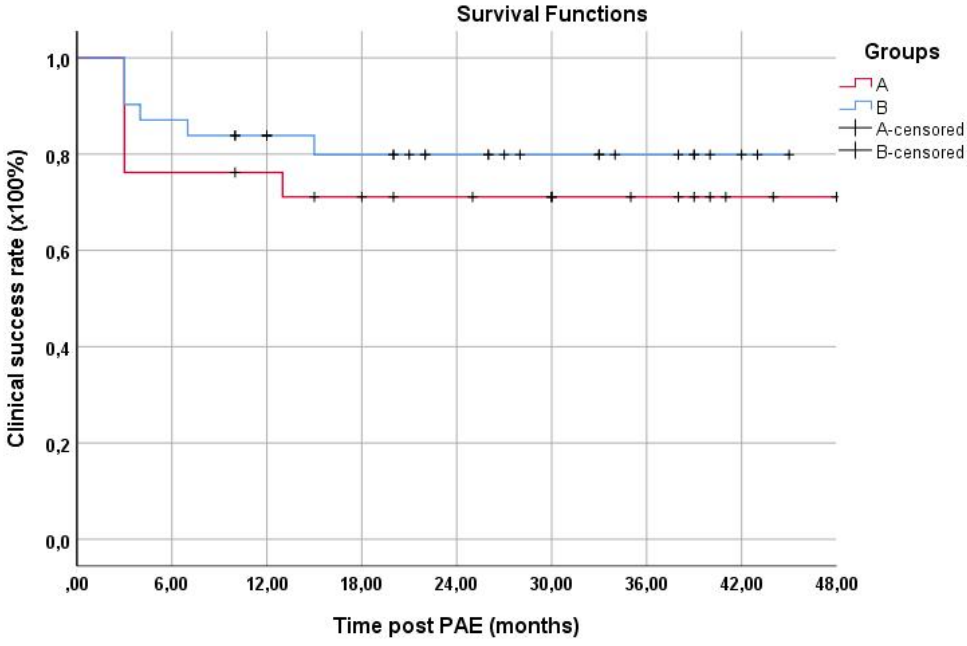

Figure 2. Kaplan–Meier curves showing no statistically significant difference ($P = 0.451$) in the clinical success rates between group A (PAE with TGM 40–120) and group B (PAE with TGM 100-300). PAE, prostatic artery embolization; TGM, trisacryl gelatin microspheres.

Table 3. Comparison of mid-term outcome parameters for the two patient subgroups

Variable	Subgroup A (n = 9)	Subgroup B (n = 11)	Significance of differences (*P*)
Time post-PAE (min/max/mean/SD, months)	12/48/34/11.6	12/36/22/9.2	-
PV reduction (mean ± SD, %)	31.4 ± 21.4	12.2 ± 10.2	0.030
IPSS reduction (mean ± SD, %)	47.1 ± 24.6	37.3 ± 20.7	0.414

Welch's t-test was used for comparisons.
PAE, prostatic artery embolization; SD, standard deviation; PV, prostate volume; IPSS, International Prostate Symptom; min/max, minimum/maximum.

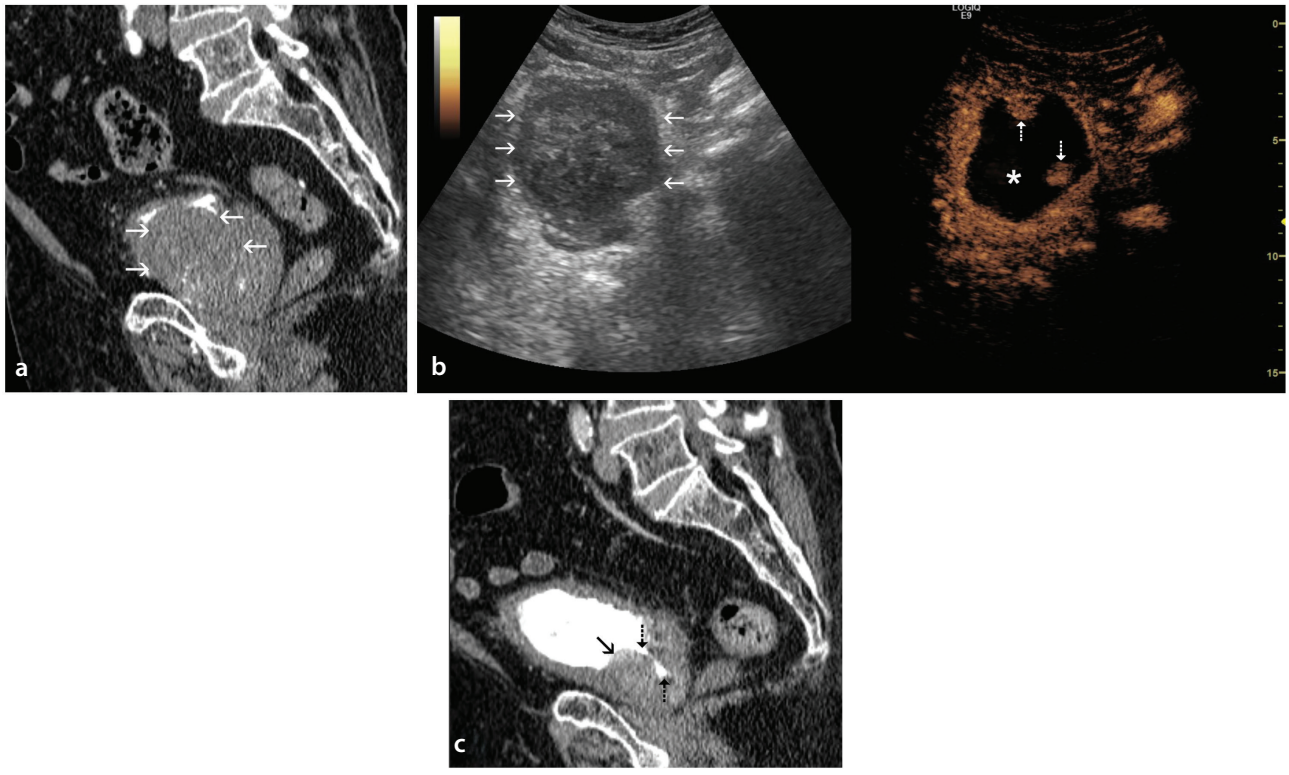

Figure 3. Representative images from a clinically successful case of prostatic artery embolization (PAE) with TGM 40–120. Sagittal reformatted planning computed tomography (CT) image prior to PAE; **(a)** shows prostatic enlargement and massive intravesical prostatic protrusion (arrows). Axial dual sonographic image obtained 2 days post-PAE **(b)**; with reference gray-scale image on the left showing the bulky intraprostatic protrusion (arrows) and corresponding contrast-enhanced ultrasonography image on the right showing extensive prostatic infarction (*) and minimal residual enhancing tissue (dotted arrows). Sagittal reformatted CT image **(c)**; obtained 12 months post-PAE for an unrelated reason, shows marked prostate shrinkage (arrow) and relief of pressure on the prostatic urethra (dotted arrows).

TGM, trisacryl gelatin microspheres.

Table 4. Comparison of adverse events for the two patient groups

Adverse event	Group A (n = 21)	Group B (n = 31)	Significance of differences (*P*)
Post-embolization syndrome	10	11	0.404
Severe intraprocedural pain	2	0	0.158
Mild rectal bleeding*	1	0	0.404
Mild hemospermia*	0	1	1.000
Complication			
Grade 1b	1	0	0.404
Grade 2	3	4	1.000
Grade 3a	3	0	0.060
Total number of complications	7	4	0.095

Fisher's exact test was used for comparisons. *Not considered true complications, despite specific reporting.

of the left middle rectal artery. This vessel had not been identified on planning CTA or DSA, and intraprocedural CBCT had not been performed. No rectal wall thickening or dehiscence was observed on CT post-PAE, and colonoscopy was not performed. Despite favorable long-term urologic outcomes and substantial, durable prostate debulking, this case was registered as a clinical failure because the pain worsened the patient's QoL for at least 6 months post-PAE (from 5 to 6). An additional imaging finding in this case was the appearance of linear hyperdensities with vascular distribution on follow-up CT (Figure 5).

Discussion

To the best of the authors' knowledge, this is the first systematic comparison between the smallest and the second smallest sizes of TGM (diameters 40–120 µm and 100–300 µm, respectively) as the main embolic agents for PAE. Compared with other TGM sizes, TGM 40–120 is characterized not only by a smaller average diameter but also by narrower size calibration; consequently, this very small TGM is capable of deeper penetration into the prostatic microvasculature and of inducing more severe and irreversible tissue ischemia.[8] These characteristics of TGM 40–120 are reflected in the significantly higher pPI (almost 60%) observed in group A in the first days post-PAE in this study. Notably, previous work using a similar PAE technique but microspheres ≥ 100 µm[16] and the same method for measuring prostatic infarction, demonstrated

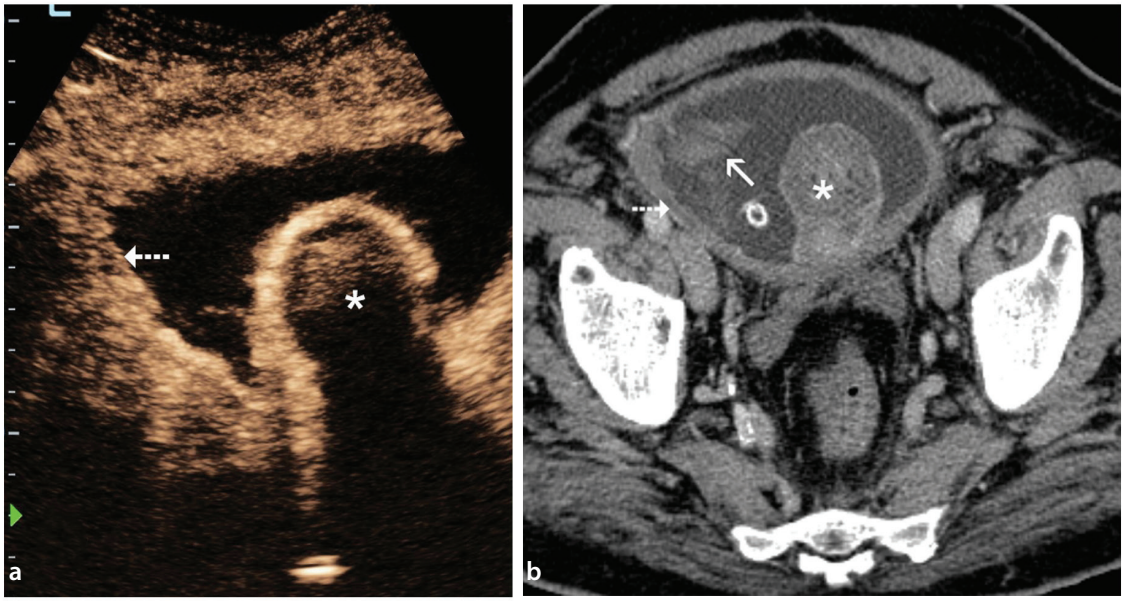

Figure 4. Representative images from a complicated case of prostatic artery embolization (PAE) with TGM 40–120. Three days post-PAE, axial contrast-enhanced ultrasonography image **(a)**; shows extensive infarction (*) of a large intravesical prostatic protrusion. A small area of reduced enhancement is suspected in the right lateral bladder wall (dotted arrow). Axial contrast-enhanced computed tomograpgy image **(b)**; performed immediately thereafter, also shows prostatic infarction (*), along with hypoenhancement (dotted arrow) and irregularity of the right lateral bladder wall. An irregularly shaped intraluminal mass (arrow) corresponds to sloughed necrotic superficial layers of the bladder wall, as confirmed by cystoscopy (images not shown).

TGM, trisacryl gelatin microspheres.

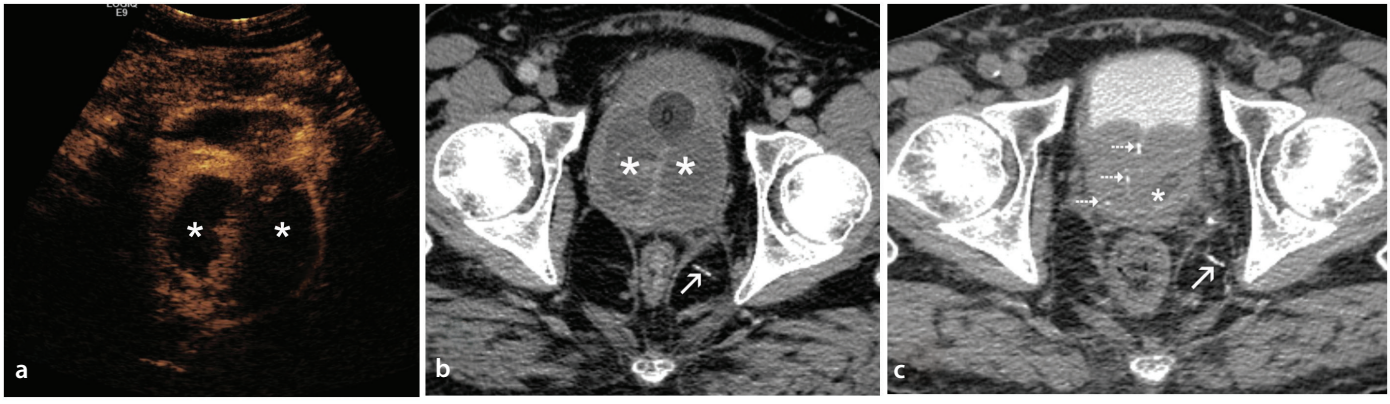

Figure 5. Representative images from a complicated case of prostatic artery embolization (PAE) with trisacryl gelatin microspheres 40–120 (severe persistent anal and perineal pain). Two days post-PAE, contrast-enhanced ultrasonography image **(a)** shows extensive infarction (*) of both prostatic lobes. Axial contrast-enhanced computed tomograpgy (CT) image obtained the same day **(b)** confirms prostatic infarction (*) and additionally shows linear hyperdensity (indicating the presence of the embolic mixture) along the expected trajectory of the left middle rectal artery (arrow) in the ischiorectal fossa. Four years post-PAE, axial contrast-enhanced CT image **(c)** shows significant prostate shrinkage with minimal residual prostatic infarction (*) and persistent linear hyperdensity in the left ischiorectal fossa (arrow). Similar, smaller hyperdensities are noted at the expected locations of intraprostatic arterial branches (dotted arrows). These findings were not present prior to PAE (baseline images not shown).

significantly lower pPI (25.1% in the previous study vs. 48.3% with TGM 40–120 in this study). By contrast, two other PAE studies, one using small (50 and 100 μm) PVA particles[17] and the other using a combination of temperature-sensitive liquid embolic agent with PVA,[18] reported even higher mean proportions of prostatic ischemia (70% and 62%, respectively). Conversely, larger spherical or non-spherical PVA particles appear to cause less pronounced prostatic ischemia, in the range of 10%–11%.[19]

Laboratory investigation has shown that the small size and narrow calibration of microspheres promote not only more distal penetration but also tighter intraluminal packing and more complete vascular occlusion.[20] This may explain the significantly larger volume of microsphere–contrast medium suspension required for PAE with the smaller microspheres (25.1 mL per procedure in group A vs. 20.0 mL per procedure in group B, despite comparable prostate size between groups). Of note, a previous study of TGM 100–300[4] reported a mean suspension volume of 11.9 mL per procedure (mean PV of 63.8 mL), which is roughly equivalent to the 20.0 mL per procedure observed in the present study (mean PV of 107.6 mL in group B). No corresponding published data are available for TGM 40–120.

Prostatic infarction detected early post-PAE is considered a good predictor of subsequent prostate debulking.[11,21] Therefore, it is not surprising that TGM 40–120 was associated not only with larger prostatic infarctions

but also with greater prostate shrinkage. Importantly, this difference became more pronounced and statistically significant at mid-term evaluation, suggesting a reduced risk of prostate regrowth/revascularization with the smaller microspheres. Prostate regrowth between 3 and 12 months post-PAE with TGM 100–300 has been reported previously, but in that study,[4] the comparison was made with larger (300–500 µm) microspheres of the same type. In contrast to the present findings, that study[4] suggested that larger TGMs (not smaller ones) provide more durable PV reduction. The reasons for this discrepancy are unclear. A "rebound" increase in PV (147.9 mL, or a 3.1% increase compared with baseline) has also been reported[22] after a mean of 25.7 months post-PAE with PVA 100 and 200 µm. Compared with these studies,[4,22] the sustained prostate shrinkage observed with TGM 40–120 in the present study appears superior; however, these findings were observed in a small subgroup of patients.

It is also unclear whether the additional PVA particles (used at the end of embolization of each PA in the present study) contributed to the prevention of prostate regrowth. The amount of injected PVA was minimal and was used in both groups. Although PVA 150–250 may not be the ideal embolic material for PAE, its use for complementary proximal embolization was dictated by availability and low cost. Larger PVA particles were selected rather than the smaller TGMs already used because reflux from this proximal position, particularly after occlusion of smaller peripheral branches, is high, and the risk of ischemic complications would be much higher if small TGMs (either 40–120 or 100–300) were to reflux.

Although TGM 40–120 appeared superior in terms of prostatic infarction and mid-term prostate shrinkage, no significant differences were observed in clinical efficacy (as reflected by IPSS and QoL improvement) between the two patient groups. This contrasts with the finding of the only other study with TGM 40–120,[7] which (although limited to a small number of catheter-dependent patients), reported higher clinical success rates with the smaller microspheres (70.6% vs. 42.9%). Prostatic ischemia and its consequences are probably not the only therapeutic mechanisms of PAE. For example, symptomatic improvement has been observed post-PAE even in the absence of detectable prostatic infarction on contrast-enhanced magnetic resonance.[21,23] The role of prostate shrinkage in symptomatic improvement post-PAE is also debated, as some studies have failed to demonstrate a consistent relationship between PV reduction and IPSS improvement. Indeed, patients with minimal prostate shrinkage post-PAE may still experience substantial symptomatic relief.[24] Similarly, two patients in group B in the present study experienced sustained improvement in IPSS and QoL indices despite prostate regrowth. Conversely, none of the patients with persistent prostate shrinkage at mid-term follow-up in this study experienced symptomatic recurrence. This may represent a potential advantage of TGM 40–120, which requires validation with larger studies.

Regarding baseline characteristics, patients in group A were significantly older, and mild prostatic enlargement was more common in this group. However, evidence regarding the clinical predictive value of these features is contradictory,[11] and the authors believe their overall impact on clinical outcomes in this study was limited.

The adverse events and complications associated particularly with the smaller microspheres in this study merit attention. Severe intraprocedural pain during PAE with larger microspheres has rarely been reported;[25] in fact, the relatively painless and minimally invasive profile of PAE is considered a major advantage. Nevertheless, two patients in group A experienced severe pain almost immediately after injection of TGM 40–120, likely due to rapid and intense ischemia. In one of these patients, pain directly affected the technical outcome, causing early termination of the procedure and precluding bilateral embolization.

The most severe (grade 3) complications in this study also occurred exclusively in group A, in contrast to the previous small study with TGM 40–120,[7] where no such events were reported. The two cases of bladder wall ischemia can be explained by the specific type of PA origin (common vesicoprostatic trunk) and reflux of the microspheres into the superior vesical artery. This type of PA origin is the most common in many populations.[26] Consistent with previous reports,[27,28] recovery was complete, and no permanent sequelae occurred, although additional care and cystoscopic intervention were required.

The incident of severe anal and perineal pain post-PAE was associated with inadvertent occlusion of the middle rectal artery, as demonstrated on follow-up CT. This type of non-target embolization occurs occasionally during PAE, and its sequelae are usually mild and self-limiting; however, prolonged perineal pain lasting 3 months post-PAE has been reported.[28] The severity and duration of pain in the present case may at least partially be attributed to the small size of the TGM used. It is unclear whether the patient's prior sigmoidectomy (and any associated arterial ligations) contributed to the severity of the complication by reducing collateral circulation. An interesting (not previously reported) imaging finding on follow-up CT in the same patient was the appearance of linear hyperdensities with vascular distribution. These likely represent calcifications, either dystrophic or due to calcification of the TGM themselves, with a mechanism similar to that proposed for uterine fibroids after successful embolization.[29] Finally, the failure of both CTA and DSA to identify the middle rectal artery in this patient highlights the potential role of CBCT in visualizing small but clinically relevant pelvic branches and preventing non-target embolization.

Several limitations of the present study should be acknowledged. The small sample size (particularly for group A and the subgroups with mid-term data) limits the generalizability of the conclusions of this study. Magnetic resonance imaging, which may be more accurate than CT and US/CEUS for PV and pPI measurement, was not systematically used. Uroflowmetry was not consistently performed, thus limiting the objective evaluation of functional outcomes.

In conclusion, compared with TGM 100–300, PAE with TGM 40–120 appears to provide comparable clinical benefit but significantly greater prostatic infarction and greater mid-term prostate shrinkage. The latter may indicate a more durable treatment effect of the smaller microspheres and merits further investigation. However, ischemic complications may be more frequent and more severe with the smaller microspheres.

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Footnotes

Conflict of interest disclosure

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Supplementary Tables 1–3.: https://d2v96fxpocvxx.cloudfront.net/beb8919b-f013-4ea1-b1c8-40332e840fe1/content-images/b037e4c9-9cc6-4bd8-a188-8a86268af6b1.pdf

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