COMMENTARY

Dealing with Prostatic Arteries—How Many Roads Must a Man Walk Down?

Tiago Bilhim, MD, PhD, EBIR, FCIRSE, FSIR

The study by de Assis et al titled "Angiographic Findings during Repeat Prostate Artery Embolization" (1) highlights 2 unanswered questions that have been pending since prostatic artery embolization (PAE) arose as an alternate treatment option for patients with benign prostatic hyperplasia (BPH) and lower urinary tract symptoms (LUTS). First, how many independent prostatic arteries (PAs) do we have (and how should we call them)? Second, should we embolize all arteries going into the prostate? If we did not know the answers before reading this manuscript, we will still probably not be able to answer them afterward. However, this study raises the potential role of additional PAs that are left nonembolized during PAE. The authors hypothesize that these arteries may be a reason for prostatic revascularization responsible for symptomatic relapse needing reintervention. A retrospective analysis of 10 patients who underwent repeated PAE 2–8 years after the first embolization is presented. All patients responded with symptomatic relief after the first PAE. However, relapsing LUTS prompted reintervention and repeated PAE was performed. During repeated PAE, almost 60% of pelvic sides had more than 1 PA feeding the prostate, whereas in the remainder 40% of pelvic sides only 1 PA was identified. The authors report 2 independent PAs feeding the prostate in 30% of pelvic sides during the first PAE, with 1 PA left nonembolized. This 30% rate of independent PAs is strikingly different from the previously reported 8% rate from the same group in a larger cohort of 143 patients (2). One could probably argue that this is due to selection bias, meaning that this is a subgroup of patients who previously underwent PAE and were "prone" to have more than 1 independent PA feeding the prostate. The sample size of 10 patients is just too small to answer this.

If we look into anatomy studies with cadavers, the reported rates of independent PAs feeding the prostate ranges from 0 (3), to 30% (4) and even 100% (5,6). When using computerized tomographic (CT) angiography (CTA) and digital subtraction angiography (DSA) to study the anatomy of the PAs, the reported rate of independent PAs is 40% (7). With the use of intraprocedural cone-beam CT (CBCT) and digital subtraction angiography, the reported rate of independent PAs ranges from 7% (8) to 20% (9). With such a huge variability in the reported rates of independent PAs, even when using the same technical approach (eg, cadavers, CTA+DSA, DSA alone, and DSA+CBCT) or within the same cohort (1,2), it seems obvious that something is elusive. Where is the truth? It depends on what you consider to be true or false, but usually it is somewhere in between extremes. There is more here than meets the eye; and probably these discrepancies depend on how hard you look for these arteries and how you name them. How should we call these arteries? The names of anterior-lateral and posterior-lateral PAs have been proposed based on the penetration of these branches in the prostatic capsule (7,10). The "main" PA for patients with BPH would naturally be the artery feeding the central gland. This artery has been found to penetrate the prostatic capsule in the anterior-lateral quadrants, so it is called the anterior-lateral (AL-PA) or central-gland PA. The artery feeding most of the peripheral zone of the prostate and prostate apex was found to penetrate the prostatic capsule in the posterior-lateral quadrants, so it is called the posterior-lateral (PL-PA) or peripheral-gland PA (1,5,7,10).

In the study by Assis et al (1) the PL-PA was present and independent from the AL-PA in 30% of pelvic sides during the first PAE and was held to be the main "culprit" for the prostatic revascularization in 21% of pelvic sides. Naturally, all nonembolized PL-PAs during the first PAE had vascularization to the prostate. But was this vascularization any different from the first PAE compared with the repeated PAE? Should these arteries have been embolized during the first procedure? One is left to wonder. The authors compared patients who underwent PL-PA embolization with those who did not during the first PAE and found that embolizing the PL-PA during the first PAE reduced the risk of revascularization through that artery. But what about other arteries feeding the prostate? The authors also showed that the distal segments of the obturator, superior vesical, and penile arteries were responsible for prostatic revascularization in 63% of pelvic sides. Should these arteries have been embolized in the first PAE? Were they even present at that

From the Centro Hepato-Bílio-Pancreático e da Transplantação, Hospital Curry Cabral and Saint Louis Hospital, Lisbon, Portugal. Received January 24, 2019; accepted January 25, 2019. **Address correspondence to** T.B., Saint Louis Hospital, R. Luz Soriano 182, 1200-249 Lisbon, Portugal; E-mail: tiagobilhim@hotmail.com

T.B. is a paid consultant for Terumo (Tokyo, Japan) and Merit Medical (South Jordan, Utah), an advisory board member for Merit Medical, a paid speaker for Philips (Eindhoven, The Netherlands), and a shareholder in Embolx (Sunnyvale, California).

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time? Probably not. PAs arising from penile arteries have been reported as collateral routes in patients with PA occlusion due to atherosclerosis (11). Naturally, they can also develop after nonatherosclerotic iatrogenic occlusions of the PAs. Most importantly, the AL-PA was also found to be the major PA responsible for prostatic revascularization in 42% of pelvis sides, leaving us wondering about the presumed "permanent" nature of the embolic materials we are using. If we put all the mathematics together (with or without the help of our friend the calculator), one could argue that AL-PA revascularization and the collaterals arising from the distal segments of the internal iliac branches accounted for virtually all prostatic revascularizations, questioning the added value of PL-PA embolization during PAE.

Assis et al imply that independent PL-PAs should be looked for and embolized during PAE (1). We should probably be cautious about advising to "seek and destroy" all potential PAs identified in the pelvis, because this approach may lead to more radiation exposure to patients and interventional radiologists with the potential to increase nontarget embolization. PAE is already a "feared" and "complicated" procedure owing to the complex anatomy of the pelvic vessels, with reported severe adverse events due to high radiation exposure and nontarget embolization (12,13). There are no doubts that repeated PAE makes this procedure even more complex with a higher rate of independent PAs feeding the prostate. Assis et al embolized 31 PAs in 10 patients (1) but, unfortunately, provide no data on fluoroscopy and procedural times, air kerma, or dose-area-product. So maybe instead of making a complex procedure even harder, we could look into ways of reducing the revascularization of the AL-PAs. Should we look into more "permanent" embolics? Naturally, we can not prevent collaterals from the distal segments of the internal iliac artery to develop after a successful PAE, but we can try to prevent revascularization of the embolized PAs. Preliminary data on PAE with liquid agents (ethylene vinyl alcohol copolymer) showed disappointing results (14). Placing coils in the AL-PA or gelfoam after embolization with particles is another option. But, as with uterine artery embolization, this is probably not a very good idea. Instead of preventing AL-PA revascularization, we are just making our lives miserable in the near future: closing an "easy" entrance door to the prostate to stimulate the development of collateral and tortuous "difficult" new doors. Maybe AL-PA revascularization or even PL-PA revascularization is "good news" when dealing with repeated PAE, because these arteries are probably easier to selectively catheterize. We are left with many unanswered questions, as always.

I am not sure about the suggestion of embolizing all potential feeders to the prostate during PAE. It has been shown recently in a retrospective analysis that embolizing 3 or more PAs did not lead to better clinical outcomes than embolizing 2 PAs, 1 on each pelvic side (15). I also doubt that the answers will be "blowin' in the wind." Should we follow all roads that lead into the prostate to perform PAE more effectively? One can only hope for future studies to tell, but interventional radiologists have been doing PAE quite well for the last decade.

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