Am J Clin Exp Urol 2024;12(3):141-148
www.ajceu.us /ISSN:2330-1910/AJCEU0157983

Original Article

The role of PSA kinetics in men with a negative MRI-targeted prostate biopsy

Marcelo P Bigarella, Arighno Das, Diana Garcia, Krista Brackman, Glenn Allen, David Jarrard

University of Wisconsin, Madison, Wisconsin, USA

Received May 22, 2024; Accepted June 24, 2024; Epub June 25, 2024; Published June 30, 2024

Abstract: Objective: To evaluate rebiopsy rates and clinicopathologic outcomes in patients after a negative MRI-guided biopsy to better inform the management of these patients. Methods: Patients were included with a clinical suspicion of prostate cancer (PCa) referred for fusion biopsy for a PI-RADS v2.1 lesion ≥ 3 on multiparametric MRI and a negative MRI fusion biopsy. Biopsies included targeted and systematic cores. Patients with a prior cancer diagnosis were excluded. Both baseline and follow-up clinicopathological data, and long-term PSA values were examined in these patients. Statistical analyses included Wilcoxon rank-sum test and one-way tests. Results: Of 685 total patients, 188 (27%) had a negative fusion biopsy. Of these 88 (47%), 74 (39%), and 26 (14%) had PI-RADS 3, 4, 5 lesions, respectively. Complete follow-up was available for 182/188 patients (97%), with a median of 24 months (interquartile range: 12-38). Post-biopsy PSA levels decreased the first and the second year (-0.24; and -0.84 ng/ml/yrs respectively). In follow-up, 44 patients had an MRI (24%) and 20 had a biopsy (10%). A positive PSA velocity was the only predictive variable for repeat MRI in univariate analysis. On repeat MRI, 9 (27%) patients had disappearance of the initial lesion, 21 (48%) had a lower PIRADS score and 14 (32%) higher. Only 12/182 (6.6%) were found to have PCa during follow-up, of those 7 (3.8%) were clinically significant. Conclusion: For patients with non-malignant biopsy findings after an initial mpMRI showing a suspicious PI-RADS lesion, the majority of patients will have their PSAs return to baseline over time. To support this, repeat MRI frequently demonstrated a disappearance or downgrading of PIRADS lesions. These data support monitoring patients with this clinical scenario.

Keywords: Prostate cancer, PSA, PSA kinetics, prostate mpMRI, MRI targeted fusion biopsy, PSA velocity

Introduction

New biomarkers and imaging modalities are being analyzed to both improve the specificity and predictive value of prostate specific antigen (PSA) as well as reduce the burden of biopsies resulting from a false-positive PSA test. Prostate multiparametric MRI (mpMRI) has been shown in randomized clinical trials to improve the predictive value of PSA by reducing the number of men that undergo biopsies while detecting more clinically significant cancer [1]. To date, there are still no published guidelines on the management of patients after a negative mpMRI-guided biopsy or how to best counsel these patients about their future risk of PCa detection.

Changes in PSA over time (i.e. PSA kinetics or PSA velocity) may be helpful in differentiating men with PCa from benign causes including benign prostate hyperplasia (BPH) or prostatitis [2]. However, the low predictive value of PSA in the pre-MRI era and the extended period of observation time required to evaluate PSA kinetics reduce its role in early-stage prostate cancer detection [3]. Typically, PSA kinetics use a PSA velocity (PSAV) threshold of 0.75 ng/ml/yr, or in patients with PSA of < 4 ng/ml a value of 0.35 ng/ml/yr is employed [4]. Despite a potential role for PSA kinetics, PSA progression in PCa is nonlinear and is thought to have an exponential pattern of increase suggesting a complex relationship among PSA kinetics, cancer detection, BPH and prostatitis [5].

In combination with PSA, mpMRI has been used as an imaging strategy to differentiate clinically significant and insignificant PCa from benign conditions. Rates of PCa detection for each scoring increase from PI-RADS 3 (15%), to PI-RADS 4 (50-60%) to PI-RADS 5 (70-80%) [6].

https://doi.org/10.62347/IWIB8107

A false-positive biopsy may arise due to a targeting error, or MRI reader variability or other conditions mimicking a tumor lesion such as BPH and prostatitis [7]. In the latter case, when the prostate is inflamed, PSA levels often increase dramatically and subsequently decrease spontaneously [8] and the risk of finding cancer is reduced when there is a decrease in a follow-up PSA done before rebiopsy [9].

Given the lack of information on how to counsel patients who present with a discrepancy between mpMRI and biopsy results, the goal of this study was to examine features that might inform this result. We determined that PSA significantly decreases during follow-up in most patients suggesting a noncancer etiology for their PSA rise. Additionally, we found that biopsied PIRADS lesions on MRI frequently disappear or are downgraded in this population. These data provide reassurance for patients who experience a negative MRI targeted fusion biopsy triggered by a clinically significant lesion on mpMRI.

Material and methods

Study population and follow-up

Between March 2018 to July 2022, patients referred for an MRI fusion prostate biopsy at a single tertiary care system who had a mpMRI with at least one lesion suspicious for prostate cancer greater or equal to PI-RADS 3 were evaluated under an IRB approved protocol. Patients with prior prostate cancer diagnosis were on Active Surveillance. Three main domains were evaluated in this study including: (1) clinical, such as age, race, prior biopsy history, serum PSA (ng/mL), and PSA density (ng/mL^2), (2) imaging history and characteristics, and (3) histopathological reports from all prostate biopsies done and other pertinent procedures, notably bladder outlet obstruction (BOO) surgeries. PSA kinetics, or PSA dynamics, were used to describe any measure of PSA along time, such as PSA velocity or PSA doubling time. PSA velocity or rate of PSA variation is the absolute difference in PSA in an elapsed period of time.

Repeat procedures were also reviewed, such as new mpMRI and new prostate biopsies - referred hereafter as repeat or follow-up MRI (Fu-MRI) and repeat follow-up biopsies (Fu-Bx). The data registry was compliant with the Health Insurance Portability and Accountability Act of 1996 (HIPAA), received institutional IRB approval and only unidentified information was made available for final analysis.

mpMRI and fusion biopsy protocol

All study patients underwent a 3T mpMRI exam. Imaging data collected included prostate volume, lesion score and size (maximal transverse diameter). Lesions were categorized according to the most updated version of the PI-RADS v2.1 score system by dedicated experienced uro-radiologists experienced in a high-volume National Cancer Institute designated center. Lesions reported to be PIRADS $\geq$ 3, 4 or 5 were targeted for biopsy on all patients. Fusion biopsies were done only by high-volume urologists. The prostate and PI-RADS score 3, 4 and 5 lesions were contoured (DynaCAD, Philips Healthcare, Massachusetts, USA) and imported to ultrasound (bk3000, BK Medical, Massachusetts, USA) with image-fusion software (UroNav, Philips Healthcare), and at least 3 targeted biopsy cores were taken for every suspicious lesion with a PI-RADS score $\geq$ 3. All patients underwent systematic template biopsy in addition to the targeted biopsy with at least 12 cores spatially distributed cores being taken. Cancer findings were reported according to the "International Society of Urological Pathologists" (ISUP) classification and the PSA value at time of biopsy was used to stratify patients with AUA risk classification.

Statistical analysis

Continuous variables were reported with the median and interquartile range (IQR), whereas categorical variables were reported with frequencies and proportions. Parametric (ANOVA) and nonparametric (Wilcoxon rank-sum) methods were used to evaluate differences in the baseline characteristics among the different groups. Additionally, differences between PSA values and rates at different time intervals were evaluated among the different groups. PSA velocity was calculated considering the absolute difference in PSA within a period. The two-tailed Fisher's exact test was used to evaluate differences in categorical variables, and univariate analysis using logistic regression was used to test differences between two groups. All *p* values were two-sided, with a sta-

Table 1. Baseline characteristics of patients with a positive MRI (PI-RADS ≥ 3) and a subsequent nonmalignant MRI fusion biopsy

Variable	
Number of Patients	188
Age, median (IQR), years	65 (60-69)
History of prior systematic prostate biopsies	83 (44%)
Prostate vol, median (IQR), mL	60.5 (43-87)
PSA, median (IQR), ng/mL	7.2 (5.14-11.1)
PSA density, median (IQR), ng/mL^2	0.12 (0.08-0.17)
Highest PI-RADS for the patient	
3	88 (47%)
4	74 (39%)
5	26 (14%)
Number of lesions PI-RADS ≥ 3 lesions	
1	102 (54%)
2	64 (34%)
≥ 3	22 (12%)
Max diameter of lesion, median (IQR), mm	1.4 (1.2-2.0)
Max diameter of lesion with highest score, median (IQR), mm	1.2 (0.9-1.8)
Interval (initial MRI, initial fusion biopsy), days	54 (41-77)
Mean number of cores	17 (16-20)

tistical significance set at $P < 0.05$. Statistical analyses were performed using Stata (17.0, StataCorp LLC, College Station, TX).

Results

Baseline characteristics

A total of 685 patients had a mpMRI with at least one lesion suspicious for prostate cancer ≥ PI-RADS 3 underwent an MRI-transrectal ultrasound (MRI-TRUS) targeted fusion biopsy. Of these patients, a total of 188/685 (27%) patients had a negative no cancer detected fusion biopsy (**Table 1**).

This negative biopsy subset had a median age of 65 years and 93.2% identified as Caucasian. Fewer than half had a prior biopsy (44%). Median PSA, PSA density, and prostate volume were 7.21 ng/mL, 0.12 ng/mL^2, and 60.5 mL, respectively (**Table 1**). Targeted biopsies were based in PI-RADS score 3, 4 and 5 in 73 (47%), 70 (39%) and 35 (14%) patients. Most patients had just one conspicuous lesion (54%) or 2 lesions (34%), with a median maximum diameter of 1.4 (1.2-2.0) mm. The median interval time between the mpMRI and the MRI targeted biopsy was 54 days. MRI targeted fusion biopsy was done in addition to systematic sampling, and on average patients had 17 cores taken (IRQ: 16-20), including 12 from the systematic distribution and the remainder from the targeted lesions.

Outcomes of patients with nonmalignant MRI fusion biopsy

Follow-up information was available for 182/188 (97%) of individuals with a median follow-up interval of 24 months (IQR: 12-38). In patients that received no further imaging, 14 (8%) patients had a urological surgery, the majority (12/14) underwent HoLEP (Holmium laser enucleation of the prostate) and had pathological specimens available. The median interval between the negative initial fusion biopsy and the surgery was 8 months (IQR: 4-27 mo). During follow-up, 44/182 (24%) patients had a repeat mpMRI which led to a repeat biopsy in 20/182 (11%) patients (**Figure 1**).

With a median FU of 40 months (IQR: 25-46) months, overall 12/182 (6.6%) patients received the diagnosis of PCa by either additional biopsy (n = 8) or surgical intervention for BOO (n = 4). The median time to PCa diagnosis was 21 (IQR: 11-33) mo overall. Among the 12 cancers

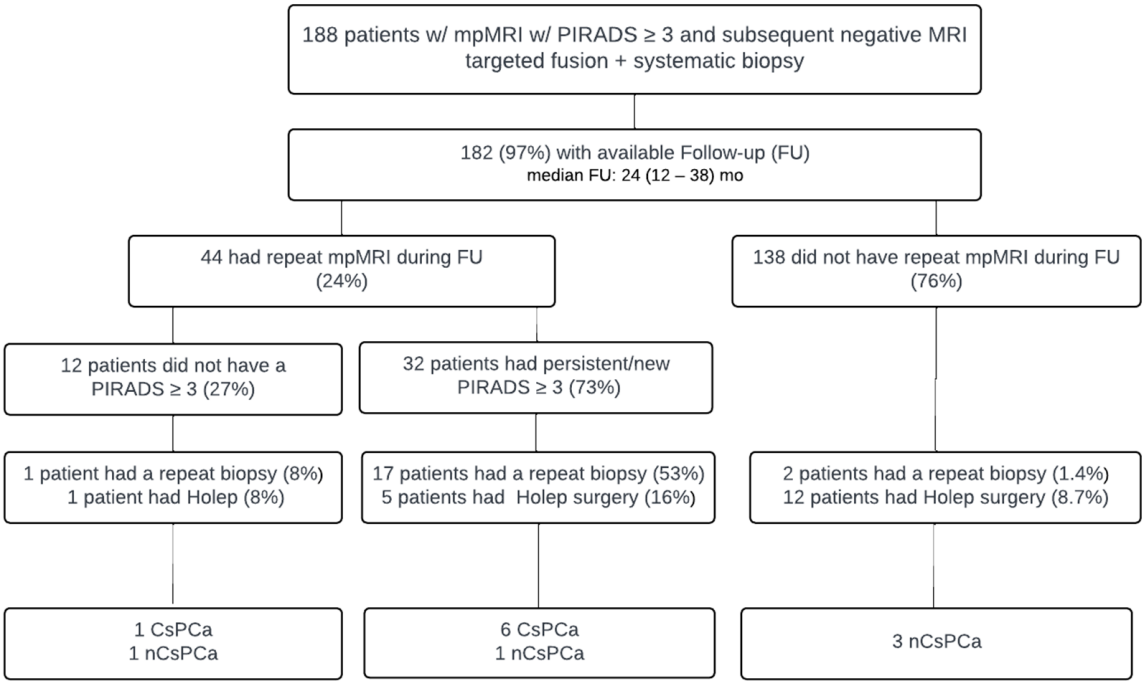

Figure 1. Outcomes of patients after a negative biopsy. Most of the PCa detected in follow-up was after repeat imaging (75%). Most of patients who had a repeat mpMRI during FU had a persistent PI-RADS lesion (73%). CsPCa = clinically significant prostate cancer (Grade group, ISUP ≥ 2), nCsPCa = not clinically significant prostate cancer (Grade group, ISUP = 1), Holep = Holmium laser enucleation of prostate, FU = follow-up, MRI = CsPCa = clinically significant prostate cancer, PI-RADS = Prostate Imaging Reporting & Data System.

found, 7 (58%) were clinically significant (3 patients ISUP = 2, 2 ISUP = 3, 1 ISUP = 4, and 1 ISUP = 5). Five (42%) were pathologically insignificant. For the 12 patients in whom prostate cancer was found, the initial MRI lesion was a PI-RADS score 3, 4, and 5 in 41%, 25% and 27%, respectively. All 4/12 (33%) patients found to have prostate cancer after a HoLEP procedure were placed on active surveillance. Out of 8/12 (67%) patients found cancer on repeat biopsy, 4/8 (50%) were treated with radiation, 1/8 (12.5%) had a radical prostatectomy, and 3/8 (37.5%) were placed on active surveillance.

PSA decreases in patients after a negative biopsy during follow-up

For all 182 patients with follow-up, 2651 PSA measurements were obtained for an average of 13.6 PSA measurements per patient. To track changes in PSA velocity, values were pooled into annual intervals categories before and after the index event (2 years before, 1 year before, index PSA, 1 year after, 2 years after) (**Figure 2A**). When considering the entire cohort, PSA values decreased annually after the initial MRI and fusion biopsy termed the 'index PSA' (7.1 ng/ml (5.0-11.1) to 6.0 (4.7-8.6) to 5.3 (4.2-7.5)). In contrast, PSAs taken prior the index PSA significantly increased annually (5.3 ng/ml (4.2-7.5) to 5.7 (4.7-8.3) to 7.1 (5.0-11.1); P = 0.002, P = 0.0009, respectively) (**Figure 2A**). When plotted temporally, an increase reaching the index PSA was noted for most of patients (54% and 65% of the entire cohort, at 1 and 2 years respectively), followed by a continuous decrease, and return to baseline levels. Positive PSA velocities of 0.84 and 1.68 ng/ml/yrs (two and one year before the index PSA) were noted that triggered the initial MRI and MRI fusion biopsy.

In patients who had a repeat follow-up MRI, PSA kinetics differed substantially. A continued PSA increase in the first 24 months above the index PSA was seen. PSA velocities for patients having a repeat MRI were positive compared to the subset that did not have repeat MRI (P < 0.05 for both) (**Figure 2B**). Using logistic regression, no clinicopathologic variables (prostate volume, PI-RADS score at initial MRI, PSA den-

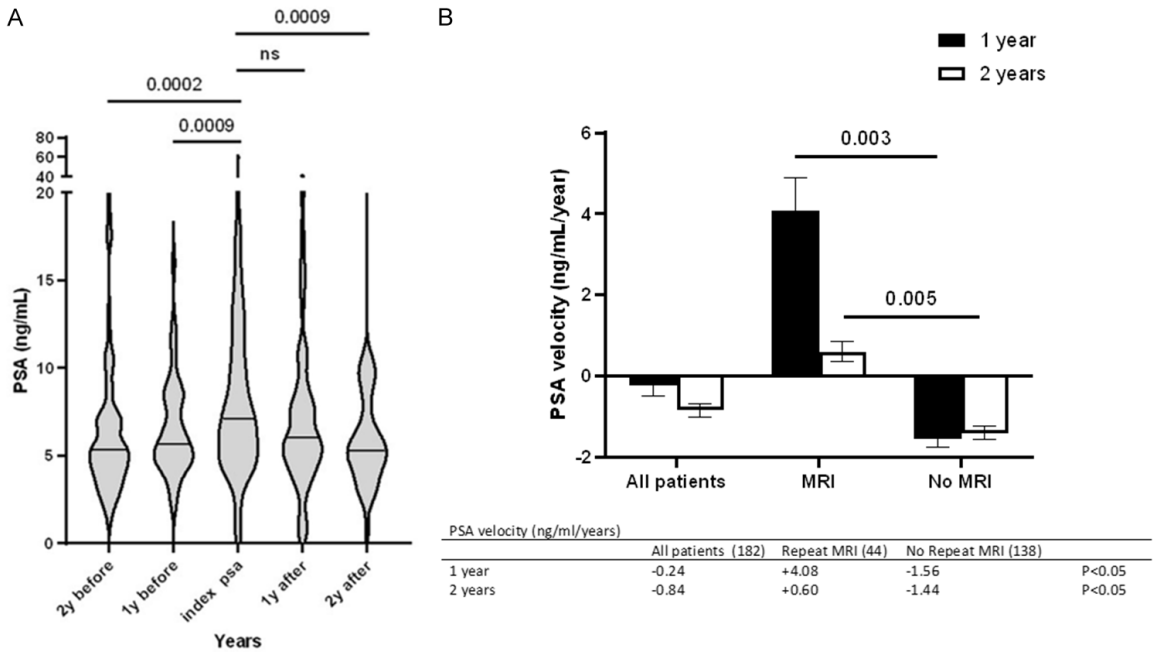

Figure 2. PSA decreases after a negative MRI fusion biopsy. A. PSA values before and after index PSA, at different time intervals (1 year and 2 years). A rise in PSA, followed by a decrease and return to baseline levels was noted for the patients with a negative biopsy during those 4 years. B. PSA velocity between patients who had a follow-up MRI and those who did not, and the entire cohort, at different time interval after index PSA. Negative values mean decrease in PSA over time, whereas positive ones mean increase in PSA.

sity, number of lesion, size of lesion) were different for the patients who had a FU-MRI, with exception of the PSA velocity at 1 year (OR 1.42, CI 95% 1.14-1.76, P = 0.001) and 2 years (OR 2.22, CI 95% 1.12-4.41, P = 0.022).

Repeat MRI demonstrates changes in lesion PIRADS scoring

Forty-four of 182 (24%) patients had a repeat mpMRI during FU, among which 9/44 (20%) patients were found to have PCa (Supplementary Table 1). Within those nine patients, the initial MRI lesion was a PI-RADS score 3, 4, and 5 in 33%, 22% and 44%, and on the repeat MRI a PI-RADS score ≤ 2, 3, 4, and 5 in 22%, 11%, 0% and 67%, respectively.

The median interval between the initial MRI and a repeat MRI was 25 months (IQR: 15-35), and this interval was shorter for patients with a more concerning initial PIRADS lesion fluctuating from 28 (14-35), 23 (15-36), 17 (12-28) months for PI-RADS score 3, 4 and 5, respectively (P < 0.05).

When we compared that subset of 44 patients who had a FU-MRI, marked changes in the total number of lesions, size, and highest score were seen. The initial mpMRI showed PI-RADS 3, 4 and 5 in 19 (43%), 14 (32%) and 11 (25%), respectively. In contrast, the FU-MRI showed PI-RADS 3, 4 and 5 present in 12 (27%), 10 (23%), 10 (23%) of patients, respectively (**Figure 3**). Another 12 (27%) of patients had a complete resolution of the previously reported lesion. Alterations in PIRADS scoring occurred in 35/44 (80%) with 21 (48%) developing a lower score and 14 (32%) a higher score. Of note, among the 10 patients with a new or persistent PI-RADS 5 lesion on MRI, cancer was detected in 7 (70%) of them.

On FU-MRI 32 patients had a persistent/new PIRADS ≥ 3 lesion, among which 17 (53%) had a repeat biopsy. 12 patients had an MRI with no PIRADS ≥ 3 lesion - only 1 patient (8%) got a subsequent biopsy.

Discussion

Prostate cancer diagnosis has significantly changed with the advent of mpMRI. The PROMIS and PRECISION trials demonstrated the superiority of pre-biopsy mpMRI, as well as

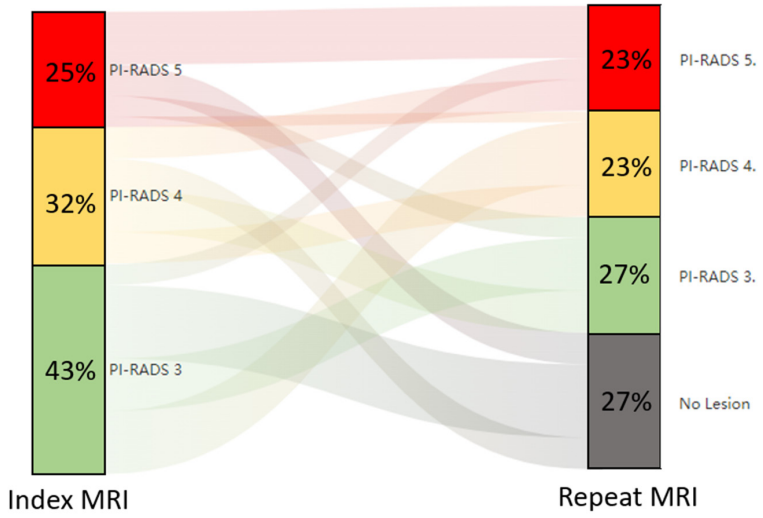

Figure 3. Sankey diagram demonstrating differences in the percentage of PI-RADS 3, 4, and 5 lesions from the index MRI-guided biopsy compared with the repeat MRI done during follow-up.

MRI-targeted prostate biopsy over systematic biopsy in the detection of clinically significant prostate cancer [1, 10]. However, factors that affect mpMRI diagnostic accuracy, including small lesions or chronic inflammation, reduce the mpMRI positive predictive value (PPV). Thus, a non-negligible proportion of patients with a positive mpMRI do not harbor clinically significant disease. The outcome for those patients (suspicious mpMRI lesions and non-malignant biopsy histology) are still not well-defined. In the current study, we found that PSA velocity decreases significantly for most patients in longitudinal follow-up suggesting a noncancer etiology. Furthermore, when MRI is performed in the subset with persistently elevated PSAs, concerning PIRADS lesions commonly disappear. These longitudinal data support mpMRI as an accurate biomarker to assist in the detection of clinically significant PC.

To date, there is a lack of longitudinal data regarding the false-negative rate of mpMRI-targeted biopsy and how PSA kinetics might be a useful addition to imaging in the era of mpMRI prior to prostate biopsies [11]. In the current dataset with complete follow-up (97%) and a time interval spanning up to 24 months following the initial workup for prostate cancer, we found an overall decrease in the PSA with levels eventually returning to baseline. PSA levels prior to this index biopsy event continuously increased. With prostate inflammation, PSA levels increase dramatically and subsequently decrease spontaneously [8]. Guidelines support a repeat PSA prior to biopsy as the risk of PCa is reduced when there is a decrease in a repeat PSA done before biopsy [9].

We also found that PSA kinetics, specifically the PSA velocity, defined those patients who ended up having a repeat MRI (24%) from the patients who did not in the years following the MRI fusion biopsy. Other clinicopathologic features (e.g. PSA density, inflammation) were not predictive. In patient imaging FU, almost one third (12/44, 27%) of FU-MRIs converted to negative over a median interval between consecutive MRIs of 25 mo. Twenty patients out of the total 182 patients (11%) underwent a follow-up biopsy. For those patients, eight (40%) had either a persistent or a newly diagnosed PIRADS 5 lesions. This finding suggests closer patient surveillance is required when a PIRADS 5 lesion is present on the repeat MRI since most of the patients (5/7, 71%) with clinically significant prostate cancer had this finding. Other authors also retrospectively examined this negative MRI guided biopsy subset of patients aiming to find baseline predictors of clinically significant PCa being detected. Barletta et al. found that 4.9% of these patients were found to have clinically significant PCa and that persistent positive mpMRI was associated with a higher risk, whereas low PSA density (< 0.15 g/mL^2) decreased this risk. Lastly, von Landerberg et al. found a rate of 7.95% for newly detected PCa in the same population, with high PSA density (> 0.13 g/mL^2) and small prostate volume being significant predictors of future PCa detected during FU.

Limitations include the retrospective nature of this study and consequently no standardized FU biopsy or labs were in place that might limit the generalizability of our findings. However, the majority of patients adhered to a PSA annually or biannually (63% in the first 2 years) and our data supports this as an important component

of follow-up. Being a single institution has limitations, but imaging and reporting was performed in a standardized setting, with experienced urologists doing the biopsy and academic radiologists dedicated to prostate MRI. Stabile and et al. have shown factors influencing variability in the performance of mpMRI in detecting PCa and found that the experience levels of the radiologists and/or biopsy operators are key confounders [12]. Others include invisible cancer, the reader's misinterpretation or targeting failure (missed target or undersampled by MRI-targeted biopsy).

Conclusion

To date in studies examining MRI prior to prostate biopsies and advanced targeting techniques none of them reported how PSA performed over time after the initial nonmalignant fusion biopsy results. We find that PSA decreases over time in patients with a positive MRI but negative fusion biopsy indicating a nonmalignant reason for the elevated PSA in this population. Supporting this, in our cohort the rates of prostate cancer detection were low (6.6%), even lower when only considering significant prostate cancer with ISUP ≥ 2 (3.7%). Our data suggests that many patients will have their PSAs return to baseline if monitored after a fusion biopsy. The monitoring of PSA kinetics rather than reimaging or rebiopsy is supported for the primary management of this clinical scenario.

Disclosure of conflict of interest

None.

Address correspondence to: Marcelo P Bigarella and David Jarrard, University of Wisconsin, Madison, Wisconsin, USA. Tel: 608-421-4918; E-mail: bigarella@urology.wisc.edu (MPB); Tel: 608-262-0759; E-mail: jarrard@urology.wisc.edu (DJ)

References

[1] Kasivisvanathan V, Rannikko AS, Borghi M, Panebianco V, Mynderse LA, Vaarala MH, Briganti A, Budäus L, Hellawell G, Hindley RG, Roobol MJ, Eggener S, Ghei M, Villers A, Bladou F, Villeirs GM, Virdi J, Boxler S, Robert G, Singh PB, Venderink W, Hadaschik BA, Ruffion A, Hu JC, Margolis D, Crouzet S, Klotz L, Taneja SS, Pinto P, Gill I, Allen C, Giganti F, Freeman A, Morris S, Punwani S, Williams NR, Brew-Graves C, Deeks J, Takwoingi Y, Emberton M and Moore CM; PRECISION Study Group Collaborators. MRI-targeted or standard biopsy for prostate-cancer diagnosis. N Engl J Med 2018; 378: 1767-77.

[2] Berger AP, Deibl M, Steiner H, Bektic J, Pelzer A, Spranger R, Klocker H, Bartsch G and Horninger W. Longitudinal PSA changes in men with and without prostate cancer: assessment of prostate cancer risk. Prostate 2005; 64: 240-5.

[3] Vickers AJ, Savage C, O'Brien MF and Lilja H. Systematic review of pretreatment prostate-specific antigen velocity and doubling time as predictors for prostate cancer. J Clin Oncol 2009; 27: 398-403.

[4] Potter SR and Carter HB. The role of prostate-specific antigen velocity in prostate cancer early detection. Curr Urol Rep 2000; 1: 15-9.

[5] Carter HB and Pearson JD. PSA velocity for the diagnosis of early prostate cancer. A new concept. Urol Clin North Am 1993; 20: 665-70.

[6] Mazzone E, Stabile A, Pellegrino F, Basile G, Cignoli D, Cirulli GO, Sorce G, Barletta F, Scuderi S, Bravi CA, Cucchiara V, Fossati N, Gandaglia G, Montorsi F and Briganti A. Positive predictive value of prostate imaging reporting and data system version 2 for the detection of clinically significant prostate cancer: a systematic review and meta-analysis. Eur Urol Oncol 2021; 4: 697-713.

[7] Kitzing YX, Prando A, Varol C, Karczmar GS, Maclean F and Oto A. Benign conditions that mimic prostate carcinoma: MR imaging features with histopathologic correlation. Radiographics 2016; 36: 162-75.

[8] Gallo L. The effect of a pure anti-inflammatory therapy on reducing prostate-specific antigen levels in patients diagnosed with a histologic prostatitis. Urology 2016; 94: 198-203.

[9] De Nunzio C, Lombardo R, Nacchia A, Tema G and Tubaro A. Repeat prostate-specific antigen (PSA) test before prostate biopsy: a 20% decrease in PSA values is associated with a reduced risk of cancer and particularly of high-grade cancer. BJU Int 2018; 122: 83-8.

[10] Ahmed HU, El-Shater Bosaily A, Brown LC, Gabe R, Kaplan R, Parmar MK, Collaco-Moraes Y, Ward K, Hindley RG, Freeman A, Kirkham AP, Oldroyd R, Parker C and Emberton M; PROMIS study group. Diagnostic accuracy of multi-parametric MRI and TRUS biopsy in prostate cancer (PROMIS): a paired validating confirmatory study. Lancet 2017; 389: 815-22.

[11] Wei JT, Barocas D, Carlsson S, Coakley F, Eggener S, Etzioni R, Fine SW, Han M, Kim SK, Kirkby E, Konety BR, Miner M, Moses K, Nissenberg MG, Pinto PA, Salami SS, Souter L,

Thompson IM and Lin DW. Early detection of prostate cancer: AUA/SUO Guideline part I: prostate cancer screening. J Urol 2023; 210: 46-53.

[12] Stabile A, Giganti F, Kasivisvanathan V, Giannarini G, Moore CM, Padhani AR, Panebianco V, Rosenkrantz AB, Salomon G, Turkbey B, Villeirs G and Barentsz JO. Factors influencing variability in the performance of multiparametric magnetic resonance imaging in detecting clinically significant prostate cancer: a systematic literature review. Eur Urol Oncol 2020; 3: 145-67.

Supplementary Table 1. Comparison between patients who had a repeat MRI and those who did not during their follow-up

	Patients with a repeat MRI (n = 44; 24%)	Patients without a repeat MRI (n = 138; 76%)	
Age, median (IQR), years	63 (58-66)	67 (62-71)	
Prior prostate biopsies	20 (45%)	60 (44%)	
Prostate vol, median (IQR), mL	56 (44-80)	56 (38-78)	
PSA, median (IQR), ng/mL	7.3 (4.8-13)	6.6 (4.6-9.1)	*P < 0.05*
PSA density, median (IQR), ng/mL	0.13 (0.08-0.19)	0.12 (0.08-0.16)	
Highest PI-RADS			
3	18 (42%)	56 (41%)	
4	11 (33%)	58 (42%)	
5	10 (23%)	24 (17%)	
Number of lesions PI-RADS > 3			
1	28 (64%)	71 (51%)	
2	12 (27%)	48 (35%)	
> 3	2 (5%)	17 (12%)	
Max diameter of all lesions, median (IQR), mm	1.2 (0.9-1.7)	1.2 (0.9-1.5)	
Interval mpMRI and MRI Bx	1.8 (1.3-2.8)	1.5 (1.1-2.2)	
Interval MRI and FU-MRI	25 (14-34)		
FU-MRI PIRADS, mo (IQR)			
3	28 (14-35)		
4	23 (15-36)		
5	17 (12-28)		
Fu-Bx	18 (41%)	1 (1%)	*P < 0.05*
Prostate Cancer detected	9 (20%)	3 (2.2%)	*P < 0.05*
CsPCa detected	7 (16%)	0	*P < 0.05*
Time to first PSA (mo)	8.5 (7.2-11.5)	7.9 (6.0-9.8)	