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Focal Therapy for Prostate Cancer: Evolutionary Parallels to Breast Cancer Treatment. Letter.

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To the Editor: We read with great interest the recent manuscript by Labbate et al reviewing the development of focal therapy in breast and prostate cancer (PCa).[1] Breast focal therapy (bFT) represents a successful model for organ-sparing cancer treatment, so as urologists sensible to prostate focal therapy (pFT) we can learn much about the complex process of bFT recognition by the scientific community.

In 2017, we wrote about the analogy between bFT and pFT, and much has changed since our publication.[2] Labbate et al elegantly described latest updates on the comparison between bFT and pFT.

Still, we would like to report some evidence coming from recently published retrospective and prospective studies on pFT:

First, PCa screening based on PSA levels alone has led to an ethical dilemma in that a large proportion of patients undergo radical treatment, suffering permanent side effects by treating a disease that might never have progressed in their lifetime. A recent study showed that cancers invisible on multiparametric magnetic resonance imaging (mpMRI) are at lower risk of progressing than mpMRI-positive cancers.[3] Thus, systematic biopsy might be able to be avoided in favor of mpMRI-directed targeted biopsy that could decrease overdiagnosis and subsequent overtreatment of PCa.[3]

Second, similarly to bFT, criteria for pFT patient selection have changed. Recently, clinical trials on pFT only enrolled patients with biopsy-proven Gleason Grade (GG) 3+4 or GG 4+3 cancers, with patients having GG 3+3 cancer allocated to active surveillance (AS) programs.[4] Of note, pFT is a good option for patients not eligible for up-front AS, providing the chance to undergo an AS regimen following successful ablation.

Third, similar to bFT, limitations associated with mpMRI are leading to the development of new imaging modalities aimed to better characterize intraprostatic disease burden, and more accurately guide treatment planning and surveillance for pFT. Combining micro-US targeted biopsy with mpMRI-targeted biopsy could better select patients eligible for pFT.[5] Prostate-specific membrane antigen–targeted radiotracers combined with mpMRI may improve index lesion detection, intraprostatic gross tumor volume, and better predict the presence of adverse pathology.[5] Using contrast-enhanced US for real-time evaluation of tissue microvasculature, focal therapists may be potentially able to intraoperatively determine the adequacy of ablation and perform an immediate re-treatment, as needed.[5]

Similar to breast cancer, PCa outcomes of interest are changing over time. We believe that defining oncologic outcome by progression-free survival is a milestone in the history of pFT, leading to a higher quality of results.[4] In addition, quality of life outcomes are becoming increasingly important, and, as for bFT, pFT has a much lower rate of adverse effects compared to radical techniques.[5]

Finally, we should learn from the work of our breast colleagues that a better understanding of the disease biology will lead to effective combinations of local and systemic therapies for PCa, and this will be the key to prevent, detect, and treat the recurrence in pFT.[2] A comprehensive, biological-based, and multimodal approach to pFT will help to improve progression-free survival and patient quality of life.

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AUA-recommended Antibiotic Prophylaxis for Primary Penile Implantation Results in a Higher, Not Lower, Risk for Postoperative Infection: A Multicenter Analysis. Letter.

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To the Editor: Barham and colleagues continue a run of our field evaluating antibiotic use for surgical prophylaxis, which is to be commended.[1] However, their conclusions are premature and appear based on incomplete data. Specifically, they claim both that the "AUA-recommended antibiotic prophylaxis for primary penile implantation results in a higher, not lower, risk for postoperative infection" and that "antifungals significantly decreased the risk of infection by 92%."[1]

The first claim appears based upon their interpretation of the most recent AUA recommendations which include an aminoglycoside and vancomycin as first-line prophylaxis.[2] While other authors have commented on their classification of certain antibiotic selections that are in the guidelines as "other," none have addressed the second part of the AUA recommendation: the duration ($\leq$24 hours). In addition to this omission, albeit mentioned in their limitations, they do not control for potential oral antibiotics at discharge; vancomycin/gentamicin with a week of oral antibiotics would not count as AUA-recommended therapy despite it being classified as such by the authors. Duration matters as evidenced by the fact that both over- and undertreatment can increase the risk of infection.[3] Other key information missing from their models is perioperative variables (eg, approach, duration, time to infection), antibiotic use prior to explantation, which would influence Table 4 in the article, and surgeon effect. While stated that "all surgeons were high-volume, experienced prosthetic surgeons," this assumption of equivalence denies the importance of surgical technique on surgical site infection, not to mention recent urological literature suggesting high-volume status might not be a proxy for skill.[4,5] Furthermore, to fully understand the impact of antibiotics on infections, one should include the susceptibilities of the isolated organisms, and if present, prior colonization with multidrug-resistant organisms. Lastly, the gram-positive anaerobes isolated in this cohort (*Propionibacterium, Peptococcus*, and *Finegoldia magna*) are susceptible to vancomycin, questioning the utility of additional coverage as proposed by the authors and in one editorial comment.[6,7]

As for the claim that antifungals decrease postoperative infection, it seems to imply in this case that correlation means causation. Table 4 in the article presents Candida species in 4% (1/25) of the gentamicin/vancomycin group's explantations compared to 0% (0/22) in the other regimen group, while no organism was identified in 20% (5/25) and 50% (11/22), respectively. Claiming that antifungals decrease the risk of infection by 92%, or rephrased, given the lack of shown fungal growth, that antifungals decreased the risk of bacterial infection by 92%, should cause the reader to pause. In the absence of a culprit organism and correction for the aforementioned excluded covariates, one must ask if this association is a proxy for an unidentified confounder, thus disproving the authors' stated causation.

We applaud the work that went into the preparation of this large series, but we would encourage further work before some of the presented claims can truly be supported. We do agree, however, that we should evaluate the influence of guideline adherence on infection after primary penile implant surgery which, unfortunately, was not done in the present article, given the lack of key information.

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