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Commentary

Biochemical Recurrence of Prostate Cancer Towards More Precise Stratification

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DESCRIPTION

Prostate Cancer (PCa), the second most diagnosed cancer in men, remains a leading contributor to cancer-related mortality globally. While treatments such as Radical Prostatectomy (RP) and Radiotherapy (RT) offer curative potential for localized PCa, up to half of treated patients experience a rise in prostate-specific antigen (PSA) a condition known as Biochemical Recurrence (BCR). However, not all BCR cases signify an immediate or life-threatening relapse. Distinguishing between low-risk and High-Risk BCR (HR-BCR) is essential for tailoring patient management and avoiding unnecessary treatment.

Historically, BCR definitions have focused on PSA thresholds alone, yet these do not sufficiently predict clinical outcomes such as metastasis or cancer-specific mortality. Recent refinements, such as those proposed by the European Association of Urology (EAU) and the EMBARK trial, incorporate PSA kinetics and tumor characteristics to better stratify patients at risk. A population-based study in Stockholm has now provided robust real-world evidence comparing these definitions and their association with PCa-Specific Mortality (PCSM), offering critical insights for clinical practice. The Stockholm study showed that these definitions not only vary in the proportion of patients classified as HR-BCR but also in their prognostic implications. Specifically, EMBARK identified a smaller subset of patients with the highest risk of PCSM 30% after RP and 50% after RT over ten years underscoring its value in identifying those who may benefit most from intensified therapy.

Despite growing evidence supporting risk-adapted management of biochemical recurrence, implementing stratification tools like the EMBARK criteria in real-world practice remains a challenge. Many clinics still rely heavily on fixed PSA thresholds without accounting for PSA kinetics or tumor grade. To bridge this gap, there is a need for clear clinical pathways and electronic health record integration that prompt clinicians to calculate PSA doubling times and reference histopathologic data when BCR is detected. Additionally, broader clinician education and guideline updates are crucial to shift practice patterns from reactive to proactive, evidence-based decision-making. The Stockholm study reinforces that refining recurrence definitions is not merely an academic exercise but a practical necessity with tangible consequences for patient survival and quality of life.

Among over 17,700 men with localized PCa treated between 2003 and 2021, approximately 21% of RP patients and 15% of RT patients experienced BCR. However, only 9% and 7% met the EAU HR-BCR criteria, and an even smaller proportion 3% and 8% respectively met the EMBARK criteria. Importantly, many patients with BCR never progressed to HR-BCR, and those who did not meet the HR-BCR definitions had substantially lower PCSM rates.

This distinction is critical. While traditional definitions might flag BCR early, they can also lead to overtreatment in patients with indolent disease. The EMBARK criteria, though more restrictive, appear more precise in isolating patients at genuine high risk for poor outcomes. For instance, while 10% of patients developed EAU HR-BCR over ten years, the EMBARK criteria narrowed this to 4% after RP and still identified those with the highest PCSM.

The study also revealed temporal patterns in BCR progression. Most HR-BCR cases occurred within three years after RP and within five years after RT. This finding has practical implications: PSA monitoring should be more frequent and vigilant during these periods, particularly for patients with high-grade tumors or unfavorable PSA kinetics. Moreover, a significant share of patients with BCR remained in a low-risk state for prolonged periods. This suggests that immediate salvage therapy is not always necessary and that a period of close observation could be a safe and effective approach for many.

The challenge in managing BCR lies in distinguishing signal from noise. Rising PSA levels post-treatment can trigger alarm, but not all relapses warrant the same urgency. The Stockholm study offers a compelling argument for more sophisticated tools like the EMBARK criteria that focus not just on PSA values but on the rate of change and underlying tumor biology. This move toward precision is particularly important in the era of value-based care. Avoiding overtreatment minimizes side effects and preserves quality of life, while ensuring timely intervention for high-risk patients improves outcomes.

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CONCLUSION

The management of biochemical recurrence after prostate cancer treatment is evolving. While traditional definitions of BCR have served as useful clinical markers, they are increasingly being supplemented or even replaced by risk-adapted frameworks that consider PSA kinetics and histologic features. The EMBARK criteria stand out as a powerful predictor of prostate cancer-specific mortality, helping to focus treatment where it's needed most. Incorporating refined definitions into clinical practice can optimize outcomes, reduce unnecessary interventions, and improve the overall quality of care for men with prostate cancer.