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Diagnostic challenges for the distinction of high-grade prostatic adenocarcinoma and high-grade urothelial carcinoma of simultaneousoccurrences - A literature review

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Abstract: Two of the most prevalent types of cancer in men are prostate adenocarcinoma and urothelial carcinoma. Both can appear separately in the prostate and bladder, simultaneously as separate tumors affecting either organ or sporadically as a collision tumor. Distinguishing these tumors by the pathologist can be challenging, especially when the high-grade, poorly differentiated forms infiltrate the surrounding organs. The correct approach by the pathologistis important due to the different treatment modalities for these two entities. This review of theliterature gives a comprehensive overview, our succinct understanding of the significance of correctly differentiating between these two tumors, the challenges involved in doing so, and the best collection of crucial and useful immunohistochemical markers for better diagnostic performance.

The scientific papers used in this review were retrieved from the PubMed and Google Scholar databases. All the studies in this review have recently been peer-reviewed and published in academic journals.The literature was sifted through to find the most relevant and up-to-date information for medical professionals, specifically pathologists.

The review concluded that: 1) Prostatic and urothelial markers such as NKX3.1, p63, thrombomodulin, and GATA3 are very useful for distinguishing prostatic adenocarcinoma from urothelial carcinoma. 2) Prostate Specific Antigen (PSA) is a good (clinical) screening tool, but because of its inverserelationship with tumor grade (the higher the grade, the lower the sensitivity of PSA staining),it is not recommended for high-grade tumor differentiation. 3) HMWCK (34βe12) and

p63 aresaid to be more effective than thrombomodulin and S100p in detecting urothelial cancer. 4) Thrombomodulin is only moderately sensitive to urothelial carcinoma. 5) Cytokeratins 7 and 20 can be positive in both urothelial carcinoma and prostatic adenocarcinoma, therefore their use is restricted. The optimal combination of these markers may improve the ability to distinguish these tumors.

Keywords: *prostate adenocarcinoma, urothelial carcinoma, immunohistochemistry, pathologic diagnosis, collision tumor*

Introduction: Prostate adenocarcinoma (PAC) and urothelial carcinoma (UC) are two of the most common cancers affecting men worldwide. Both can manifest independently in each organ (prostate and urinary bladder), concurrently as separate tumors involving either organ or occasionally as a collision tumor. Pathologic differentiation between these tumors can be difficult, particularly in poorly differentiated, high-grade forms that infiltrate neighboring organs. Because of the different treatment modalities for these two entities, the distinction between histologic and immunohistochemical patterns is important. This review of the literature provides an overall summary and our concise understanding of the importance of proper differential diagnosis between these two tumors, the difficulties encountered in this process, and the best set of critical immunohistochemical markers for improved diagnostic performance.

Methods: The scientific papers used in this review were retrieved from the PubMed and GoogleScholar databases using various combinations of the following search keywords: prostate adenocarcinoma, urothelial carcinoma, immunohistochemistry, pathologic diagnosis, differential diagnosis, and collision tumor. Papers were restricted to human subjects and the English language. All the studies in this review have recently been peer-reviewed and published in academic journals. Small-scale studies that produced no statistically significant results were excluded. The inclusion criteria for UC were muscle-invasive, a high-grade diseasewith no variant morphology. The inclusion criteria for PAC were high grade tumors with Gleason score of 9 or 10, according to WHO/ISUP 2014. Studies that had assessed patients who had received neoadjuvant chemotherapy, hormone therapy, or radiation therapy were not includedin this study. A total of 27 publications were selected. The literature was critically evaluated, to find the most relevant and up-to-date information for medical professionals, specifically pathologists, to help distinguish between high-grade urothelial carcinoma and high-grade prostatic adenocarcinoma.

Discussion: Prostate and urothelial carcinoma are two of the most common cancers in men worldwide. One in every eight men will be diagnosed with prostate cancer during his lifetime.Prostate cancer is more common in older men and non-Hispanic Black men. About 6 out of 10cases are diagnosed in men 65 and older, and it is uncommon in men under 40.

Men are diagnosed at an average age of 66. Bladder cancer is more common in older people.

Approximately 9 out of 10 people diagnosed with this cancer are over the age of 55. People are diagnosed at an average age of 73. Overall, men have a one in 27 chance of developing this cancer during their lifetime. Women have a chance of about 1 in 89 [1]. In the Western world,prostate cancer is the most common cancer in men aged 60 and up, while urothelial carcinomais more common in men aged 65 to 84, and is more common in men than women.

These cancers can appear as separate carcinomas, collision tumors, or tumors infiltrating the bladder or the prostate. A collision tumor is a rare but well-studied type of neoplastic lesion composed of two benign tumors, one benign and one malignant tumor, or two malignant tumors. Because of the close anatomic proximity of these organs, UC invasion into the prostate and vice versa is a common occurrence [3]. UC can affect the prostate by directly invading cancer cells into the prostatic stroma or intraductal extensions without invasion. Prostatic adenocarcinoma can involve the bladder either through metastasis or through direct extension,accounting for 12% of all UCs [2].

Diagnostic difficulties do not arise in well-differentiated PAC or even well-differentiated UCbecause the hematoxylin and eosin stain easily distinguish these tumors based on their common histologic features. The urothelial origin is suggested by the presence of surface neoplasia, nested growth, prominent nuclear pleomorphism, glassy eosinophilic cytoplasm, and high mitotic activity. Furthermore, squamous differentiation foci strongly suggest UC. PAC, on the other hand, is distinguished by predominantly acinar or cribriform architecture, minimal nuclear pleomorphism, nucleolar prominence, foamy and pale cytoplasm, and low mitotic activity [1].

Distinguishing poorly differentiated urothelial carcinoma from high-grade prostatic adenocarcinoma is a common challenge in genitourinary pathology, especially when the tumor involves the bladder neck, or prostatic urethra. Because of the morphologic overlap,hematoxylin and eosin staining are ineffective.

PSA is a serine protease found in the prostatic epithelium and seminal fluid that has remainedthe mainstay biomarker for prostate cancer diagnosis and management since its widespread use as a screening tool nearly 25 years ago. Although it has resulted in a significant increase in prostate cancer detection, PSA has significant drawbacks in terms of sensitivity and specificity, which is especially noticeable in high-grade adenocarcinomas. As the Gleason score rises, so does the drop in PSA sensitivity. According to immunohistochemistry, up to 13% of high-grade cancers are completely negative for PSA [4].

The distinction between poorly differentiated prostate cancer of the urinary bladder neck andhigh-grade urothelial carcinoma with prostatic extension has important therapeutic and staging implications. For example, cystoprostatectomy, the standard surgical procedure for the treatment of bladder cancer, is ineffective for prostatic cancer, and determining the tumor stagefor prognosis would necessitate correct diagnosis because the extension of bladder cancer intothe prostate, as well as prostate cancer into the bladder, would indicate pT4 disease. As a result,distinguishing them is critical to providing appropriate treatment [5].

Many studies have used immunohistochemistry to assess the use of various lineage markers in distinguishing urothelial carcinomas from prostate adenocarcinomas. In most cases, a panelof markers is useful in distinguishing between the two entities. Prostatic differentiation is supported by markers such as prostate-specific antigen (PSA), prostate-specific acidphosphatase (PSAP), prostate-specific membrane antigen (PSMA), P501s, NKX3.1, and erythroblast transformation specific-related gene (ERG); whereas urothelial differentiation andorigin are supported by markers such as high molecular weight cytokeratin (34βe12), CK7, p63,thrombomodulin, uroplakin III, GATA 3 [6, 7, 8, 9, 10, 11, 12]. Not all of these indicators are required in every case. It is best, to begin with, a few markers with high sensitivity and specificity and then add markers as needed. In the majority of cases, PSA, CK34βe12, and p63 are excellent starting points.

Prostate-specific membrane antigens include prostate marker protein (P501s) (prostein), prostate-specific membrane antigen (PSMA), and NKX3.1 [27].

P501s, a 553-amino acid protein found in the Golgi complex, is a newer prostate-specific protein discovered using high-throughput microarray screening and cDNA subtraction. Both benign and malignant prostatic epithelial cells contain P501s [2].

PSMA, a type II membrane glycoprotein containing 750 amino acids, is expressed by both benign and malignant prostatic epithelial cells, with malignant prostatic epithelial cells stainingmore strongly. PSMA is a highly specific marker of prostatic lineage, but it is also found in non-prostatic tissues such as the duodenum, neuroendocrine cells, endothelial cells in some neoplasms, and proximal renal tubules [2].

NKX3-1 is an androgen-regulated, prostate-specific homeobox gene with predominant expression in the prostate epithelium. It functions as a transcription factor and is essential for prostate development and tumor suppression. It inhibits the growth of epithelial cells in prostate tissue. The NKX3-1 gene encodes the NKX3-1 homeobox protein, which is also foundin urothelial cells, normal testis, lobular breast carcinoma, and bronchial mucous glands [2].

In prostate cancer, AMACR (alpha-Methylacyl-CoA Racemase) is consistently overexpressed compared to benign prostatic tissue. It codes for a cytoplasmic protein that participates in the β-oxidation of branched-chain fatty acids. AMACR is not specific to prostate cancer; it is also expressed by other cancers, most notably colorectal carcinomas and papillary renal cell carcinomas. The expression of AMACR is cytoplasmic, with a granular staining pattern. Apicalpredominance and heterogeneity are evident in the staining. Currently, AMACR is used to supplement basal cell markers in antibody cocktail formats. The average sensitivity for detecting limited prostate carcinoma in needle biopsies is 70-80%, with lower sensitivity reported in certain morphologic variants such as foamy, pseudohyperplastic, and atrophic variants of typical acinar prostate adenocarcinoma [13, 14, 15].

Urothelial markers include high molecular weight cytokeratin (HMWCK), p63, thrombomodulin,

S100P (placental S100), and GATA3.

HMWCK (34βe12) and p63 are more sensitive to high-grade urothelial cancer than novel markers like thrombomodulin and S100P [27]. HMCWCK (34βe12) is a highly sensitive urothelial lineage marker (CK), which also includes CK1, CK5, CK14, and CK20. It is only reactive against high-molecular-weight cytokeratins (CKs). It has the same sensitivity as p63 and is said to outperform uroplakin III and thrombomodulin [2].

p63, a tumor suppressor gene homolog, encodes at least six different proteins with various biologic functions, one of which is urothelial differentiation. With consistent diffuse nuclear positivity, p63 is a fairly sensitive and highly specific marker of urothelial carcinoma [2,16].

HMCWCK (34βe12) and p63 are basal cell markers in prostatic tissue and are typically absentin invasive prostatic adenocarcinoma.

Thrombomodulin, also known as CD141, is a surface glycoprotein that regulates intravascular coagulation and is expressed in a variety of tumors including mesothelioma, endothelial vascular tumors, squamous carcinomas, urothelial carcinomas, and various adenocarcinomas in both primary and metastatic settings. This marker's lack of specificity to urothelial differentiation limits its utility in this context. However, as demonstrated in several studies mentioned in this section, this marker can be useful in the workup of a potential urothelial tumor when used in conjunction with other markers [2, 17].

S100P is a protein from the S100 family discovered in the placenta and was thus named S100P(it is different from the S100 widely used in melanocytic and nerve sheath tumors). S100P expression by IHC has been described in benign and malignant urothelial cells, pancreatic carcinoma, esophageal squamous mucosa, and breast carcinoma, in addition to the placenta [2,11].

GATA3 is a transcription factor of the GATA family that regulates genes involved in the luminal differentiation of breast epithelium, genes involved in T-cell development, and genes involved in the development or maintenance of skin, trophoblasts, and some endothelial cells. GATA3 has been identified as an IHC marker for both primary and metastatic mammary and urothelial carcinomas. Despite the promising specificity and sensitivity, recent studies have shown that not all cases of prostatic adenocarcinoma can be positive for GATA3, posing a potential diagnostic challenge. McDonald, Timothy M recently represented nine cases of prostatic adenocarcinoma with aberrant positive GATA3 staining. All nine cases were PAC, with a Gleason grade of 5. GATA3 positivity was strong and diffuse in four cases, strong and patchy in two cases, and strong and focal in three cases. All of the patients tested positive for NKX3.1, six tested positive for p501s, and six tested positive for PSA, with seven of the nine cases expressing at least two prostate-specific markers. To avoid the diagnostic blunder, poorlydifferentiated carcinomas of the prostate, bladder neck, or trigone should be assessed not only with GATA3 but also with prostate-specific markers, according to the current research. GATA3 can still be useful in the workup of a neoplasm with a possible urothelial origin if usedin the right context and in the right conjunction with other antibodies [2, 18, 19, 20, 21, 22].

Uroplakins are widely considered to be urothelium-specific proteins of terminal urothelial cell differentiation, and they are positive in both primary and metastatic urothelial carcinoma. Despite being specific to urothelial differentiation, they are not very sensitive because some urothelial carcinomas do not express these markers, limiting their practical use and necessitating the inclusion of other markers in the workup for a potential urothelial tumor [23, 24, 25, 26].

Conclusion: High-grade prostatic adenocarcinoma and urothelial carcinoma of the urinary bladder can have ambiguous morphologic features that make a definitive diagnosis impossible.The distinction between these two tumors has important implications for staging and treatment. As a result, accurate diagnosis is critical for optimal patient care and may necessitate the use of highly sensitive immunohistochemical markers.

The review concluded that: 1) Prostatic and urothelial markers such as NKX3.1, p63, thrombomodulin, and GATA3 are very useful for distinguishing prostatic adenocarcinoma from urothelial carcinoma. 2) PSA is a good (clinical) screening tool, but because of its inverserelationship with tumor grade (the higher the grade, the lower the sensitivity of PSA staining), it is not recommended for high-grade tumor differentiation. 3) HMWCK (34βe12) and p63 aresaid to be more effective than thrombomodulin and S100p in detecting urothelial cancer. 4) Thrombomodulin is only moderately sensitive to UC. 5) Cytokeratins 7 and 20 can be positive in both UC and Prostatic adenocarcinoma, therefore their use is restricted.

The optimal combination of these immunohistochemical markers may improve the ability to distinguish PCA from UC.

Limitations: The immunohistochemical markers for urothelial carcinoma and prostatic adenocarcinoma are based on the research and clinical expertise of pathologists who have worked on cases and conducted studies to support their hypotheses. However, in a clinical scenario, the immunohistochemical panel is not absolute but depends on the individual case presented. This panel mentioned is solely based on research and functions to provide an overallunderstanding of the topic.

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Abbreviations:

PAC - Prostatic AdenoCarcinomaUC - Urothelial Carcinoma
WHO/ISUP – World Health Organization/the International Society of Urologic PathologistsPSA - Prostate-Specific Antigen
PSAP - Prostate-Specific Acid Phosphatase PSMA - Prostate-Specific Membrane Antigen
ERG - Erythroblast transformation specific-Related Gene
AMACR - Alpha-Methyl-Acyl-CoA Racemase
HMWCK - High Molecular Weight Cytokeratin

დიაგნოსტიკური სირთულეები მაღალი ხარისხის ავთვისებიანობის პროსტატის ადენოკარცინომისა და მაღალი ხარისხის ავთვისებიანობის უროთელური კარცინომის თანაარსებობისას - ლიტერატურის მიმოხილვა

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აბსტრაქტი: მამაკაცებში ორ ყველაზე გავრცელებულ სიმსივნეს მიეკუთვნება პროსტატის ადენოკარცინომა და გარდამავალუჯრედული, იგივე უროთელური კარცინომა. ორივე სიმსივნე შესაძლოა გამოვლინდეს ერთმანეთისაგან დამოუკიდებლად პროსტატაში ან შარდის ბუშტში ან ერთდროულად ცალკეულ სიმსივნეებად, რომლებიც ერთ-ერთ ორგანოშია გავრცელებული ან სპორადულად ე.წ. შერწყმული სიმსივნის სახით. პათოლოგანატომისათვის ამ სიმსივნეების ერთმანეთისაგან განსხვავება წარმოადგენს დიაგნოსტიკურ სირთულეს მაღალი ხარისხის ავთვისებიანობის მქონე ფორმების დროს, როდესაც ხდება სიმსივნის ირგვლივმდებარე ორგანოებში გავრცელება. აღნიშნული

სიმსივნეების განსხვავებული მკურნალობის ტაქტიკის გამო მნიშვნელოვანია მათი სწორი დიაგნოსტიკა.

ჩვენი მიმოხილვითი სტატიით შეკრებილია ინფორმაცია დიაგნოსტიკური სირთულეებისა და გადამწყვეტი იმუნოჰისტოქიმიური მარკერების შესახებ პროსტატის ადენოკარცინომისა და უროთელური კარცინომის სწორად დიფერენცირების მიზნით. ამ მიმოხილვაში გამოყენებულია PubMed და Google Scholar მონაცემთა ბაზების აკადემიურ ჟურნალებში გამოქვეყნებული სამეცნიერო ნაშრომები. მათგან მოპოვებულია პათოლოგანატომებისათვის მნიშვნელოვანი, აქტუალური და უახლესი ინფორმაცია.

წარმოდგენილი მიმოხილვით გამოვლინდა რომ: 1) პროსტატის ადენოკარცინომისა და უროთელური კარცინომის დიფერენცირების მიზნით ყველაზე ინფორმატიული იმუნოჰისტოქიმიური მარკერებია: NKX3.1, p63, თრომბომოდულინი და GATA3. 2) პროსტატის სპეციფიკური ანტიგენი (PSA) არის კარგი კლინიკური სასკრინინგო მარკერი, თუმცა მაღალი ხარისხის ავთვისებიანობის მქონე სიმსივნეებში ის კარგავს სენსიტიურობას (რაც უფრო მაღალია ავთვისებიანობის ხარისხი, იგივე გრეიდი, მით უფრო დაბალია PSA მგრძნობელობა/შეღებვის ინტენსივობა) და სწორად ვერ ადიფერენცირებს აღწერილ სიმსივნეებს ერთმანეთისაგან. 3) უროთელური კარცინომის გამოსავლენად HMWCK (34βe12) და p63 უფრო ეფექტურია, ვიდრე თრომბომოდულინი და S100p. 4) თრომბომოდულინი გამოირჩევა ზომიერი მგრძნობელობით უროთელური კარცინომის დიაგნოსტიკაში. 5) ციტოკერატინი 7 და 20 შესაძლოა დადებითი იყოს როგორც უროთელური კარცინომის, ასევე პროსტატის ადენოკარცინომის დროს, ამიტომ მათი გამოყენება ნაკლებად სასარგებლო ინფორმაციის მომცემია.

წარმოდგენილი მარკერების ოპტიმალური კომბინაციით შესაძლებელია გაუმჯობესებულ იქნას აღწერილი სიმსივნეების დიფერენციული დიაგნოსტიკა.

საძიებო სიტყვები: *პროსტატის ადენოკარცინომა, უროთელური კარცინომა, იმუნოჰისტოქიმია, პათოლოგიური დიაგნოზი, შერწყმული სიმსივნე*