2026 • Vol. 4, No. 1 • pp. 49-55
Trends in Pharmaceutical Biotechnology (TPB)

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Print ISSN: 3006-0516 Online ISSN: 3006-0354

Genomic Landscape, Biomarkers, and Precision Therapeutic Advances in Urinary Tract Cancers

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Access this article online

REVIEW ARTICLE
Received: 29.03.2026 **Revised:** 28.04.2026
Accepted: 03.06.2026
DOI: 10.57238/tpb.2026.153196.1007

OPEN ACCESS

ABSTRACT

Urinary tract malignancies are a heterogeneous group of tumors arising from the kidney, renal pelvis, ureter, bladder, and urethra. Urothelial (transitional-cell) carcinoma is the most common histological subtype and accounts for the majority of malignancies of the bladder and upper urinary tract. Despite advances in detection and therapy, advanced disease carries substantial risk of recurrence, progression, and death. Molecular profiling over the past decade has identified recurrent genomic alterations that shape tumor behavior, prognosis, and treatment response, including changes in TP53, the TERT promoter, FGFR3, KDM6A, PIK3CA, ERBB2, RB1, STAG2, ARID1A, and DNA-repair genes. These alterations increasingly inform precision-medicine strategies. Immune checkpoint inhibitors, FGFR-targeted agents, antibody–drug conjugates, and biomarker-guided treatment selection are reshaping management alongside conventional surgery and chemotherapy. This narrative review summarizes the molecular etiology, diagnostic approaches, and therapeutic advances in urinary tract cancers, with an emphasis on urothelial and bladder carcinoma — the subtype for which molecular and clinical evidence is most mature — while also addressing renal cell carcinoma and upper-tract urothelial carcinoma where relevant. The review highlights the growing role of genomic characterization in individualizing patient care and distinguishes findings that are well established from those that remain investigational.

Keywords: Biomarkers, Bladder Cancer, FGFR3, Immunotherapy, Precision Medicine, Targeted Therapy, Urinary Tract Cancer, Urothelial Carcinoma

1 Introduction

THIS is a narrative review rather than a systematic review. Relevant literature was identified through targeted searches of PubMed/MEDLINE and cBioPortal for Cancer Genomics, using combinations of the terms "urinary tract cancer," "urothelial carcinoma," "bladder cancer," "renal cell carcinoma," "FGFR3," "TERT promoter," "immune checkpoint inhibitor," "antibody–drug conjugate," and "circulating tumor DNA." The search was not date-restricted but prioritized landmark genomic studies (e.g., The Cancer Genome Atlas), pivotal clinical trials underpinning current regulatory approvals, and guideline documents from the European Association of Urology, supplemented with more recent publications through 2024. Because this is a narrative rather than systematic review, formal inclusion/exclusion criteria and risk-of-bias assessment were not applied; the selection reflects the authors' judgment of clinical and scientific relevance, which introduces the potential for selection bias inherent to this review format.

Urinary tract malignancies arise in the kidney, renal pelvis, ureters, urinary bladder, and urethra. The most clinically significant categories are renal cell carcinoma (RCC), upper-tract urothelial carcinoma (UTUC), and urothelial (bladder) carcinoma; urothelial carcinoma accounts for the great majority of bladder tumors and most tumors of the upper urinary tract [1]. These tumors are biologically heterogeneous, ranging from indolent superficial lesions with a favorable outcome to aggressive muscle-invasive and metastatic disease.

Bladder cancer is among the most common cancers worldwide and is characterized by a high recurrence rate that requires lifelong surveillance [2]. Early-stage disease is often curable, but progression to muscle-invasive bladder cancer (MIBC) substantially worsens prognosis. Genome sequencing has revealed that bladder malignancies are shaped by alterations in cell-cycle control, DNA repair, chromatin remodeling, and receptor tyrosine kinase signaling [3]. Large-scale genomic studies have further shown that urothelial cancers can be subdivided into molecular subtypes, each with distinct mutational patterns, pathway activation, and therapeutic vulnerabilities, supporting the use of molecular profiling in treatment selection and patient classification [4, 5].

Molecular subtyping, biomarker testing, and the development of new targeted agents have transformed bladder cancer care in recent years. Current approaches include immune checkpoint blockade, fibroblast growth factor receptor (FGFR) inhibitors, antibody–drug conjugates, and treatment selection guided by tumor genomic profile [6].

Urinary tract malignancies are a substantial contributor to the global cancer burden. Bladder cancer is the most frequent urinary tract malignancy, followed by kidney cancer and upper-tract urothelial carcinoma. Bladder malignancies are conventionally classified into three clinical categories:

- Non-muscle-invasive bladder cancer (NMIBC): tumors confined to the mucosa or lamina propria, including carcinoma in situ, Ta, and T1 lesions. NMIBC has a high recurrence rate but comparatively low mortality.
- Muscle-invasive bladder cancer (MIBC): a biologically aggressive disease state defined by invasion into the detrusor muscle, carrying a high risk of early hematogenous and lymphatic metastasis and typically requiring aggressive systemic therapy. Consensus transcriptional subtyping divides MIBC into five molecular subtypes — luminal papillary, luminal unstable, stroma-rich, basal-squamous, and neuroendocrine-like. Basal-squamous tumors tend to show high immune checkpoint expression and remain relatively responsive to neoadjuvant chemotherapy, whereas luminal papillary tumors are enriched for FGFR3 alterations, show relative chemoresistance, and are candidates for targeted therapy [4].
- Metastatic urothelial carcinoma: associated with poor survival and requiring systemic treatment with chemotherapy, immunotherapy, and/or targeted agents.

Upper-tract urothelial carcinoma (UTUC), affecting the renal pelvis and ureter, represents approximately 5–10% of urothelial cancers but frequently presents with invasive features [7]. Renal cell carcinoma is biologically distinct from urothelial cancer and includes clear-cell, papillary, and chromophobe subtypes; these tumors frequently involve alterations in hypoxia signaling, including the VHL pathway [8].

Table 1 summarizes the most frequently altered genes in bladder cancer based on publicly available datasets in cBioPortal for Cancer Genomics, including data from the cancer genome Atlas and other urothelial carcinoma cohorts [9]. These recurrent alterations provide insight into tumor biology and identify clinically relevant targets for precision therapy.

Recurrent alterations in telomere maintenance (TERT promoter), tumor suppression (TP53, RB1), receptor tyrosine kinase signaling (FGFR3, ERBB2), the PI3K pathway (PIK3CA), chromatin remodeling (KDM6A, ARID1A, STAG2), and DNA repair (ATM, BRCA1/2) collectively characterize the genomic landscape of urothelial carcinoma and provide a foundation for molecular classification and precision therapeutic approaches [10].

Among these alterations, FGFR3 is one of the most therapeutically relevant targets. FGFR3 mutations and fusions are enriched in low-grade, non-muscle-invasive tumors but also occur in advanced disease [10]. FGFR signaling promotes proliferation, survival, angiogenesis, and tumor growth; aberrant activation triggers downstream MAPK, PI3K/AKT, and STAT signaling, contributing to treatment resistance [11].

Mutations in TP53 and RB1 are common in muscle-invasive disease and are associated with aggressive clinical behavior. Loss of DNA-repair genes such as ATM, BRCA1, and BRCA2 contributes to genomic instability and, in some contexts, increased sensitivity to DNA-damaging therapy [12]. More broadly, defects in the DNA damage response can influence tumor response to platinum-based chemotherapy and immune checkpoint blockade, supporting their evaluation as predictive biomarkers [13].

Epigenetic dysregulation is also prominent in bladder cancer, reflected by frequent mutations in chromatin-remodeling genes such as KDM6A and ARID1A [10].

Table 1. The most frequently altered genes in bladder cancer.

Rank	Gene/Target	Approx. Alteration Frequency (TCGA/cBioPortal)	Core Biological & Epigenetic Function	Clinical Significance & Phenotypic Implications	Precision Therapeutics & Clinical Trial Settings
1	TERT promoter	50–80%	Telomerase reactivation; maintains telomere length, avoiding replicative senescence.	Early truncal driver event; a non-invasive urinary biomarker for early screening and surveillance.	Under evaluation for targeted telomerase inhibitors and cancer vaccines (active trials).
2	TP53	40–55%	Cell-cycle checkpoint control (G1/S) and DNA damage response initiation.	Enriched in muscle-invasive disease (MIBC); associated with aggressive behavior and metastatic risk.	Correlates with relative resistance to neoadjuvant cisplatin; predictive marker for combination checkpoint inhibition.
3	KDM6A	20–30%	Histone H3K27 demethylation; chromatin remodeling and transcriptional regulation.	Loss-of-function mutations impair differentiation; prevalent across bladder-cancer lineages.	Explored preclinically as a synthetic-lethal vulnerability to EZH2 inhibitors.
4	FGFR3	15–30%	Receptor tyrosine kinase signaling; drives MAPK/ERK and PI3K/AKT proliferation cascades.	Enriched in low-grade, non-muscle-invasive disease (NMIBC); promotes growth and treatment evasion.	Directly targetable by the FDA-approved pan-FGFR inhibitor erdafitinib in advanced/metastatic disease.
5	PIK3CA	15–20%	Catalytic subunit of the PI3K/AKT/mTOR survival and metabolism pathway.	Drives clonal expansion and metabolic adaptation; limits apoptosis.	Evaluated in combination strategies with pan-PI3K, AKT, and mTOR inhibitors.
6	ARID1A	15–20%	Core subunit of the SWI/SNF chromatin-remodeling complex.	Epigenetic deregulation; drives dedifferentiation and genomic instability.	Induces synthetic lethality; explored with ATR or PARP inhibitors in DNA-repair-deficient tumors.
7	STAG2	15–20%	Cohesin-complex component regulating sister-chromatid cohesion.	Inactivation drives aneuploidy and clonal heterogeneity.	Potential biomarker for sensitivity to microtubule-stabilizing agents and DDR-targeted therapy.
8	RB1	10–20%	G1/S checkpoint suppression via E2F inactivation.	Loss is coupled with high-grade, muscle-invasive progression and early metastasis.	Alters responsiveness to CDK4/6 inhibitors; associated with need for aggressive multi-agent chemotherapy.

Rank	Gene/Target	Approx. Alteration Frequency (TCGA/cBioPortal)	Core Biological & Epigenetic Function	Clinical Significance & Phenotypic Implications	Precision Therapeutics & Clinical Trial Settings
9	ERBB2 (HER2)	10–15%	EGFR-family receptor tyrosine kinase signaling.	Amplification/mutation defines an aggressive subset of advanced urothelial carcinoma.	Targetable by antibody–drug conjugates such as trastuzumab deruxtecan.
10	ATM/BRCA2	10–15%	Homologous recombination and double-strand DNA repair.	Loss increases mutational burden (TMB) and genomic instability.	Associated with sensitivity to platinum-based chemotherapy and PARP inhibitors.

Early and accurate diagnosis is central to improving outcomes in urinary tract cancers. Current diagnostic pathways combine clinical evaluation, imaging, endoscopy, histopathology, and molecular testing.

Urinalysis, urine cytology, and cystoscopy remain the standard first-line tests for suspected bladder cancer. Cystoscopy is the diagnostic gold standard, allowing direct visualization and biopsy, though it is invasive and has reduced sensitivity for flat lesions [14]. Urine cytology has high specificity for high-grade urothelial carcinoma but limited sensitivity for low-grade disease; molecular urine-based diagnostics have been developed to improve detection and surveillance [14].

Imaging modalities such as computed tomography urography (CTU) and magnetic resonance imaging are valuable for staging and for assessing upper urinary tract involvement; in muscle-invasive disease, imaging is important for identifying nodal and distant metastasis [15].

Genomic and molecular testing have improved early diagnosis and patient stratification. Next-generation sequencing panels allow simultaneous assessment of multiple genetic alterations and help identify patients suitable for targeted therapy [16]. Clinically relevant biomarker categories include:

- TERT promoter mutations: among the most common and earliest genetic events in bladder tumorigenesis; detectable in urine, they represent a promising non-invasive biomarker [17].
- FGFR3 alterations: mutations, fusions, and amplifications of FGFR3 are clinically actionable, and testing for FGFR3 status is now standard for patients who may benefit from FGFR-targeted therapy [18].
- DNA-repair alterations: alterations in homologous-recombination genes such as ATM, BRCA1, and BRCA2 can influence responsiveness to DNA-damaging therapy and immunotherapy [12].
- Immune markers: PD-L1 expression and tumor mutational burden (TMB) are used in some clinical settings to help predict response to checkpoint inhibitors, though their predictive value varies by patient, and integrated molecular testing is increasingly preferred. The tumor immune microenvironment and antigen-presentation pathways also contribute to the variability of checkpoint-inhibitor response [19].

Treatment selection depends on tumor stage, histological subtype, molecular alterations, and patient characteristics. Recent advances have shifted bladder cancer treatment from a uniform approach toward precision oncology.

Surgery remains the mainstay of therapy for localized disease. For bladder cancer, transurethral resection of bladder tumor (TURBT) serves both diagnostic and therapeutic roles in NMIBC, while radical cystectomy with lymph node dissection remains standard for selected patients with MIBC [6]. Partial or radical nephrectomy is performed for kidney cancer depending on tumor size, location, and patient factors.

Platinum-based chemotherapy has long been the standard treatment for advanced urothelial cancer. The combination of gemcitabine and cisplatin provides significant benefit and remains an important option for eligible patients [20], although many patients are ineligible for cisplatin because of renal impairment or comorbidities.

Immune checkpoint blockade has transformed the treatment of advanced urinary tract cancer. Major targets include PD-1, PD-L1, and CTLA-4. Agents directed against the PD-1/PD-L1 axis restore anti-tumor immune responses by counteracting tumor-mediated immune suppression [21], and checkpoint inhibitors have produced durable responses in a subset of patients with metastatic urothelial carcinoma, including those previously treated with chemotherapy [22].

FGFR alterations represent one of the clearest examples of precision medicine in bladder cancer. FGFR3 activation promotes tumor cell proliferation, survival signaling, migration, and angiogenesis. The development of selective FGFR tyrosine kinase inhibitors such as erdafitinib has

changed the treatment paradigm for patients with actionable FGFR3 mutations or fusions (e.g., FGFR3-TACC3) [18].

Acquired resistance remains a clinical challenge. Molecular profiling indicates that resistance is often mediated by secondary point mutations in the FGFR3 kinase domain — notably the gatekeeper mutation V555M and the molecular-brake mutations N540K and E466A — which impair drug binding or lock the receptor in a hyperactive conformation. Concomitant activation of bypass signaling through MET or EGFR can also allow tumor cells to escape FGFR inhibition, motivating multi-targeted combination strategies [23]. These findings underscore the importance of genomic profiling in guiding treatment selection, even as acquired resistance continues to limit durable benefit for some patients [18].

Antibody–drug conjugates combine targeted antibodies with cytotoxic payloads to enable selective delivery of anti-cancer agents. In urothelial carcinoma, the leading targets are Nectin-4 and HER2. Enfortumab vedotin, a Nectin-4-directed antibody–drug conjugate, has shown substantial activity in advanced urothelial cancer and represents a significant step toward biomarker-focused therapy [24]. It comprises a fully human monoclonal antibody against Nectin-4 — a cell-surface adhesion protein overexpressed in more than 90% of urothelial cancers — conjugated via a protease-cleavable linker to the microtubule-disrupting agent monomethyl auristatin E (MMAE). Following binding to Nectin-4, the conjugate is internalized and cleaved by lysosomal enzymes, releasing MMAE, which binds tubulin and induces G2/M cell-cycle arrest and apoptosis; the membrane-permeable MMAE payload also produces a bystander effect on neighboring antigen-negative tumor cells within a heterogeneous tumor. These agents have demonstrated meaningful activity in advanced urothelial cancer beyond that achieved with conventional chemotherapy (Table 2) [25].

Beyond conventional tissue biopsy, liquid biopsy platforms that detect circulating tumor DNA (ctDNA) represent a shift in how disease dynamics are monitored. In patients undergoing radical cystectomy for localized muscle-invasive disease, detection of post-operative ctDNA is a sensitive marker of minimal residual disease that can precede radiologic recurrence by several months; ctDNA-positive patients experience high rates of relapse and may be candidates for adjuvant immune checkpoint inhibition or antibody–drug conjugate therapy [26]. Serial ctDNA monitoring — tracking mutations such as TP53, FGFR3, or TERT promoter variants — may also help clinicians detect subclonal evolution and treatment-driven resistance, potentially enabling earlier therapeutic switches [26].

Artificial-intelligence-based imaging and molecular prediction models are emerging tools that may improve diagnosis and treatment selection. Integration of AI with genomic and clinical data has the potential to improve risk prediction and identify novel therapeutic targets, although these approaches remain largely investigational in urinary tract cancers and require prospective validation before routine clinical use [27].

The strength of evidence varies considerably across the biomarkers and therapies discussed in this review. FGFR3-targeted therapy, PD-1/PD-L1 checkpoint inhibition, and Nectin-4-directed antibody–drug conjugates are supported by phase 3 randomized trial data and are incorporated into regulatory approvals and clinical guidelines. In contrast, ctDNA-based surveillance, AI-based imaging and prediction models, and several proposed epigenetic and synthetic-lethality strategies remain investigational, with supporting data drawn largely from single-arm or retrospective studies. Drug resistance, tumor heterogeneity, and the need for robust, prospectively validated biomarkers remain among the principal unresolved challenges in the field.

Table 2.

Diagnostic/Therapeutic Approach	Clinical Application	Clinical Value
Cystoscopy	Bladder tumor detection	Gold-standard diagnostic method
CT urography	Staging and upper-tract imaging	Detects upper urinary tract disease
Urine-based biomarkers	Surveillance	Non-invasive monitoring
Genomic sequencing	Mutation profiling	Guides precision therapy selection
Platinum-based chemotherapy	Advanced/metastatic disease	Historic standard of tumor control
Immune checkpoint inhibitors	Metastatic disease	Durable responses in a subset of patients
FGFR inhibitors	FGFR3/2-altered tumors	Targeted treatment option
Antibody–drug conjugates	Advanced urothelial carcinoma	Emerging biomarker-directed option

Conflict of Interest: The author declares no conflict of interest.
Financing: The study was performed without external funding.
Ethical consideration: The study was approved by University of Maisan, Amarah, Iraq.

REFERENCES

[1] Siegel RL, Kratzer TB, Giaquinto AN, Sung H, Jemal A. Cancer statistics, 2025. *CA Cancer J Clin*. 2025;75(1):10-45. doi: 10.3322/caac.21871.

[2] Lenis AT, Lec PM, Chamie K, Mshs M. Bladder cancer: a review. *JAMA*. 2020;324(19):1980-91. doi: 10.001/jama.2020.17598.

[3] Robertson AG, Kim J, Al-Ahmadie H, Bellmunt J, Guo G, Cherniack AD, et al. Comprehensive molecular characterization of muscle-invasive bladder cancer. *Cell*. 2017;171(3):540-56.E25. doi: 10.1016/j.cell.2017.09.007.

[4] Kamoun A, de Reynies A, Allory Y, Sjödahl G, Robertson AG, Seiler R, et al. A consensus molecular classification of muscle-invasive bladder cancer. *Eur Urol*. 2020;77(4):420-33. doi: 10.1016/j.eururo.2019.09.006.

[5] The Cancer Genome Atlas Research Network. Comprehensive molecular characterization of urothelial bladder carcinoma. *Nature*. 2014;507:315-22. doi: 10.1038/nature12965.

[6] Witjes JA, Bruins HM, Cathomas R, Compérat EM, Cowan NC, Gakis G, et al. European Association of Urology guidelines on muscle-invasive and metastatic bladder cancer: summary of the 2020 guidelines. *Eur Urol*. 2021;79(1):82-104. doi: 10.1016/j.eururo.2020.03.055.

[7] Roupret M, Babjuk M, Burger M, Capoun O, Cohen D, Comperat EM, et al. European Association of Urology guidelines on upper urinary tract urothelial carcinoma: 2020 update. *Eur Urol*. 2021;79:62-79. doi: 10.1016/j.eururo.2020.05.042.

[8] Linehan WM, Srinivasan R, Schmidt LS. The genetic basis of kidney cancer: a metabolic disease. *Nat Rev Urol*. 2010;7:277-85. doi: 10.1038/nrurol.2010.47.

[9] Cerami E, Gao J, Dogrusoz U, Gross BE, Sumer SO, Aksoy BA, et al. The cBio cancer genomics portal: an open platform for exploring multidimensional cancer genomics data. *Cancer Discov*. 2012;2(5):401-4. doi: 10.1158/2159-8290.CD-12-0095.

[10] Knowles MA, Hurst CD. Molecular biology of bladder cancer: new insights into pathogenesis and clinical diversity. *Nat Rev Cancer*. 2015;15:25-41. doi: 10.1038/nrc3817.

[11] Babina IS, Turner NC. Advances and challenges in targeting FGFR signalling in cancer. *Nat Rev Cancer*. 2016;17(5):318-32. doi: 10.1038/nrc.2017.8.

[12] Lord CJ, Ashworth A. The DNA damage response and cancer therapy. *Nature*. 2012;481(7381):287-94. doi: 10.1038/nature10760.

[13] Teo MY, Seier K, Ostrovnaya I, Regazzi AM, Kania BE, Moran MM, et al. Alterations in DNA damage response and repair genes as potential marker of clinical benefit from PD-1/PD-L1 blockade in advanced urothelial cancers. *J Clin Oncol*. 2018;36(17):1685-94. doi: 10.200/JCO.2017.75.7740.

[14] Babjuk M, Burger M, Capoun O, Cohen D, Compérat EM, Escrig JLD, et al. European Association of Urology guidelines on non–muscle-invasive bladder cancer (Ta, T1, and carcinoma in situ). *Eur Urol*. 2022;81(1):75-94. doi: 10.1016/j.eururo.2021.08.010.

[15] Cowan NC. CT urography for hematuria. *Nat Rev Urol*. 2012;9:218-26. doi: 10.1038/nrurol.2012.32.

[16] Rose KM, Huelster HL, Meeks JJ, Faltas BM, Sonpavde GP, Lerner SP, et al. Circulating and urinary tumour DNA in urothelial carcinoma—upper tract, lower tract and metastatic disease. *Nat Rev Urol*. 2023;20:406-19. doi: 10.1038/s41585-023-00725-2.

[17] Kinde I, Munari E, Faraj SF, Hruban RH, Schoenberg M, Bivalacqua T, et al. *TERT* promoter mutations occur early in urothelial neoplasia and are biomarkers of early disease and disease recurrence in urine. *Cancer Res*. 2013;73(24):7162-7. doi: 10.1158/0008-5472.CAN-13-2498.

[18] Loriot Y, Necchi A, Park SH, Garcia-Donas J, Huddart R, Burgess E, et al. Erdafitinib in locally advanced or metastatic urothelial carcinoma. *N Engl J Med*. 2019;381(4):338-48. doi: 10.1056/NEJMoa1817323.

[19] Mariathasan S, Turley SJ, Nickles D, Castiglioni A, Yuen K, Wang Y, et al. TGFβ attenuates tumour response to PD-L1 blockade by contributing to exclusion of T cells. *Nature*. 2018;554:544-8. doi: 10.1038/nature25501.

[20] von der Maase H, Hansen S, Roberts J, Dogliotti L, Oliver T, Moore M, et al. Gemcitabine and cisplatin versus methotrexate, vinblastine, doxorubicin, and cisplatin in advanced or metastatic bladder cancer: results of a large, randomized, multinational, multicenter, phase III study. *J Clin Oncol*. 2000;18(17):3068-77. doi: 10.1200/JCO.2000.18.17.3068.

[21] Bellmunt Molins J, Bajorin DF, Investigators K-. Pembrolizumab as Second-Line Therapy for Advanced Urothelial Carcinoma. *N Engl J Med*. 2017;376(11):1015-26. doi: 10.56/NEJMoa1613683.

[22] Powles T, Csőszi T, Özgüroğlu M, Matsubara N, Géczi L, Cheng SY, et al. Pembrolizumab alone or combined with chemotherapy versus chemotherapy

as first-line therapy for advanced urothelial carcinoma (KEYNOTE-361): a randomised, open-label, phase 3 trial. *Lancet Oncol*. 2021;22(7):931-45. doi: 10.1016/S470-2045(21)00152-2.

[23] Ibrahim T, Gizzi M, Bahleda R, Loriot Y. Clinical development of FGFR3 inhibitors for the treatment of urothelial cancer. *Bladder Cancer*. 2019;5(2):87-102. doi: 10.3233/BLC-180205.

[24] Powles T, Rosenberg JE, Sonpavde GP, Loriot Y, Durán I, Lee J-L, et al. Enfortumab vedotin in previously treated advanced urothelial carcinoma. *N Engl J Med*. 2021;384(12):1125-35. doi: 10.056/NEJMoa2035807.

[25] Rosenberg JE, O'Donnell PH, Balar AV, McGregor BA, Heath EI, Yu EY, et al. Pivotal trial of enfortumab vedotin in urothelial carcinoma after platinum and anti-programmed death 1/programmed death ligand 1 therapy. *J Clin Oncol*. 2019;37(29):2592-600. doi: 10.1200/JCO.19.01140.

[26] Christensen E, Birkenkamp-Demtröder K, Sethi H, Shchegrova S, Salari R, Nordentoft I, et al. Early detection of metastatic relapse and monitoring of therapeutic efficacy by ultra-deep sequencing of plasma cell-free DNA in patients with urothelial bladder carcinoma. *J Clin Oncol*. 2019;37(18):1547-57. doi: 10.200/JCO.18.02052.

[27] Bi WL, Hosny A, Schabath MB, Giger ML, Birkbak NJ, Mehrtash A, et al. Artificial intelligence in cancer imaging: clinical challenges and applications. *CA Cancer J Clin*. 2019;69(2):127-57. doi: 10.3322/caac.21552.

How to cite this article

Salih S.L.; Al-Omary T.S.S.; Al-hraishawi H.; Genomic Landscape, Biomarkers, and Precision Therapeutic Advances in Urinary Tract Cancers. Trends in Pharmaceutical Biotechnology (TPB). 2026;4(1):49-55. doi: 10.57238/tpb.2026.153196.1007