Arch Iran Med. March 2026;29(3):181-185

doi:10.34172/aim.35506

http://journalaim.com

ARCHIVES OF IRANIAN MEDICINE

Review Article

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HER2: Biology and Testing in Gastrointestinal, Genitourinary, and Gynecologic Malignancies

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Abstract

This article explores the role of HER2 as an oncogenic driver across multiple cancer types, with a specific focus on gastrointestinal, genitourinary, and gynecologic malignancies. HER2, a member of the ErbB receptor family, is implicated in cell growth, survival, and oncogenesis through its activation of downstream signaling pathways such as MAPK and PI3K/AKT. Aberrant HER2 activation, through gene amplification or activating mutations, is a well-established contributor to the aggressive biological behavior of breast and gastric cancers. Recent studies have expanded the relevance of HER2 beyond breast and gastric cancers to include colorectal, urothelial, and gynecologic cancers, making it a valuable therapeutic target in these settings. This review highlights the molecular mechanisms behind HER2 activation, its clinical significance, and current testing methodologies. The article discusses the evolution of HER2 testing, including IHC, ISH, and NGS approaches, and examines their role in guiding personalized therapy. The challenges of implementing standardized HER2 testing across various cancer types are also considered. Finally, the review examines the clinical outcomes associated with HER2-targeted therapies and discusses future directions in HER2 research, including the integration of HER2 with immune checkpoint inhibitors and other novel treatments.

Keywords: Gastrointestinal cancers, HER2 signaling, HER2 testing, Targeted therapy

Cite this article as: Ben Kridis W, Khanfir A. HER2: biology and testing in gastrointestinal, genitourinary, and gynecologic malignancies. Arch Iran Med 2026;29(3):181-185. doi:10.34172/aim.35506

Received: November 18, 2025, **Accepted:** February 15, 2026, **ePublished:** March 1, 2026

Introduction

The *human epidermal growth factor receptor 2* (HER2, also known as *ERBB2*) is a member of the ErbB family of receptor tyrosine kinases, which also includes EGFR (HER1), HER3, and HER4. In normal physiology, HER2 regulates cellular proliferation, differentiation, and survival through ligand-independent dimerization or heterodimerization with other ErbB receptors, activating downstream RAS/MAPK and PI3K/AKT pathways.[1,2]

Aberrant activation of HER2 (by gene amplification, protein overexpression, or activating mutations) has been well established as a key oncogenic driver in breast and gastric cancers, where it confers aggressive biological behavior and serves as a target for monoclonal antibodies and antibody–drug conjugates.[3–5] Over the past decade, HER2 has emerged as a clinically relevant biomarker across several other malignancies, including gastrointestinal (GI), genitourinary (GU), and gynecologic (GYN) tumors.[6–8]

In GI cancers, HER2 overexpression and amplification rates vary by tumor site and histology. In gastric and gastroesophageal junction adenocarcinomas, HER2 positivity occurs in approximately 15–22% of cases.[9,10] The landmark ToGA trial demonstrated that the addition of trastuzumab to chemotherapy significantly improved survival in HER2-positive advanced gastric cancer, establishing HER2 as a predictive biomarker in this setting.[11] In colorectal cancer, HER2 amplification (present in ~2–5% of cases) is associated with resistance to anti-EGFR therapy and represents a potential target in refractory disease.[12,13]

In the GU tract, HER2 alterations are being actively explored, particularly in urothelial carcinoma, where HER2 amplification or overexpression occurs in 6–20% of cases depending on detection methods.[14,15] Early clinical data suggest potential benefit from HER2-directed therapies, including trastuzumab deruxtecan and disitamab vedotin.[16]

Similarly, in gynecologic malignancies, HER2 is increasingly recognized beyond endometrial serous carcinoma, where overexpression is seen in up to 30% of tumors.[17] Recent studies have reported HER2 amplification or mutation in subsets of ovarian and cervical cancers, expanding the relevance of HER2-targeted therapies to these entities.[18,19] The tumor-agnostic approval of trastuzumab deruxtecan for HER2-positive metastatic solid tumors further underscores the need for standardized HER2 testing across organ systems.[20]

Given the biological heterogeneity and varying testing methodologies across these cancer types, a comprehensive understanding of HER2 biology, diagnostic evaluation, and clinical relevance is essential. This review will summarize the molecular underpinnings of HER2 activation, discuss the current landscape of HER2 testing in GI, GU, and GYN malignancies, and highlight therapeutic advances and future directions in this rapidly evolving field.

HER2 Biology

The *human epidermal growth factor receptor 2* (HER2,

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ERBB2) belongs to the *ErbB* family of receptor tyrosine kinases, which includes EGFR (*ERBB1*), HER3 (*ERBB3*), and HER4 (*ERBB4*).[1]

Each receptor comprises three functional domains: an extracellularligand-bindingregion,asingletransmembrane helix, and an intracellular tyrosine kinase domain.[2] Unlike other *ErbB* members, HER2 has no known ligand but adopts a conformation that favors spontaneous dimerization, making it the preferred partner for HER3, whose potent activation of PI3K/AKT signaling contributes to oncogenic transformation.[3,4]

Following dimerization, autophosphorylation of HER2's intracellular tyrosine residues activates several downstream signaling cascades, including the RAS–RAF–MEK–ERK (MAPK) and PI3K–AKT–mTOR pathways, which regulate cell growth, proliferation, and survival (Figure 1).[5,6] Secondary routes (such as JAK–STAT and PLCγ–PKC) further influence motility and differentiation.[5] Through these interconnected networks, HER2 functions as a central regulator of epithelial biology and a potent oncogenic driver when dysregulated.[7]

HER2-driven tumorigenesis arises through gene amplification, protein overexpression, or activating mutations (Figure 2).[8–10] Amplification on chromosome 17q12 leads to excessive receptor expression, resulting in constitutive signaling independent of ligand binding.[9]

HER2 protein overexpression may also occur due to transcriptional or post-translational dysregulation and is typically detected by immunohistochemistry (IHC). Activating mutations, commonly in the kinase or extracellular domains (e.g. L755S, S310F, V777L), can drive oncogenesis in tumors without amplification, as shown in breast, colorectal, lung, and gynecologic cancers.[10–12]

Beyond intrinsic signaling, HER2 profoundly affects the tumor microenvironment (TME) by promoting angiogenesis via VEGF upregulation and enhancing epithelial–mesenchymal transition (EMT), thereby facilitating invasion and metastasis.[13,14] HER2 activation can also increase PD-L1 expression and remodel immune infiltration, supporting the rationale for combining HER2-targeted and immune checkpoint inhibitors.[15]

HER2 biology exhibits context-specific diversity across tumor types. In breast and gastric cancers, HER2 amplification often yields strong, diffuse membranous expression. In contrast, colorectal, urothelial, and gynecologic tumors frequently harbor heterogeneous, subclonal, or mutation-driven alterations.[16–18] This biological heterogeneity underpins organ-specific variations in testing standards and therapeutic response.

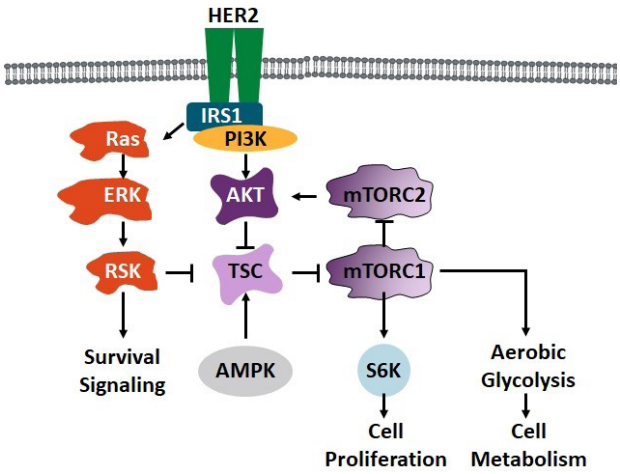

Figure 1. HER2 Receptor Signaling Pathway [5, 6]

HER2 Testing

Evolution of HER2 Testing

Accurate determination of HER2 status is critical for identifying candidates for HER2-targeted therapy across solid tumors. Since HER2 amplification was first validated as a predictive biomarker in breast cancer, diagnostic approaches have evolved from single-modality assays to integrated, multimodal platforms combining IHC, *in-situ* hybridization (ISH), and next-generation sequencing (NGS).[7,19,20] IHC remains the frontline technique for assessing HER2 protein expression, whereas ISH confirms gene amplification in equivocal cases (IHC 2+).

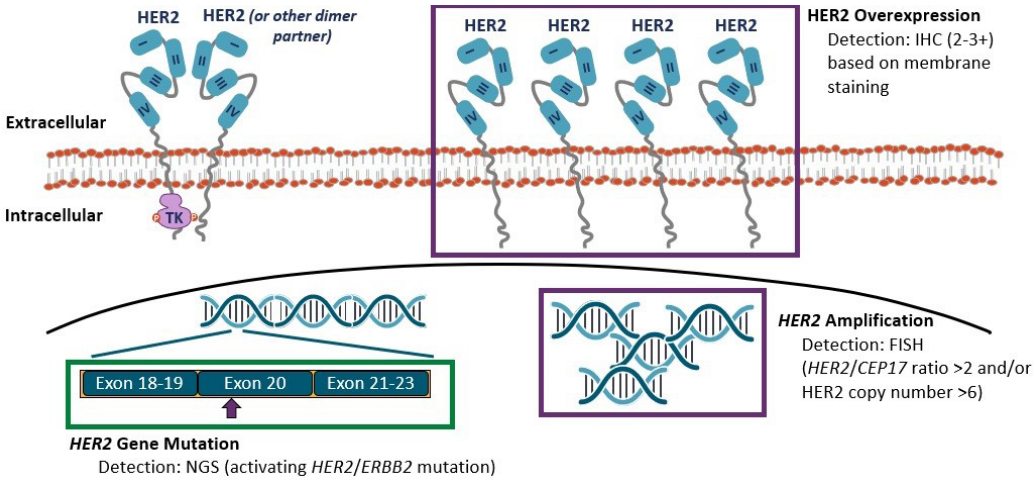

Figure 2. Differences in HER2 Alterations.[8, 10, 12]

With the advent of genomic profiling, HER2 mutations and copy-number changes are increasingly recognized as clinically relevant alterations, expanding the concept of HER2 positivity beyond overexpression (Figure 2).[10,12]

HER2 Testing in Gastrointestinal Malignancies

Gastric and Gastroesophageal Junction Cancers

HER2 testing is standard of care in gastric and gastroesophageal junction (GEJ) cancers, as amplification predicts benefit from trastuzumab-based therapy.[21] However, HER2 expression in these tumors is often heterogeneous and basolateral, differing substantially from the diffuse membranous staining seen in breast carcinoma.[7,22] The *ToGA* trial established the clinical value of HER2 testing in this context, leading to the development of gastric-specific scoring criteria endorsed by CAP and ASCO.[21,23] Tumors are classified as IHC 3+ (strong complete or basolateral staining in ≥ 10% of cells), IHC 2+ (equivocal, requiring ISH confirmation), or IHC 0/1+ (negative).

Recent *DESTINY-Gastric01* and *DESTINY-Gastric04* trials demonstrated that trastuzumab deruxtecan benefits patients even with low or heterogeneous HER2 expression, highlighting the need to refine scoring and recognize the HER2-low spectrum (Table 1) .[24,25]

Colorectal and Pancreatic Cancers

HER2 amplification occurs in approximately 2–5% of metastatic colorectal cancers (mCRC), particularly within the RAS/BRAF wild-type subset, and is associated with resistance to anti-EGFR therapy.[26]

The *HERACLES* and *MyPathway* studies confirmed HER2 as a therapeutic target in this setting, supporting dual IHC/ISH testing in RAS wild-type, refractory mCRC.[27,28] Although rare in pancreatic and biliary cancers (< 2%), HER2 amplification or activating mutations may define small subsets responsive to HER2-directed therapies identified through genomic profiling.[29]

HER2 Testing in Genitourinary Malignancies

HER2 dysregulation is reported in approximately 5–10% of urothelial carcinomas, especially in micropapillary variants.[17] While no standardized scoring algorithm exists for these tumors, preliminary data suggest clinical benefit from HER2-directed antibody–drug conjugates (ADCs) such as trastuzumab deruxtecan.[30,31] HER2 alterations in renal cell carcinoma and prostate cancer are uncommon, though emerging NGS data have identified rare activating *ERBB2* mutations in advanced, treatment-resistant prostate cancer.[32]

HER2 Testing in Gynecologic Malignancies

Endometrial Cancer

HER2 overexpression and amplification are enriched in uterine serous carcinoma (USC), where adding trastuzumab to carboplatin–paclitaxel significantly improves survival.[33] Given the distinct histopathologic features of endometrial tissues, CAP guidelines recommend organ-specific scoring, defining HER2 positivity as complete or lateral membranous staining in ≥ 30% of tumor cells, with ISH testing required for equivocal cases.[34,35] HER2 testing is increasingly advocated in all high-grade endometrial carcinomas, including carcinosarcoma, supported by emerging clinical evidence (Table 2).[36]

Ovarian and Cervical Cancers

HER2 amplification is rare in high-grade serous ovarian cancer (< 3%) but occurs more frequently in mucinous and clear cell subtypes, where targeted therapy may be beneficial. [37,38] In cervical adenocarcinomas, HER2 alterations occur in 3–5% of cases and are associated with aggressive behavior, as shown in TCGA analyses.[39]

Technical and Interpretative Considerations

Outside the breast and gastric settings, HER2 testing faces major challenges, including tumor heterogeneity, fixation variability, and organ-specific staining patterns.[23] Pathologists must interpret IHC and ISH results within histologic context, considering membranous completeness, intensity, and distribution. Emerging

Table 1. Summary of HER2 Testing Across Cancer Types

Cancer Type	HER2 Overexpression/Amplification Rate	Diagnostic Method	Clinical Relevance
Gastric and GEJ Cancers	15–22%	IHC, ISH	Predicts benefit from trastuzumab-based therapy
Colorectal Cancer	2–5%	IHC, ISH	Potential therapeutic target in refractory disease
Urothelial Cancer	6–20%	IHC, ISH, NGS	Potential benefit from ADCs like trastuzumab deruxtecan
Endometrial Cancer	Up to 30%	IHC, ISH	Improves survival in uterine serous carcinoma with trastuzumab
Ovarian Cancer	< 3% in high-grade serous, higher in mucinous/clear cell subtypes	IHC, ISH, NGS	Targeted therapy potential in mucinous and clear cell types

Table 2. HER2 Testing Methodologies

Method	Description	Strengths	Limitations	Clinical Application
IHC (Immunohistochemistry)	Detects protein expression on cell membrane	Quick, widely available	Limited sensitivity, subject to inter-observer variability	Screening for HER2 positivity in many cancers
ISH (In Situ Hybridization)	Detects gene amplification by fluorescent or chromogenic probes	Accurate, reliable for ambiguous IHC 2+	Requires more technical expertise, slower	Confirmation of IHC results, especially in gastric cancer
NGS (Next-Generation Sequencing)	Detects mutations and amplifications at the genomic level	Comprehensive, can detect mutations	Expensive, requires specialized equipment	Used for identifying HER2 mutations and co-alterations in advanced cancers

digital pathology and AI-based image analysis platforms have shown promise in improving reproducibility, while NGS-based approaches allow simultaneous detection of amplification, mutation, and co-alterations such as *PIK3CA* or *TP53* mutations that may influence therapeutic response.[12,40] The evolving recognition of HER2-low and HER2-mutant states underscores the need for a molecularly integrated, continuum-based definition of HER2 positivity in precision oncology.

Conclusion

In conclusion, HER2 has emerged as a pivotal biomarker in oncology, transcending its initial association with breast cancer to play a critical role in gastrointestinal, genitourinary, and gynecologic malignancies. The molecular mechanisms underlying HER2 activation (through gene amplification, protein overexpression, and activating mutations) have been well-documented and are central to its oncogenic potential. As our understanding of HER2's role in tumor biology deepens, the diagnostic methodologies for detecting HER2 alterations, including IHC, ISH, and NGS, have evolved, allowing for more precise identification of patients who could benefit from HER2-targeted therapies. Although challenges remain, particularly in standardization of testing and management of heterogeneous tumor expression, the integration of HER2-directed treatments has significantly improved clinical outcomes in HER2-positive cancers. Future research should focus on refining testing approaches, exploring combination therapies, and investigating the potential of HER2-targeted therapies across a broader range of cancers. The ongoing development of personalized treatment strategies promises to enhance the efficacy of HER2-based therapies, ultimately improving survival and quality of life for patients across multiple cancer types.

Authors' Contribution

Conceptualization: Wala Ben Kridis
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Visualization: Afef Khanfir
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Competing Interests

None declared.

Funding

The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Provenance and Peer Review

Not commissioned; internally peer reviewed.

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