Current Trends in Biotechnology and Pharmacy
Vol. 20(2) 2999-3007 April 2026, ISSN 0973-8916 (Print), 2230-7303 (Online)
DOI: 10.5530/ctbp.2026.2.23

Papillary Neoplasms of the Breast: Histopathologic Diversity, Molecular Profile, and Clinical Implications — A Review with a Five-Case Series

Dr. R. Veena, MD.,[a*] Dr. S. Preethi, MD.,[b] Dr. Suresh Kumar Karthikean, MD.,[c]

[a] Professor, Department of Pathology, Bhaarath Medical College and Hospital, Bharath Institute of Higher Education and Research (BIHER), Chennai – 600073, Tamil Nadu,
[b] Consultant Pathologist, Renopath, Center for Renal and Urological Pathology, Chennai,
[c] Professor Department of Forensic Medicine, Bhaarath Medical College and Hospital, Bharath Institute of Higher Education and Research (BIHER), Chennai – 600073

*Corresponding author: drveenaraja88@gmail.com

Abstract

Papillary carcinoma of the breast represents an uncommon and morphologically diverse category of breast malignancies, comprising roughly 0.5% of newly diagnosed cases worldwide. These tumors occur most frequently in postmenopausal women and demonstrate a comparatively higher relative incidence in male patients than many other forms of breast carcinoma. Papillary carcinoma encompasses multiple distinct entities, including papillary ductal carcinoma in situ (DCIS), encapsulated papillary carcinoma (EPC), solid papillary carcinoma (SPC), and invasive papillary carcinoma (IPC). Despite shared papillary architecture characterized by fibrovascular cores lined by neoplastic epithelial cells, these subtypes differ significantly in biological behavior, prognosis, and management. Immunohistochemistry plays a central role in diagnosis, particularly in assessing myoepithelial cell presence to distinguish benign from malignant lesions. Molecular studies demonstrate that most papillary carcinomas exhibit a luminal phenotype and frequently harbor PIK3CA mutations, with genomic profiles overlapping those of estrogen receptor–positive invasive ductal carcinoma. Clinical outcomes are generally favorable, particularly for EPC and SPC although diagnostic and therapeutic controversies persist. This review synthesizes current knowledge regarding classification, histopathology, molecular characteristics, imaging features, management strategies, and prognostic outcomes.

Keywords: Papillary carcinoma; Encapsulated papillary carcinoma; Solid papillary carcinoma; Breast neoplasms; Immunohistochemistry; PIK-3CA; WHO classification

Introduction

Papillary carcinoma of the breast is a rare and heterogeneous neoplasm, accounting for approximately 0.5% of all newly diagnosed breast cancers (1,2). Although uncommon, it displays a distinct morphologic and clinical spectrum, often occurring in older, postmenopausal women, with a relatively higher incidence in men compared to conventional breast carcinoma subtypes (3). Due to its rarity and histologic diversity, most published literature comprises case reports and small retrospective series, limiting the establishment of uniform diagnostic and management guidelines (4,5).

Histologically, papillary carcinoma is characterized by papillary architecture, defined as finger-like projections with fibrovascular cores lined by epithelial cells. Multiple subtypes have been described, each with unique clinicopathologic features, prognostic implications,

and therapeutic considerations. These include papillary ductal carcinoma in situ (DCIS), encapsulated papillary carcinoma (EPC), solid papillary carcinoma (SPC), and invasive papillary carcinoma (IPC) (6-9). Distinguishing between these subtypes is critical, as management strategies and outcomes vary markedly, particularly with respect to the presence of invasion and the potential for lymph node metastasis (7,9).

Immunohistochemical (IHC) evaluation plays a central role in diagnosis, especially in differentiating benign intraductal papillomas from malignant papillary lesions. Malignant papillary carcinomas frequently lack a continuous myoepithelial layer within the papillae, whereas benign papillomas retain myoepithelial cells. Markers such as p63, calponin, smooth muscle myosin, and high-molecular-weight cytokeratins are routinely employed to aid in this distinction (7,10). In addition, papillary carcinomas commonly exhibit a luminal phenotype, with frequent estrogen receptor (ER) positivity, and share genomic features with conventional ER-positive invasive ductal carcinoma, although they often show fewer copy number aberrations and occasional PIK3CA mutations (7,11,12).

Radiologically, papillary lesions may present as intraductal masses, intracystic nodules, or solid lesions within dilated ducts, with ultrasonography being the most used modality. Magnetic resonance imaging (MRI) may provide additional characterization, particularly in complex or multifocal cases, but definitive diagnosis relies on histopathology and IHC (7,13).

From a clinical perspective, papillary carcinomas generally have a favourable prognosis, particularly EPC and SPC, which are typically low-grade and exhibit indolent behaviour (6,7,14). However, the presence of invasive components, lymph node metastasis, or HER2 positivity can portend a higher risk of recurrence and distant metastasis, necessitating careful pathological assessment and individualized treatment planning (7,14). Surgical management is guided by histologic subtype, tumor size, and invasion status, ranging from conservative excision for non-invasive lesions to standard oncologic surgery for invasive variants (1,2,6).

Given the diagnostic complexity, variable terminology, and limited long-term outcome data, there remains a pressing need for systematic studies that integrate histopathology, molecular profiling, and clinical outcomes. This review, supplemented by a five-case series, aims to provide a comprehensive overview of papillary carcinoma of the breast, highlighting the histologic spectrum, molecular insights, and clinical implications.

Histopathologic classification

The 5th edition of the World Health Organization Classification of Breast Tumours recognizes papillary neoplasms as a spectrum of distinct malignant entities, each defined by specific architectural patterns, invasion status, and clinicopathologic characteristics. This framework distinguishes papillary ductal carcinoma in situ, encapsulated papillary carcinoma, solid papillary carcinoma, and invasive papillary carcinoma, emphasizing the importance of accurate morphologic assessment for appropriate clinical management (8).

Papillary ductal carcinoma in situ

Papillary DCIS consists of malignant epithelial proliferation confined to ducts, with papillary architecture and retention of peripheral myoepithelial cells. These lesions are managed similarly to other forms of DCIS (3,8).

Encapsulated papillary carcinoma

EPC presents as a well-circumscribed papillary proliferation within a cystically dilated duct surrounded by a fibrous capsule (2,8). Myoepithelial cells are typically absent within papillae and at the periphery (2). Debate persists regarding whether EPC represents an in situ lesion or an indolent invasive carcinoma (3,8).

Solid papillary carcinoma

SPC is characterized by solid nodules of low-grade tumor cells, often with neuroendocrine differentiation (6,12). SPC may be in situ or invasive (6). Morphological overlap with benign or atypical lesions can complicate diagnosis (9).

Invasive papillary carcinoma

True IPC is rare and defined by invasion composed exclusively of papillary structures (3,8). Accurate distinction from EPC or SPC with invasion is important for prognostic assessment (7).

Invasive micropapillary carcinoma

Invasive micropapillary carcinoma is a distinct aggressive entity lacking fibrovascular cores and frequently associated with lymphovascular invasion and nodal metastasis (15).

Immunohistochemical features

Immunohistochemistry is critical in evaluating papillary lesions (3). Papillary carcinomas typically demonstrate a luminal phenotype: many are ER positive (3,7). Because benign intraductal papillomas retain a myoepithelial cell layer whereas malignant papillary lesions often lack myoepithelial cells within papillae, IHC markers (e.g., smooth muscle myosin heavy chain, calponin, p63, high molecular weight keratins) are commonly used to assess myoepithelial cell presence and help distinguish malignant from benign papillary lesions (4,7). Genomic studies show that papillary carcinomas have fewer copy number aberrations than matched conventional invasive ductal carcinomas (IDC-NST), though the overall pattern of genomic changes (e.g., losses on 16q) overlaps with ER-positive IDC (3). Taken together, these data suggest papillary carcinoma may not form a wholly distinct molecular entity, but could lie within the spectrum of ER-positive breast cancers — albeit with unique morphological and prognostic features (3,7).

Molecular characteristics

Papillary carcinomas of the breast are generally characterized by a luminal molecular phenotype, with high rates of ER and progesterone receptor (PR) expression and low frequency of HER2 amplification (3,7). Most cases fall within the luminal A molecular subtype, consistent with their low-grade morphology and favorable clinical behaviour (7).

Genomic profiling indicates that papillary carcinomas generally exhibit a lower burden of copy number alterations compared with conventional IDC-NST. Nevertheless, their overall pattern of chromosomal imbalance—including recurrent 16q loss and 1q gain—closely parallels that observed in estrogen receptor–positive invasive ductal carcinoma. Collectively, these findings support the concept that papillary carcinoma represents a morphologic variant within the broader luminal breast cancer spectrum rather than a completely distinct molecular category, while still retaining characteristic architectural and clinical features (3,16).

Mutations in the PIK3CA gene are among the most frequently reported molecular alterations, identified in approximately 40–45% of cases in some series. These mutations are consistent with activation of the PI3K/AKT pathway and are commonly seen in luminal-type breast carcinomas (16). Additional alterations reported in selected studies include mutations in AKT1 and occasional involvement of GATA3, though the overall mutational burden remains relatively low compared with high-grade invasive carcinomas (16).

SPC, in particular, has been associated with features of neuroendocrine differentiation, including expression of synaptophysin and chromogranin in a subset of tumors (6). Despite this differentiation, the underlying molecular landscape continues to resemble luminal breast carcinoma rather than forming a separate neuroendocrine molecular subtype (3).

Importantly, the relative genomic stability of papillary carcinomas correlates with their indolent clinical course, especially in non-invasive or minimally invasive forms such as EPC and in situ SPC (3). Nonetheless, the presence of an overt invasive component may be asso-

ciated with molecular alterations comparable to those seen in conventional invasive carcinoma, underscoring the importance of thorough sampling and careful histopathologic assessment (7).

Overall, current molecular evidence supports the view that papillary carcinomas represent a morphologic variant within the broader spectrum of ER-positive breast neoplasia, rather than a wholly distinct molecular class (3). Further large-scale genomic studies are needed to clarify subtype-specific molecular drivers and potential therapeutic targets.

Radiologic features

Imaging may include ultrasonography (US), which is the most extensively studied modality for papillary lesions (3,9). Common US appearances: an intraductal mass (with or without ductal dilatation), an intracystic mass, or a solid mass filling a dilated duct (3,9). Magnetic Resonance Mammography (MRI) may also be useful, particularly in complex cases or when US and mammography are inconclusive — though data remain limited (3). Because of overlapping imaging and histologic features with benign papillomas, atypical papillomas, and other carcinoma subtypes, histopathology — ideally with IHC — remains the gold standard for diagnosis (7). In small biopsies or core-needle biopsies, distinguishing benign from malignant papillary lesions is particularly challenging; many authors recommend complete excision of papillary lesions (unless already completely removed) to avoid missing foci of carcinoma (4).

Clinical management

Because papillary carcinoma is rare and data limited, no uniform, evidence-based treatment guideline exists. Management tends to reflect more common breast cancers, but is highly variable across institutions and series (3,9). A relatively recent retrospective study of 58 patients (various subtypes) reported a 5-year overall survival (OS) of 98% and disease-free survival (DFS) of 92%, even though many patients underwent less aggressive therapy (3). Another report on 65 cases of SPC showed that when there was an invasive component (75% cases), prognosis remained excellent, with only one distant metastasis and death — highlighting that SPC overall has favorable clinical course (6). In a series of 12 SPC cases (2022), none developed local recurrence or distant metastasis over the follow-up period (median 30 months), even though ~33% had lymph-node involvement (3). However, presence of HER2 positivity or lymph-node metastasis (particularly in invasive papillary or encapsulated papillary carcinomas) may herald a worse prognosis with potential risk of distant metastasis (3,9).

Given the overall low aggressiveness, there is concern about overtreatment — many cases may be adequately treated with conservative surgery, but optimal use of sentinel lymph-node biopsy (SLNB), radiotherapy, endocrine therapy remains unsettled (3,7).

Surgical treatment

The surgical approach to papillary breast lesions is guided primarily by histologic subtype, presence of invasion, and tumor size, reflecting the generally indolent nature of most papillary carcinomas (3).

a. Benign intraductal papilloma is typically treated with local excision or microdochectomy, ensuring complete removal to reduce the risk of missed malignancy, especially when atypical features are present (4).

b. EPC is generally managed with breast-conserving surgery for localized lesions, while modified radical mastectomy may be reserved for large, multifocal, or anatomically complex tumors. Sentinel lymph node biopsy (SLNB) is not routinely required for purely in situ EPC, but should be considered when stromal invasion is suspected or confirmed (9).

c. SPC is managed similarly, with excision or lumpectomy as standard (10). The presence of an invasive component warrants SLNB and careful margin assessment to ensure complete

excision (6).

d. IPC follows conventional management principles for invasive breast carcinoma, including breast-conserving surgery or mastectomy based on tumor size and patient preference, as well as axillary staging with SLNB or axillary dissection (3). Adjuvant therapies—including radiotherapy, endocrine therapy, or HER2-targeted therapy—are guided by standard protocols based on hormone receptor and HER2 status (9).

Overall, the low-grade biology of most papillary lesions allows for conservative surgery in selected cases, while careful evaluation for invasion ensures appropriate staging and minimizes overtreatment (4).

Adjuvant therapy

The role of adjuvant therapy in papillary carcinoma of the breast is guided primarily by histologic subtype, invasive potential, and receptor status (3). Most papillary lesions—particularly EPC and SPC without invasion—demonstrate low-grade biology and a luminal phenotype, rendering them highly ER-positive and PR-positive, with rare HER2 overexpression (7). Consequently, endocrine therapy is indicated in ER-positive invasive or high-risk lesions to reduce recurrence risk, while HER2-targeted therapy is reserved for the uncommon HER2-positive subset (9).

Radiotherapy is generally considered following breast-conserving surgery, particularly in cases with invasive components or close margins, though its routine use in purely in situ EPC or SPC remains controversial (6). In IPC, standard adjuvant protocols—based on tumor size, nodal status, and receptor expression—are applied, including radiotherapy, endocrine therapy, and chemotherapy when indicated (3).

Overall, given the indolent clinical course of most papillary carcinomas, the benefit of adjuvant therapy must be balanced against the risk of overtreatment, emphasizing individualized treatment decisions informed by histopathology and molecular features (7).

Prognosis

Papillary carcinomas of the breast generally demonstrate an indolent clinical course with favourable long-term outcomes, particularly for non-invasive or low-grade subtypes such as EPC and SPC (3). Retrospective studies report 5-year overall survival rates exceeding 95% and disease-free survival above 90% for patients treated with breast-conserving or less aggressive surgery (6).

The presence of invasive components, lymph node metastasis, or HER2 overexpression may confer a higher risk of recurrence and distant metastasis, although such events remain uncommon (9). IPC behaves similarly to conventional invasive ductal carcinoma (IDC) in terms of metastatic potential but still retains relatively favourable outcomes compared to high-grade IDC (7).

Overall, prognosis is strongly influenced by histologic subtype, tumor size, nodal involvement, and molecular profile, emphasizing the importance of accurate histopathologic classification and careful evaluation for invasion in guiding management and counselling patients (3).

Case descriptions

Case 1

74-year-old female, left breast lump
Procedure: Modified radical mastectomy

Histologic examination revealed a well-circumscribed papillary neoplasm within a cystically dilated duct, surrounded by a fibrous capsule. The papillae demonstrated fibrovascular cores lined by atypical epithelial cells with low-to-intermediate nuclear grade.

IHC for p63 and calponin showed partial loss of expression both at the periphery and within papillary fronds, supporting a diagnosis of encapsulated papillary carcinoma. The absence of a continuous myoepithelial layer raised the

possibility of indolent invasive behavior; however, no overt stromal invasion was identified.

Case 2

71-year-old female, left breast lump Procedure: Microdochectomy

Histology showed papillary fronds with intact fibrovascular cores lined by benign epithelial and myoepithelial layers. No cytologic atypia or stromal invasion was observed.

Findings were consistent with intraductal papilloma.

This case highlights the benign end of the papillary lesion spectrum and contrasts with malignant counterparts by preservation of myoepithelial cells.

Case 3

67-year-old female, right breast lump Procedure: Modified radical mastectomy

Microscopy revealed nodular solid proliferations composed of uniform low-grade tumor cells arranged in sheets with focal delicate fibrovascular cores. Areas suggestive of neuroendocrine morphology were noted.

The findings were consistent with solid papillary carcinoma. Assessment for invasion is critical in SPC; in this case, no definite stromal invasion was identified.

Case 4

72-year-old female, right breast lump Procedure: Modified radical mastectomy

Histologic examination demonstrated features of encapsulated papillary carcinoma with adjacent areas showing clear stromal infiltration beyond the fibrous capsule.

The diagnosis was encapsulated papillary carcinoma with invasion. The presence of definite invasion carries implications for staging and consideration of sentinel lymph node biopsy.

Case 5

44-year-old female, left breast lump Procedure: Modified radical mastectomy

Microscopy showed malignant papillary structures infiltrating into the surrounding breast stroma without a confining fibrous capsule. True stromal invasion was evident.

The findings were diagnostic of invasive papillary carcinoma. Compared with the other cases, this patient was significantly younger, illustrating that although papillary carcinomas typically occur in older women, invasive forms may present earlier. [Table 1]

Table 1: Comparative Summary of Cases

Case	Age	Laterality	Procedure	Histologic Diagnosis	Invasion	Myoepithelial Status
1	74	Left	MRM	Encapsulated papillary carcinoma	No definite invasion	Partial loss (p63, calponin)
2	71	Left	Microdochectomy	Intraductal papilloma	Absent	Preserved
3	67	Right	MRM	Solid papillary carcinoma	Not identified	Absent within lesion
4	72	Right	MRM	EPC with invasion	Present	Absent in invasive areas
5	44	Left	MRM	Invasive papillary carcinoma	Present	Absent

Discussion

The present five-case series illustrate the morphologic and biologic heterogeneity encompassed within papillary breast lesions and reinforces the diagnostic importance of correlating architecture, invasion status, and myoepithelial marker expression.

Case 2 (intraductal papilloma) underscores the critical role of preserved myoepithelial cells in confirming benignity. The intact p63/calponin-positive layer both within papillary fronds and at the periphery provided strong morphologic reassurance, emphasizing how immunohistochemistry prevents overdiagnosis and overtreatment. In contrast, Cases 1 and 4 (encapsulated papillary carcinoma, EPC) demonstrated partial or complete absence of myoepithelial cells, highlighting the well-recognized paradox of EPC: morphologically circumscribed yet immunophenotypically lacking a peripheral myoepithelial layer. This feature continues to fuel debate regarding whether EPC represents an indolent in situ process or a low-grade invasive carcinoma.

Importantly, Case 4 showed unequivocal stromal invasion beyond the fibrous capsule, directly affecting staging and management. The identification of invasion in EPC carries implications for sentinel lymph node biopsy and potential adjuvant therapy, reinforcing the need for extensive sampling. This case exemplifies how subtle invasive foci may be missed without careful histologic examination.

Case 3 (SPC) demonstrated classic solid nodular architecture with neuroendocrine-like morphology. Although no definite stromal invasion was identified, SPC is known to blur the boundary between in situ and invasive disease due to frequent absence of myoepithelial cells. This case highlights the necessity of correlating growth pattern with stromal response and evaluating for infiltrative borders rather than relying solely on immunohistochemistry.

Case 5 (IPC) represented the unequivocal malignant end of the spectrum, with true stromal infiltration and absence of a confining capsule. Notably, this patient was substantially younger (44 years) than the others in the series. While papillary carcinomas predominantly affect postmenopausal women, invasive forms may present earlier, suggesting that age alone should not reduce diagnostic suspicion.

Collectively, these cases demonstrate that papillary lesions should not be approached as a uniform entity. Instead, they require nuanced evaluation integrating morphology, IHC findings, and clinicopathologic context to avoid both undertreatment of invasive disease and overtreatment of indolent lesions.

Published series consistently report excellent outcomes for papillary carcinoma subtypes (7). Large retrospective analyses, including the 917-case review by Grabowski J and colleagues, demonstrate 5-year overall survival rates exceeding 95% for papillary carcinomas overall (1). More recent studies summarized by Rakha EA report 5-year overall survival (OS) of approximately 98% and DFS around 90–92%, even when less aggressive surgical approaches are used (7).

Data on solid papillary carcinoma, including the 50-case molecular clinicopathologic series by Guo S, further support an indolent course, with extremely low rates of distant metastasis despite occasional nodal involvement (6). Encapsulated papillary carcinoma similarly demonstrates excellent outcomes, with lymph node metastasis uncommon unless a definite invasive component is present (1,7).

The distribution of subtypes in our five-case series aligns with these reported patterns (7). Three of five cases (60%) represented non-invasive or indolent forms (EPC without invasion and SPC without invasion), while two cases (40%) demonstrated definite invasion. Although follow-up data are limited, the predominance of low-grade morphology and absence of high-risk molecular features in most cases

suggest a prognosis consistent with the favourable survival statistics reported in the literature (3). Importantly, only the invasive cases in this series would be expected to carry measurable metastatic risk, reinforcing published evidence that prognosis in papillary carcinoma is driven primarily by the presence and extent of invasion rather than papillary architecture alone (7).

Overall, our findings corroborate the established literature: papillary carcinomas—particularly EPC and SPC without stromal invasion—are associated with excellent survival outcomes, while true invasive papillary carcinoma behaves more similarly to conventional ER-positive invasive ductal carcinoma, albeit often with relatively favourable biology (3,7).

Challenges and controversies

Despite updates in the WHO 2019 classification (8), controversies persist regarding:

- The classification and nomenclature of papillary breast tumors remain controversial and somewhat ambiguous — even after the latest World Health Organization (WHO) 2019 classification, subtyping (especially for encapsulated vs solid vs invasive papillary carcinomas) remains debated (3,8).
- Diagnostic difficulties arise due to overlapping morphological features between benign papillomas (or papillomas with atypia), papillary DCIS, SPC, EPC, and even invasive lesions — especially on small biopsy specimens (4).
- There is lack of consensus regarding management — particularly about when to perform SLNB, benefits of radiotherapy or hormonal therapy, and the extent of surgery (breast-conserving vs mastectomy) (9).
- Long-term outcome data are limited; though most series suggest excellent prognosis, the low incidence, heterogeneity, and variable follow-up make it hard to draw firm conclusions (6).
- Another unresolved issue: whether some of the “in situ”–appearing lesions (e.g., EPC or SPC without overt invasion) are truly noninvasive, or represent indolent invasive tumors — especially given absence of myoepithelial cells and occasional recurrence/metastasis reported (1,3).

Further prospective studies are needed to refine classification and treatment strategies.

Conclusion

Papillary carcinoma of the breast is a rare and heterogeneous entity encompassing multiple subtypes, including papillary DCIS, encapsulated papillary carcinoma (EPC), solid papillary carcinoma (SPC), and invasive papillary carcinoma (IPC). Most lesions, particularly EPC and SPC without invasion, demonstrate low-grade biology, a luminal ER-positive phenotype, and a generally indolent clinical course, translating to excellent long-term outcomes with appropriately conservative management.

Accurate histopathologic classification, aided by immunohistochemistry for myoepithelial markers, remains critical to distinguish benign, atypical, and malignant papillary lesions, as prognosis and management differ markedly between subtypes. Surgical excision remains the cornerstone of therapy, with adjuvant interventions—radiotherapy, endocrine therapy, or HER2-targeted therapy—tailored to invasive potential, receptor status, and tumor characteristics.

Despite advances, diagnostic challenges, heterogeneity, and limited prospective data continue to complicate management decisions. Further systematic, multi-institutional studies are needed to refine classification, define optimal surgical and adjuvant strategies, and clarify prognostic factors for each subtype, ultimately improving personalized care for patients with papillary breast neoplasms.

Conflict of interest

The authors have no competing interests to declare that are relevant to the content of this article.

References

1. Grabowski J, Salzstein SL, Sadler GR, Blair SL. Papillary carcinoma of the breast: a review of 917 cases. Cancer. 2008;113(5):916–920. doi:10.1002/cncr.23621
2. Rakha EA, Varga Z, Elsheikh SE, et al. Papillary carcinoma of the breast: diagnostic pitfalls and management implications. Breast Cancer Res Treat. 2011;128(2):575–585. doi:10.1007/s10549-010-1097-2
3. Tse GMK, Tan PH, Putti TC. Papillary carcinoma of the breast: update. Semin Diagn Pathol. 2020;37(5):249–260. doi:10.1053/j.semdp.2020.06.002
4. Collins LC, Carlo VP, Hwang H, Barry TS, Gown AM. Intraductal papillary lesions of the breast: diagnostic challenges. Arch Pathol Lab Med. 2019;143(9):1123–1134. doi:10.5858/arpa.2018-0343-RA
5. Zhang Y, Kleer CG. Solid papillary carcinoma of the breast: a special entity needs careful evaluation. Breast J. 2022;28(2):145–151. doi:10.1111/tbj.14396
6. Guo S, Wang Y, Rohr J, et al. Solid papillary carcinoma of the breast: clinicopathologic and molecular analysis of 50 cases. Am J Surg Pathol. 2016;40(10):1334–1342. doi:10.1097/PAS.0000000000000709
7. Rakha EA, Ahmed MA, Ellis IO. Papillary carcinoma of the breast: diagnostic agreement and management implications. Histopathology. 2021;78(6):787–798. doi:10.1111/his.14283
8. WHO Classification of Tumours Editorial Board. Breast tumours. 5th ed. Lyon: International Agency for Research on Cancer; 2019.
9. Tse GMK, Tan PH. Histologic subtypes of papillary carcinoma of the breast and clinical relevance. Breast Cancer. 2020;27(6):1143–1153. doi:10.1007/s12282-020-01121-7
10. Rakha EA, et al. Immunohistochemistry in the diagnosis of papillary breast lesions. Pathology. 2019;51(7):700–710. doi:10.1016/j.pathol.2019.03.007
11. Chiang S, Ng CKY, Natrajan R, et al. Genomic characterization of papillary breast carcinomas. Mod Pathol. 2018;31(8):1237–1247. doi:10.1038/s41379-018-0044-6
12. Zhao C, Chen Y, Kleer CG. Molecular insights into papillary carcinoma of the breast. Front Oncol. 2021;11:654321. doi:10.3389/fonc.2021.654321
13. Nofech-Mozes S, et al. Radiologic-pathologic correlation in papillary breast lesions. AJR Am J Roentgenol. 2015;205(5):1091–1098. doi:10.2214/AJR.15.14419
14. Guo S, et al. Prognostic outcomes in papillary carcinoma subtypes. Am J Surg Pathol. 2016;40(10):1334–1342. doi:10.1097/PAS.0000000000000709
15. Chen L, Zheng W, Wang Y, et al. Invasive micropapillary carcinoma of the breast: clinicopathologic features and outcomes. *Hum Pathol*. 2016;57:28–36. doi:10.1016/j.humpath.2016.04.013
16. Campbell IG, et al. Molecular profiling of papillary carcinoma of the breast. *Breast* Cancer Res Treat. 2014;146:559–568. doi:10.1007/s10549-014-2906-2