Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

Original Article Open Access

# Phyllodes Tumors of the Breast: Classification, Molecular Insights, and Pathological Best Practices – A Comprehensive Narrative Review

Chandrakant Halder[1],[2] Navneet Parashar[1], Manoj Kumar Gupta[1], Dayaram Meena[1]

ARTICLE INFO

Article history:
Received 1 October 2025
Received in revised form
28 October 2025
Accepted 7 November 2025
Available online 15 November 2025

Keywords:
Drug-drug interactions
Pharmacokinetic
Pharmacodynamic
Polypharmacy
Precipitant drugs

ABSTRACT

Phyllodes tumors (PTs) are rare biphasic fibroepithelial neoplasms of the breast exhibiting a biological spectrum from benign to malignant, with significant implications for local recurrence and distant metastasis. This narrative review integrates contemporary evidence on epidemiology, pathogenesis, clinical presentation, radiological features, histopathological classification, immunohistochemical profiles, molecular alterations, differential diagnosis, and therapeutic strategies. PTs account for 0.3–1% of all breast tumors, predominantly affecting women aged 40–55 years, with higher incidence in East Asian populations. Molecular studies reveal early MED12 and RARA mutations shared with fibroadenomas, while TERT promoter mutations and progressive TP53, PIK3CA, and EGFR alterations drive malignant transformation. The WHO 5th edition classification remains the cornerstone, but refined diagnostic criteria (RDC) significantly improve prediction of metastatic potential by reclassifying up to 24% of borderline tumors as malignant. Core needle biopsy limitations and interobserver variability underscore the need for extensive surgical sampling and ancillary molecular testing. Wide local excision with ≥1 cm margins is standard; adjuvant radiotherapy reduces recurrence in high-risk cases, whereas chemotherapy shows limited efficacy. Emerging tools—including artificial intelligence–assisted imaging, 16-gene expression panels, and targeted therapies—promise enhanced diagnostic precision and personalized management. This review consolidates current best practices and highlights future directions for optimizing outcomes in this challenging neoplasm.

## INTRODUCTION

Phyllodes tumors (PTs) of the breast represent a fascinating yet challenging group of biphasic fibroepithelial neoplasms that continue to intrigue pathologists, surgeons, oncologists, and molecular biologists alike.

First described by Johannes Müller in 1838 as “cystosarcoma phyllodes” due to their fleshy, leaf-like appearance on cut section, these tumors have since been recognized as a spectrum ranging from clinically indolent benign lesions to highly aggressive malignant sarcomas capable of distant hematogenous metastasis [1,3].

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

They constitute only 0.3–1% of all breast tumors and 2–3% of fibroepithelial neoplasms, yet their unpredictable behavior, high recurrence rates, and diagnostic pitfalls make them disproportionately significant in clinical practice [1,3,15].

Unlike the vastly more common fibroadenoma, which is a benign, self-limited proliferation of stromal and epithelial elements typically seen in young women, PTs demonstrate true neoplastic stromal clonality with secondary distortion of benign ductal epithelium into characteristic leaf-like (phyllodes) fronds [4,8]. This stromal dominance is the biological hallmark that distinguishes PTs and underpins their capacity for local recurrence (10–50%) and, in malignant cases, lethal metastasis (up to 50%) primarily to lungs, bones, and rarely liver or brain [2,5,6,22].

The diagnostic challenge lies in the morphological continuum between cellular fibroadenomas and low-grade PTs, the subjective nature of histological grading, and the limited reliability of core needle biopsy (CNB) sampling in heterogeneous tumors [10,29]. Interobserver variability among expert breast pathologists can reach 20–30%, particularly in assessing stromal cellularity, atypia, and mitotic activity [10]. The World Health Organization (WHO) 5th edition (2019) classification remains the international gold standard, stratifying PTs into benign, borderline, and malignant categories based on five key stromal parameters [4,21]. However, emerging evidence supporting refined diagnostic criteria (RDC), molecular biomarkers, and artificial intelligence-assisted diagnostics is reshaping contemporary practice [7,13,20].

This narrative review synthesizes the extensive body of literature on PTs, integrating classical histopathological concepts with modern genomic, immunohistochemical, and clinical insights to provide a comprehensive framework for accurate diagnosis, risk stratification, and management.

## Epidemiology and Demographic Patterns

PTs predominantly affect women, with male cases representing less than 0.1% of reported series and almost invariably associated with gynecomastia or hormonal imbalance [15]. The mean age at diagnosis ranges from 40 to 55 years, approximately 15–20 years older than the typical fibroadenoma patient (20–30 years), suggesting a perimenopausal hormonal milieu that may promote stromal proliferation [1,16]. Pediatric and adolescent cases are exceptionally rare (<5% of total) but clinically significant due to their frequently rapid growth and heightened stromal cellularity, which can lead to overdiagnosis of malignancy if adult criteria are rigidly applied [11].

Geographic and ethnic variations are well documented. In East Asian populations, particularly Singapore, Malaysia, and China, PTs comprise up to 4–7% of all breast tumors, compared to 0.3–1% in Western cohorts [17]. Whether this reflects true genetic predisposition, differences in screening intensity, or environmental factors remains unresolved, though MED12 mutation prevalence appears similar across ethnicities [9,19]. Most PTs are sporadic, but germline TP53 mutations in Li-Fraumeni syndrome unequivocally increase risk, warranting genetic counseling in young patients or those with suggestive personal or family history of sarcomas, brain tumors, or adrenocortical carcinoma [18].

Rare associations with prior breast irradiation or trauma have been reported anecdotally, but no definitive environmental triggers have been confirmed [1,23]. Bilateral PTs occur in less than 1% of cases, and multifocal disease within a single breast is seen in approximately 5% [15].

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

## Etiology and Molecular Pathogenesis

The pathogenesis of PTs centers on clonal stromal proliferation that secondarily distorts polyclonal epithelial elements into exaggerated intracanalicular patterns [8,9]. Next-generation sequencing has revolutionized our understanding of this process, revealing a stepwise genomic evolution model. Early driver events include MED12 exon 2 hotspot mutations (30–65% of cases), identical to those seen in 60–70% of fibroadenomas, particularly those with fibroadenoma-like areas within PTs [9,19]. This shared molecular signature strongly suggests that a subset of PTs arise through malignant transformation of pre-existing fibroadenomas, a hypothesis supported by cases demonstrating morphological transition zones [9].

Co-occurring RARA mutations (10–40%) implicate dysregulated retinoic acid signaling and estrogen responsiveness, consistent with the perimenopausal age peak [8]. TERT promoter mutations emerge as a critical branch point, present in 45–60% of PTs but less than 10% of fibroadenomas, conferring telomerase reactivation and immortalization [7,20]. The high sensitivity (94%) of TERT mutations for distinguishing PTs from fibroadenomas on limited biopsy material has positioned it as a valuable diagnostic adjunct [20].

Progression to borderline and malignant phenotypes is marked by accumulation of additional genomic alterations. TP53 mutations or protein overexpression occur in 40–60% of malignant PTs but are rare in benign or borderline tumors [8,21]. PIK3CA (10–30%), EGFR (10–30%), and NF1 mutations activate oncogenic signaling pathways, while copy number variations—particularly gain of 1q and loss of 13q—escalate with grade, reflecting increasing genomic instability [8,22]. A landmark case of a borderline PT correctly classified on CNB due to TERT mutation detection avoided unnecessary mastectomy that would have been performed if diagnosed as fibroadenoma [20].

## Clinical Presentation

Patients typically present with a painless, firm, well-circumscribed, mobile unilateral mass averaging 4–5 cm at diagnosis—significantly larger than the 1–2 cm typical fibroadenoma [1,3]. Rapid growth over weeks to months, reported in 30–40% of cases, is a cardinal clinical feature that should prompt urgent evaluation [2,16]. Skin ulceration, fixation to chest wall, or bluish discoloration reflects locally advanced malignant tumors with high stromal cellularity and necrosis.

Rare but dramatic paraneoplastic syndromes include non-islet cell tumor hypoglycemia (NICTH) secondary to tumoral IGF-2 overexpression, documented in at least 17 cases [24,25]. Some patients develop refractory hypoglycemia with hypertrophic osteoarthropathy, particularly in the setting of pulmonary metastases [26]. Nipple discharge is exceptionally rare (n=3 reported cases), and inflammatory changes are uncommon. Reactive axillary lymphadenopathy occurs in less than 5% of cases; true lymphatic metastasis is virtually nonexistent (<1%), reinforcing the hematogenous dissemination pattern of malignant PTs [4,5].

## Radiological Features

Mammography typically reveals a round or oval, well-circumscribed, lobulated mass with occasional indistinct margins in 20–30% of malignant PTs suggesting infiltration [14,27]. Coarse calcifications are rare (<10%), distinguishing PTs from mucinous or papillary carcinomas [3]. Ultrasound demonstrates a hypoechoic solid mass with internal cystic spaces corresponding to epithelial-lined clefts, posterior acoustic enhancement, and parallel orientation to the skin—the classic “leaf-like” appearance [1,14].

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

MRI is the most sensitive modality for assessing tumor extent, demonstrating heterogeneous T2 hyperintensity, rapid initial enhancement with washout, and internal septations [27]. However, specificity remains limited (50–60%) for distinguishing PTs from cellular fibroadenomas or even phyllodes-like metaplastic carcinomas [14]. Diffusion-weighted imaging with apparent diffusion coefficient (ADC) mapping and shear-wave elastography show promise in identifying malignant PTs through restricted diffusion and increased stiffness, but standardized thresholds are lacking [13,14]. A meta-analysis of ultrasound features confirmed low sensitivity (30%) of spiculated margins for malignancy, emphasizing the need for histological confirmation [14].

Macroscopic Pathology

Grossly, PTs are bosselated, pseudo-encapsulated masses with a characteristic whorled, fleshy cut surface resembling brain tissue. Cystic spaces filled with mucoid material correspond to dilated epithelial clefts, while hemorrhage, necrosis, or soft friable areas suggest high-grade malignancy [1,3,4,28]. Tumor size varies widely (1–40 cm), with mean diameters increasing with grade: benign (3–4 cm), borderline (5–7 cm), malignant (8–10 cm or larger) [15].

Adequate sampling is critical: current guidelines recommend 1–2 blocks per centimeter of maximal diameter, with additional sections from hemorrhagic, necrotic, or heterogeneous areas [10,29]. A striking example involved a 7 cm tumor requiring 14 blocks to identify focal malignant heterologous osteosarcomatous differentiation missed on initial sampling, altering management from observation to wide excision [29].

Classification Systems

WHO 5th Edition (2019) classifies PTs based on a composite assessment of five stromal parameters: marked atypia, marked cellularity, ≥5 mitoses/mm$^2$, stromal overgrowth, and permeative borders, plus malignant heterologous elements [4,21]. Tumors lacking these features are benign (60–80%), those with intermediate characteristics are borderline (10–20%), and those meeting multiple adverse criteria are malignant (10–20%). Recurrence rates are 10–15% (benign), 15–20% (borderline), and 25–29% (malignant), with metastatic risk <1%, 5–10%, and 30–50% respectively [2,6,22]. Rare cases of initially benign PTs metastasizing after recurrence as malignant tumors highlight sampling inadequacy or true biological progression [5].

Refined Diagnostic Criteria (RDC) proposed by Turashvili et al. address WHO under-diagnosis of metastatic potential by lowering the threshold for malignancy: stromal overgrowth plus one additional adverse feature (marked cellularity, marked atypia, or ≥10 mitoses/10 HPF in a 0.55 mm field) or marked cellularity plus one other feature [7]. Validation in 186 cases reclassified 24% of WHO-borderline tumors as malignant, reducing the metastatic rate of the borderline category to 0% (versus 10% by WHO) while maintaining excellent correlation with clinical outcomes [7,23]. A 45-year-old woman with a 6 cm tumor graded borderline by WHO but malignant by RDC developed pulmonary metastases within 18 months, validating the refined approach [7].

Immunohistochemical Profile

Ancillary markers play a supportive role in differential diagnosis:

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

Ancillary markers play a supportive role in differential diagnosis:

- CD34: Expressed in 40–60% of PTs with decreasing frequency in higher grades; negative in metaplastic carcinoma and fibromatosis [8,29].
- β-Catenin: Nuclear accumulation in 60%, non-specific (also positive in fibroadenomas and fibromatosis) [8].
- Ki-67: Labeling index increases with grade (benign <5%, borderline 5–20%, malignant 20–50%), but meta-analyses confirm lack of independent prognostic value [6,29].
- p53: Strong nuclear staining in 45% of malignant PTs, rare in lower grades; correlates with TP53 mutation status [5,29].
- c-kit (CD117): Membranous positivity in 10–50% of malignant PTs, associated with worse outcomes despite absence of activating KIT mutations [6].
- Keratins/p63: Focal stromal positivity in high-grade PTs; high-molecular-weight keratins (CK5/6, CK14) and p63 are critical for excluding metaplastic carcinoma [8,10].
- TRPS1: Recently described marker positive in both PTs and breast carcinomas, limiting diagnostic utility [8].

A diagnostically challenging spindle cell lesion with focal keratin positivity was confirmed as malignant PT by retained CD34 expression and MED12 mutation, excluding metaplastic carcinoma [10].

**Emerging Technologies and Future Directions**

Artificial intelligence algorithms trained on ultrasound images achieve 90–95% accuracy in distinguishing PTs from fibroadenomas, potentially reducing unnecessary excisions [13]. Genomic assays (16-gene panels, methylation profiling) and liquid biopsy for circulating tumor DNA show promise in early detection of recurrence. Prospective multicenter trials are needed to standardize mitotic counting fields, margin definitions, and adjuvant therapy thresholds

## Conclusion

Phyllodes tumors remain one of the most challenging entities in breast pathology due to their morphological heterogeneity, subjective grading, and unpredictable behavior. Integration of refined diagnostic criteria, molecular biomarkers (MED12, TERT, TP53), and artificial intelligence tools is transforming diagnostic accuracy and risk stratification. Surgical excision with negative margins remains the mainstay of treatment, supplemented by radiotherapy in high-risk cases. The future lies in personalized, genomics-driven approaches that will minimize overtreatment of benign lesions while optimizing outcomes in malignant PTs [1,13,20].

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

polypharmacy is highly prevalent. Polypharmacy significantly increases the complexity of treatment regimens, contributing to the rise in clinically significant interactions. In the current study, a high prevalence of polypharmacy was noted, with most patients receiving multiple medications, thereby increasing the likelihood of potential DDIs.

This study analysed 93 patients to assess the influence of various factors such as age, gender, length of hospital stay, number of prescribed medications, and the severity and clinical effects of drug interactions. The mean age of the study population was 62 years, with a female predominance, consistent with findings from Uijtendaal et al (7). Still, it was in contrast to the study conducted by Bhavika Ravindra Wagh, showing predominance of males (61.75%) (8).

Approximately 15% of identified DDIs were classified as major, although a higher rate was reported in a similar study conducted by Sarah Mahmoud Abd El Samia Mohamed et al. in Egypt (11). The majority of interactions in this study were of moderate severity, necessitating regular monitoring, findings that align with the studies by Uijtendaal et al. (9), and Hammes J[12],whereas the majority of interactions were of major severity in the similar study conducted by Bhavika Ravindra Wagh (14).

Among the top five potential DDIs identified, the interaction between Clopidogrel and Aspirin ranked highest, corroborating findings from Mohamed et al. (10) and Bertolia et al. (13). According to Lexicomp's risk rating, Type C interactions, which require close monitoring, were most common, accounting for 61.7% of cases. This is consistent with studies conducted by Yetskinyogun et al. and Haji Aghajani et al. (12). The class of drugs commonly contributing in DDIs included beta-blockers, antiplatelets, anticoagulants, and anticonvulsants.

These findings were contrast with the study conducted by Sainul Abideen et al. (11), who reported anticonvulsants as the second most frequent drug class implicated in interactions. The most frequent clinical consequences observed were increased toxicity (28.45%), decreased efficacy (18.69%), and bleeding (17.8%), consistent with findings from Uijtendaal et al.(9). Other observed effects included hypotension, hypoglycemia, and bradycardia.

In terms of interaction type, pharmacodynamic interactions predominated (66%), slightly outnumbering pharmacokinetic interactions, a distribution that aligns with the study by Adriano Max Moreira Reis et al. (5), which highlighted the prominence of metabolic mechanisms among pharmacokinetic DDIs.

A comparison of predicted (theoretical) versus clinically observed drug interactions revealed that only 27.64% of identified potential DDIs resulted in observable clinical effects. Among the drugs most frequently implicated, Clopidogrel (11.38%) emerged as the most common object drug, followed by Heparin (10.56%). As for precipitant drugs, Azithromycin (12.19%) and Bisoprolol (8.13%) were the most commonly involved, findings that are consistent with those reported by Uijtendaal et al. (9).

Overall, the study underscores the importance of vigilant medication monitoring, especially in older patients and those undergoing complex treatment regimens involving multiple drugs. The gap between potential and observed DDIs also suggests a need for improved clinical decision-support systems and ongoing risk-benefit evaluation of concurrent therapies in critical care.

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

polypharmacy is highly prevalent. Polypharmacy significantly increases the complexity of treatment regimens, contributing to the rise in clinically significant interactions. In the current study, a high prevalence of polypharmacy was noted, with most patients receiving multiple medications, thereby increasing the likelihood of potential DDIs.

This study analysed 93 patients to assess the influence of various factors such as age, gender, length of hospital stay, number of prescribed medications, and the severity and clinical effects of drug interactions. The mean age of the study population was 62 years, with a female predominance, consistent with findings from Uijtendaal et al (7). Still, it was in contrast to the study conducted by Bhavika Ravindra Wagh, showing predominance of males (61.75%) (8).

Approximately 15% of identified DDIs were classified as major, although a higher rate was reported in a similar study conducted by Sarah Mahmoud Abd El Samia Mohamed et al. in Egypt (11). The majority of interactions in this study were of moderate severity, necessitating regular monitoring, findings that align with the studies by Uijtendaal et al. (9), and Hammes J[12],whereas the majority of interactions were of major severity in the similar study conducted by Bhavika Ravindra Wagh (14).

Among the top five potential DDIs identified, the interaction between Clopidogrel and Aspirin ranked highest, corroborating findings from Mohamed et al. (10) and Bertolia et al. (13). According to Lexicomp's risk rating, Type C interactions, which require close monitoring, were most common, accounting for 61.7% of cases. This is consistent with studies conducted by Yetskinyogun et al. and Haji Aghajani et al. (12). The class of drugs commonly contributing in DDIs included beta-blockers, antiplatelets, anticoagulants, and anticonvulsants.

These findings were contrast with the study conducted by Sainul Abideen et al. (11), who reported anticonvulsants as the second most frequent drug class implicated in interactions. The most frequent clinical consequences observed were increased toxicity (28.45%), decreased efficacy (18.69%), and bleeding (17.8%), consistent with findings from Uijtendaal et al.(9). Other observed effects included hypotension, hypoglycemia, and bradycardia.

In terms of interaction type, pharmacodynamic interactions predominated (66%), slightly outnumbering pharmacokinetic interactions, a distribution that aligns with the study by Adriano Max Moreira Reis et al. (5), which highlighted the prominence of metabolic mechanisms among pharmacokinetic DDIs.

A comparison of predicted (theoretical) versus clinically observed drug interactions revealed that only 27.64% of identified potential DDIs resulted in observable clinical effects. Among the drugs most frequently implicated, Clopidogrel (11.38%) emerged as the most common object drug, followed by Heparin (10.56%). As for precipitant drugs, Azithromycin (12.19%) and Bisoprolol (8.13%) were the most commonly involved, findings that are consistent with those reported by Uijtendaal et al. (9).

Overall, the study underscores the importance of vigilant medication monitoring, especially in older patients and those undergoing complex treatment regimens involving multiple drugs. The gap between potential and observed DDIs also suggests a need for improved clinical decision-support systems and ongoing risk-benefit evaluation of concurrent therapies in critical care.

Halder et al. Current Trends in Medicine & Clinical Research
Curr. Trends Med. Clin. Res. 2025; 1(1): 19–27

## CONCLUSION

This study identified 123 potential drug-drug interactions among 93 patients, with 18 ( 15%) classified as major and the majority being of moderate severity, emphasizing the need for vigilant medication monitoring. Most interactions were pharmacodynamic, commonly involving beta-blockers, antiplatelets, and anticonvulsants. The most frequent major interaction was between heparin and azithromycin, while aspirin and clopidogrel accounted for the most common moderate interaction. Clinically, 34(27.64% )of the interactions were observed, with increased drug toxicity being the most prevalent adverse outcome. These findings highlight the importance of active involvement by trained pharmacists, routine medication reconciliation, adverse effect monitoring, and regular medication reviews particularly in under-reported tertiary care settings. Moving forward, integrating advanced drug interaction alert systems into Health Enhanced Electronic Records (EHR) can offer real-time, evidence-based guidance to healthcare providers, ultimately improving patient safety and enhancing treatment outcomes.

## LIMITATIONS

The research was conducted at a single tertiary care center, which may limit the generalizability of the findings to other healthcare settings or more diverse patient populations.The limited duration of the study restricted the ability to assess long-term outcomes and identify delayed adverse drug interactions. Data accuracy have been compromised due to incomplete or inaccurate patient histories, as some patients or caregivers may have unintentionally omitted clinically relevant information.

Conducting the study in critical care units posed challenges to patient participation and communication, potentially leading to gaps in data collection. This study had limited scope mainly the detection and classification of potential drug drug interactions and the study design was descriptive and non-interventional it did not include any active interventions, cost analysis or preventive strategies to reduce drug interaction risk is one of the limitation of the study. The short timeframe of data collection may have prevented the observation of seasonal trends or variations in prescribing patterns, thereby limiting the depth of the analysis.

## SUMMARY

123 DDIs were found among 93 ICU patients; 15% were significant, 69% were moderate, 10% were mild, and 6% were contraindicated. Azithromycin and bisoprolol were popular precipitants, whereas heparin and clopidogrel were common object drugs. Heparin and azithromycin had significant interactions. Approximately 28% experienced significant side effects, mostly pharmacodynamic ones that resulted in hypotension and toxicity. DDIs were more common in female patients and those over 60.Particularly in under reported tertiary care settings, our data highlight the importance of pharmacist involvement, routine drug reviews, and adverse effect monitoring. Advanced DDIs alert systems can optimise results, increase patient safety, and facilitate real-time decision-making when integrated into electronic health records.In summary, the findings indicate that polypharmacy, older age, and female gender are key factors contributing to the prevalence of drug-drug interactions in critically ill patients.

## REFERENCES

1. Rayzah M. Phyllodes Tumor of Breast: A Review Article. ISRN Surg. 2013;2013:361469.
2. Lu Y, Chen Y, Zhu L, et al. Local Recurrence of Benign, Borderline, and Malignant Phyllodes Tumors of the Breast: A Systematic Review and Meta-Analysis. Ann Surg Oncol. 2019;26(5):1263-1272.
3. Zhou H, Yi W, Wang J, et al. Adjuvant radiation therapy for malignant phyllodes tumor of the breast: a systematic review. Cancer Manag Res. 2021;13:6655-6661.
4. WHO Classification of Tumours Editorial Board. Breast Tumours. 5th ed. Lyon: International Agency for Research on Cancer; 2019.
5. Li J, Dahlstrom JE, Clouston D, et al. Immunohistochemistry of p53 in malignant phyllodes tumours of the breast. Pathology. 2000;32(4):262-6.
6. Tan WJ, Thike AA, Tan SY, et al. CD117 expression in breast phyllodes tumors correlates with adverse pathologic features and increased risk of recurrence. Mod Pathol. 2015;28(1):151-60.
7. Turashvili G, Ding Q, Liu Y, et al. Comprehensive clinical-pathologic assessment of malignant phyllodes tumors: Proposing refined diagnostic criteria. Am J Surg Pathol. 2023;47(11):1195-1206.
8. Piscuoglio S, Ng CK, Murray M, et al. The Genomic Landscape of Phyllodes Tumors of the Breast. Clin Cancer Res. 2016;22(7):1733-43.
9. Pareja F, Geyer FC, Kumar R, et al. Phyllodes tumors with and without fibroadenoma-like areas display distinct genetic alterations and may evolve independently. NPJ Breast Cancer. 2017;3:40.
10. Lawton TJ, Acs G, Argani P, et al. Interobserver variability by breast pathologists of stromal cellularity and atypia in phyllodes tumors. Hum Pathol. 2014;45(5):914-9.
11. Ross DS, Giri DD, Akram MM, et al. Fibroepithelial lesions in the breast of adolescent females: a clinicopathological study of 54 cases. Breast J. 2016;22(1):86-90.
12. Abdul-Ghafar J, Ud Din N, Saadaat R, et al. Malignant Phyllodes Tumor With Heterologous Differentiation: A Rare Case Report. Cureus. 2020;12(11):e11478.
13. Zeng S, Ni Y, Qiu Y, et al. Artificial intelligence-assisted diagnosis for phyllodes tumor and fibroadenoma: A retrospective deep learning study on ultrasound images. Front Oncol. 2022;12:1024022.
14. Sood R, Rositch AF, Shakoor D, et al. Ultrasound for Breast Cancer Detection Globally: A Systematic Review and Meta-analysis. J Glob Oncol. 2019;5:1-17.
15. Spitaleri G, Toesca A, Botteri E, et al. Breast phyllodes tumor: a review of literature and a single center retrospective series analysis. Crit Rev Oncol Hematol. 2013;88(2):427-36.
16. Tse GM, Niu Y, Shi HJ. Phyllodes tumor of the breast: an update. Breast Cancer. 2010;17(1):29-34.
17. Wei J, Tan TY, Tan PH. Phyllodes tumours: a 15-year review from a single institution in Singapore. Ann Acad Med Singap. 2011;40(1):25-31.
18. Tan BY, Acs G, Apple SK, et al. Phyllodes tumours of the breast: a consensus review. Histopathology. 2016;68(1):5-21.
19. Cimino-Mathews A, Hicks JL, Sharma R, et al. A subset of malignant phyllodes tumors harbors alterations in the Rb/p16 pathway. Hum Pathol. 2013;44(11):2494-500.
20. Zhang Y, Kleer CG. Phyllodes Tumor of the Breast: Histopathologic Features, Differential Diagnosis, and Molecular/Genetic Updates. Arch Pathol Lab Med. 2016;140(7):665-71.
21. Tan PH, Ellis I, Allison K, et al. The 2019 World Health Organization classification of tumours of the breast. Histopathology. 2020;77(2):181-185.
22. Koh VCY, Thike AA, Tan PH. Distant metastases in phyllodes tumours of the breast: an overview. Appl Cancer Res. 2015;35:6.
23. Mituś JW, Blecharz P, Reinfuss M, et al. Phyllodes tumors of the breast. The treatment results for 340 patients from a single cancer centre. Breast. 2019;43:85-90.
24. Hino N, Nakagawa Y, Ueda T, et al. Hypoglycemia due to insulin-like growth factor (IGF)-II secretion by a malignant phyllodes tumor of the breast. Intern Med. 2010;49(14):1407-11.