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Can Excision be Avoided for Atypical Ductal Hyperplasia of the Breast Diagnosed on Core Biopsy?

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ABSTRACT

Background: Atypical ductal hyperplasia (ADH) diagnosed on core biopsy (CB) is associated with an upgrade risk to ductal carcinoma in situ (DCIS) or invasive carcinoma on surgical excision (SE). Although single institutional studies have shown observation and surveillance can be considered in a select subgroup, most patients undergo surgery. We aim to identify features least associated with upgrade on SE, thereby identifying patients who may potentially be spared surgery.

Methods: We conducted a cross-sectional study at University of Miami analyzing imaging, clinical, and pathologic data of ADH diagnosed on CB. Histopathologic characteristics of ADH on CB and SE were recorded and analyzed.

Results: Seventy-one CB from 70 patients were included. CB removed >50% of the imaging target in 69% of cases and ≤50% in 31% of cases, showing complete ductule involvement in 31% and incomplete involvement in 69%. ADH was focal (≤1 focus) in 58% and non-focal (>1 focus) in 42%. On SE, 5 cases upgraded to DCIS. Upgrade was more common when CB removed ≤50% compared to >50% (18% vs. 2%). Complete ADH had a significantly higher upgrade rate than incomplete ADH, with no difference between focal and non-focal. Forty-eight percent had low-risk ADH features, defined as incomplete ADH with >50% target removal.

Conclusion: Upgrade is limited to DCIS and related to sampling adequacy and extent of ADH. Careful histologic-radiologic correlation can identify a subgroup of ADH with low-risk features, representing possible candidates for observation and surveillance.

INTRODUCTION

Atypical ductal hyperplasia (ADH) is a borderline lesion with intraductal proliferation of cells similar to low-grade ductal carcinoma in situ (DCIS) but insufficient to be diagnosed as DCIS.[1,2] The definition of ADH has undergone several modifications by various authors over the years. The World Health Organization (WHO) defines ADH with a size cutoff of ≤2 mm in contiguous extent; lesions larger than 2 mm are considered low-grade DCIS.[2] However, this size criterion applies to

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proliferations with complete duct involvement but not partial involvement.[1-3]

ADH is associated with an increased long-term risk of subsequent invasive breast carcinoma, with a relative risk of 4-5 times higher than the general population and an absolute risk of 1-2% per year, reaching 30% at 25 years of follow-up.[4-10] In addition, ADH diagnosed on core biopsy can be associated with an upgrade to DCIS or invasive carcinoma on subsequent surgical excision.

Considering the precursor potential of ADH, surgical excision has been the standard approach for ADH diagnosed on core biopsy to prevent undertreatment of those lesions that are potentially associated with unsampled invasive carcinoma. However, recent studies suggest that observation and surveillance may be considered in a subset of patients with low-risk features.[11-13] Our study aims to identify ADH features associated with upgrades to DCIS or invasive carcinoma at our institution and identify patients with low-risk features who may benefit from close clinical observation and surveillance instead of surgical intervention.

METHODS

With institutional review board approval, we conducted a cross-sectional study at the University of Miami analyzing imaging, clinical, and pathologic data from patients diagnosed with ADH on core biopsy between January 2020 and June 2023. All consecutive cases that met the inclusion criteria were included. Eligible cases were those with a biopsy diagnosis of ADH and subsequent surgical excision performed at our institution. Exclusion criteria included synchronous invasive carcinoma or DCIS in the same quadrant, ADH bordering on DCIS (intraductal proliferation with overlapping features of both ADH and low-grade DCIS),[14] atypia with apocrine features, and lack of follow-up surgical excision. The clinical history of prior atypia was obtained through review of the medical record.

All available mammography, ultrasound, and, when applicable, breast MRI examinations were re-reviewed by a breast radiologist (J.S.). Imaging characteristics were documented: imaging modality (mammogram, contrast-enhanced mammogram, ultrasound, magnetic resonance imaging [MRI]), breast composition, imaging target (calcifications, mass/distortion, or non-mass enhancement [NME]), size of the target lesion, type of biopsy and needle size, as well as the estimated percentage of the target lesion removed. The percentage of the lesion removed was determined by volumetric comparison of pre- and post-biopsy imaging, excluding post-procedural changes such as hematoma and biopsy cavity. The Breast Imaging Reporting and Data System (BI-RADS®) was used for standardized reporting.[15]

All histologic slides, including immunohistochemical stains, were re-reviewed by two breast pathologists (YT and BS) to confirm diagnoses of ADH according to WHO criteria. In the event of a diagnostic disagreement, the case would undergo joint review with a third pathologist and be discussed at a multi-headed microscope. The 2 mm size criterion was used, while the size of ADH involving intraductal papilloma was limited to 3 mm. The histopathologic characteristics of each lesion diagnosed as ADH were recorded. We divided ADH into focal (one focus) and non-focal (two or more foci), based on biopsy yield, following a similar approach as previously described by Ely *et al.*[16] Additionally, the involvement of each ductule or terminal ductal lobular unit (TDLU) by the clonal proliferation of epithelial cells was defined as complete ADH or incomplete/partial ADH as previously described by Zhang *et al.*[17] The architectural patterns were characterized as cribriform, micropapillary, solid, or mixed. We also documented calcifications and their association with ADH, along with any other lesions present in the biopsy, such as radial scars, intraductal papillomas, fibroadenomas, fibrocystic changes, flat epithelial atypia (FEA), and non-invasive lobular neoplasia (atypical lobular hyperplasia or lobular carcinoma in situ). The histologic-radiologic correlation of biopsy results was assessed as concordant or discordant, with discordance defined as pathology findings that could not explain the imaging target.[15]

Surgical excision specimens were entirely submitted or submitted in up to 20 cassettes, with larger excisions and mastectomies ensuring the entire imaging target and adjacent tissue were examined histologically. Specimens were radiographed using a Kubtec imaging system to identify all biopsy markers before tissue sampling. Pathology reports and slides of the excision specimens were reviewed, documenting the size and extent of ADH, any upgrades to DCIS or invasive carcinoma, correlation with the biopsy site, and relevant pathologic findings (histologic type, grade, size, stage, and receptor status) for each case. Upgrade assessment was performed by systematically comparing the core biopsy diagnosis with the corresponding surgical excision findings.

Statistical analyses were performed using Fisher's exact test to assess the association between categorical variables. A p-value of less than 0.05 was considered statistically significant, indicating meaningful differences and associations within the data.

RESULTS

Study population

From January 2020 to June 2023, we identified 74 core biopsies with a diagnosis of ADH. After excluding 3 histologic-radiologic discordant cases, 71 biopsies from 70 patients comprised the final study population. The median age at diagnosis was 56 years (range 34 - 82 years). Five patients had a prior diagnosis of ADH, while five had a prior diagnosis of known breast carcinoma, including 2 ipsilateral DCIS and 3 contralateral invasive carcinomas. Synchronous atypia or carcinoma was identified in different quadrants of the ipsilateral breast than the targeted ADH in 5 patients [5 ipsilateral invasive ductal carcinomas (IDC)] and in the contralateral breast in 11 patients [4 contralateral DCIS, 5 contralateral IDC, and 2 contralateral invasive lobular carcinomas (ILC)].

Breast imaging characteristics of ADH

All the patients underwent mammography; additional breast ultrasound was performed in 63% (45/71), and MRI in 44% (31/71). Breast composition, categorized using BI-RADS density standards, was fatty in 10% (7/71), scattered fibroglandular in 55% (39/71), heterogeneously dense in 31% (22/71), and extremely dense in 4% (3/71).

All image-guided core biopsies were performed using vacuum-assisted breast biopsy techniques, including 58% stereotactic-guided (41/71), 27% ultrasound-guided (19/71), 14% MRI-guided (10/71), and 1% contrast-enhanced mammogram (1/71). Imaging targets included calcifications in 54% of cases (38/71), mass-forming lesions including asymmetry/distortion in 38% of cases (27/71), and non-mass enhancement (NME) detected by MRI in 8% of cases (6/71).

Cases presenting with mammographic calcifications showed targets ranging from 3 to 42 mm. Breast ultrasound in the remainder of the cases showed mass lesions ranging from 4 to 27 mm. Most biopsy procedures used a 9-gauge (54/71, 76%) or 12-gauge (14/71, 20%) needle; a 14-gauge needle was used in two cases (3%), and needle size was not specified in one case. Post-procedure imaging demonstrated that >50% of the targeted lesion was removed in 69% of cases (49/71), whereas ≤ 50% of the lesion was removed in 31% of cases (22/71). Breast imaging results are summarized in Table 1.

Histopathological characteristics of ADH

The predominant histologic pattern of ADH was cribriform (47/71, 66%), followed by mixed patterns (12/71, 17%), micropapillary (7/71, 10%), and solid (5/71, 7%). Overall, focal ADH was identified in 42% (30/71) of core biopsies and non-focal ADH in 58% (41/71) of core biopsies; 31% (22/71) showed complete ADH and 69% (49/71) showed incomplete ADH (Figure 1).

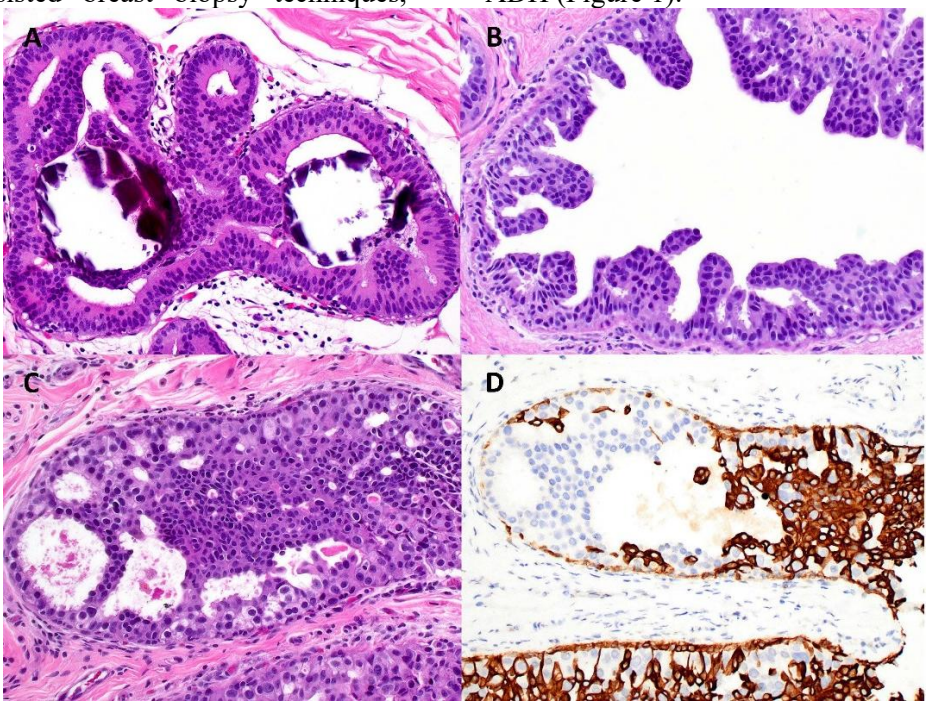

Figure 1. Atypical ductal hyperplasia (ADH) is characterized by monomorphic epithelial cells affecting terminal duct lobular units. A. Breast core biopsy targeting calcifications shows focal ADH with incomplete involvement of the ductule (Hematoxylin and Eosin [H&E], 20x). B. Core biopsy targeting a mass demonstrates ADH with micropapillary architecture involving a single duct, showing uniform cell population and rigid architectural pattern characteristic of ADH (H&E, 20x). C. ADH demonstrating cribriform pattern with uniform, punched-out spaces (H&E, 20x). D. Cytokeratin 5/6 immunostain confirms incomplete involvement of the ductule (CK5/6, 20x).

Table 1. Clinical, Radiologic, and Histologic Characteristics of Atypical Ductal Hyperplasia Cases

Characteristic	Overall (N=71)	Non-upgraded (N=66)	Upgraded (N=5)	p-value
Age, years (range)	56 (34-82)	57 (34-82)	53 (42-78)	
Imaging targets and features				
• Calcifications	54% (38 of 71)	89% (34 of 38)	11% (4 of 38)	0.3908
• Mass, distortion/asymmetry	38% (27 of 71)	96% (26 of 27)	4% (1 of 27)	
• Non-mass enhancement	8% (6 of 71)	100% (6 of 6)	-	
Biopsy of the targeted lesion				
• ≤50% of target removed	31% (22 of 71)	82% (18 of 22)	18% (4 of 22)	
• >50% of target removed	69% (49 of 71)	98% (48 of 49)	2% (1 of 49)	0.0296
Histologic features of ADH on biopsy				
• Focal	42% (30 of 71)	93% (28 of 30)	7% (2 of 30)	
• Non-focal	58% (41 of 71)	93% (38 of 41)	7% (3 of 41)	1.0
• Complete	31% (22 of 71)	82% (18 of 22)	18% (4 of 22)	
• Incomplete	69% (49 of 71)	98% (48 of 49)	2% (1 of 49)	0.0296

ADH, atypical ductal hyperplasia

In core biopsies targeting calcifications, calcifications were associated with ADH. In addition, ADH was associated with FEA (6/38), non-invasive lobular neoplasia (3/38), radial scar (2/38), intraductal papilloma (2/38), fibrocystic changes (1/38), and fibroadenoma (1/38). In core biopsies targeting mass lesions, ADH was associated with intraductal papilloma (12/27), radial scar (3/27), fibrocystic changes (3/27), fibroadenoma (3/27), adenosis/sclerosing adenosis (2/27), and non-invasive lobular neoplasia (1/27). In MRI-guided biopsies targeting NME, ADH was associated with either intraductal papilloma (1/6), non-invasive lobular neoplasia (1/6), or periductal fibrosis (1/6). All cases demonstrated histologic–radiologic concordance, with the targeted imaging abnormality corresponding to the pathologic lesion identified on biopsy. The histopathologic results are summarized in Table 1.

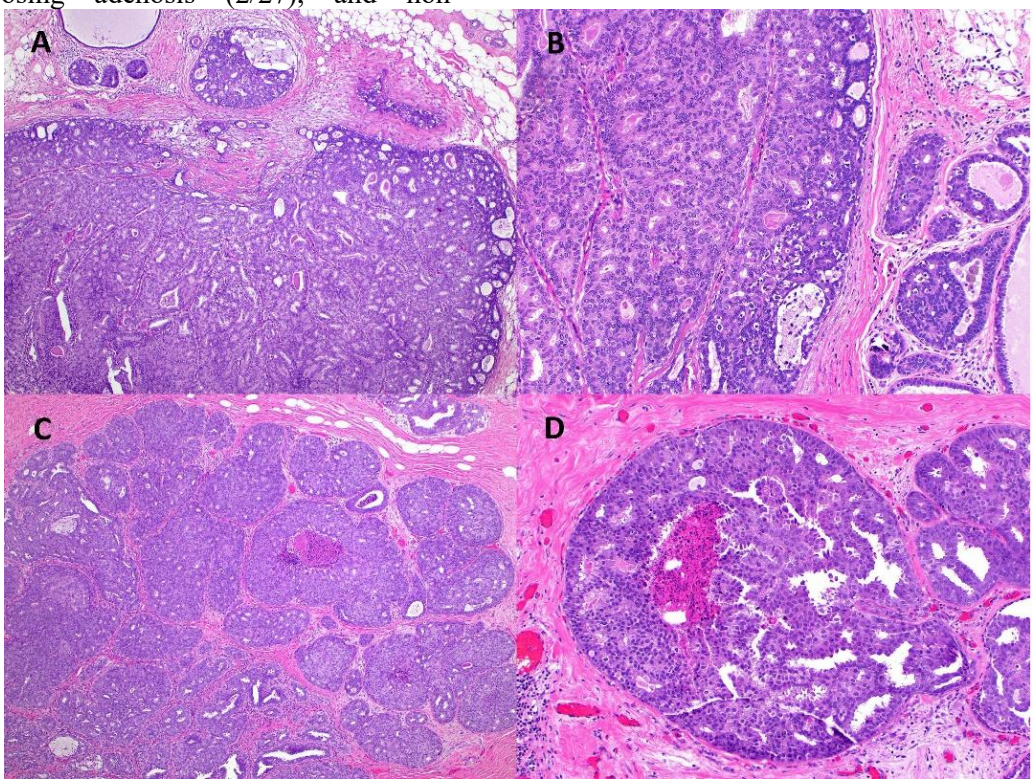

Figure 2. Cases with ductal carcinoma in situ (DCIS) identified on surgical excision following core biopsy diagnosis of atypical ductal hyperplasia. A-B. Core biopsy targeting calcifications was upgraded to DCIS on surgical excision (Case 1). Excision reveals a 3 mm focus of intermediate nuclear grade DCIS with cribriform architecture and scattered calcifications (H&E, A. 4x, B. 10x). C-D. Surgical excision targeting mass/architectural distortion demonstrates intermediate nuclear grade DCIS with associated necrosis (Case 5). The necrotic debris are better seen at higher magnification (H&E, C. 4x, D. 10x).

Characteristics of upgraded cases

Most patients (93%; 66/71) had localized excision of the biopsy site area, while 7% (5/71) of patients underwent mastectomy. On excision, an upgrade to DCIS was identified in 7% of cases (5/71), including 11% of biopsies targeting calcifications (4/38) and 4% of biopsies targeting masses (1/27). The upgrade rate was higher for biopsies of calcifications than for mass lesions (11% vs 4%), but this difference was not statistically significant (p=0.39). None of the cases targeting NME were upgraded. Invasive carcinoma was not identified in any excision specimen.

All DCIS were estrogen receptor (ER)-positive with either low nuclear grade (20%; 1/5) or intermediate nuclear grade (80%; 4/5) with predominantly cribriform architecture (Figure 2). Focal necrosis in the DCIS was identified in one upgrade (Figure 2C-D). DCIS size ranged from 3 mm to 20 mm in its greatest dimension. Ancillary testing, including loss of staining for cytokeratin 5/6 and overexpression of ER in the atypical ductal proliferation by immunohistochemistry, supported the diagnosis of DCIS in all cases. The detailed clinical, imaging, and histopathologic characteristics of each upgraded case are presented in Table 2.

Overall, upgrade was significantly more common in core biopsies with complete ADH (4/22, 18%) versus incomplete ADH (1/49, 2%) ($p = 0.029$), while no significant difference in upgrade rate was observed between focal ADH (2/30, 7%) versus non-focal ADH (3/38, 8%) ($p = 1.0$). One upgraded ADH had a history of ipsilateral atypia, while none of the upgraded patients had prior or synchronous breast carcinoma. In addition, upgrade was significantly more common when ≤50% of the imaging target was removed by core biopsy compared to when >50% was removed, 18% versus 2%, respectively ($p = 0.0296$). No upgrade was found in cases with incomplete ADH when a core biopsy removed >50% of the imaging target. Cases with both features (incomplete ADH and >50% removal of imaging target) could be assigned to a low-risk group comprising 34 patients (48%) in our study population.

Long-term follow-up outcomes

Follow-up data were available for 66 patients with a median follow-up of 27 months (range: 6-54 months). During follow-up, 5 of 66 patients (8%) had new breast lesions. One patient developed ADH in a different quadrant at 13 months, three patients developed DCIS (two at the same site and one in a different quadrant), and one patient developed a 4 mm well-differentiated IDC in a different quadrant at 23 months. All subsequent lesions were detected in the non-upgraded group.

Table 2. Characteristics of Upgraded ADH Cases

Case	Biopsy modality, needle size	Biopsy target	Associated lesion	Estimated target removed (%)	ADH characteristics on core biopsy	DCIS on excision (size)
1	Stereotactic-guided VABB, 9-gauge	Calcifications	Fibrocystic changes	80	Focal, complete, solid pattern	Intermediate grade DCIS involving IDP, 3 mm
2	Stereotactic-guided VABB, 9-gauge	Calcifications	IDP	50	Non-focal, complete, cribriform pattern	Intermediate grade DCIS, 20 mm
3	Stereotactic-guided VABB, 9-gauge	Calcifications	None	20	Non-focal, complete; cribriform pattern	Intermediate grade DCIS, 8 mm
4	Stereotactic-guided VABB, 9-gauge	Calcifications	None	20	Non-focal, complete, mixed cribriform and micropapillary pattern	Low grade DCIS, 15 mm
5	Ultrasound-guided VABB, 12-gauge	Mass/distortion	IDP	50	Focal, incomplete, micropapillary pattern	Intermediate grade DCIS involving IDP, 20 mm

Abbreviations: ADH, atypical ductal hyperplasia; DCIS, ductal carcinoma in situ; IDP, intraductal papilloma

DISCUSSION

Diagnostic Challenges in ADH

The reported incidence of ADH in core biopsies ranges from 1% of ultrasound-guided core biopsies to 14% of stereotactic-guided biopsies.[4,6,7] Atypical ductal proliferations encompass a spectrum of lesions with various degrees of quantitative and qualitative variations, but pathologic categories are often based on arbitrary thresholds rather than biological variables. For example, a low-grade atypical ductal proliferation measuring 2 mm may be classified as ADH, while an identical lesion measuring 2.5 mm

could be classified as low-grade DCIS. Subjective morphologic interpretation of atypia, along with the lack of standardized protocols, contributes to interobserver discordance with respect to borderline breast lesions. FEA is on the lower end of the ductal atypia spectrum, while ADH is on the higher end of the spectrum and overlaps with low-grade DCIS, with the key distinguishing feature being size (greater than 2 mm).[2,14] A concordance rate of 48% was reported for atypical breast categories compared to the consensus-derived reference diagnosis.[18] However, diagnostic agreement among intraductal atypical lesions can be improved through uniform criteria, ancillary studies (i.e., CK5/6 and ER), consensus agreements, and/or second pathology reviews. As management decisions do not account for these complexities, the high diagnostic disagreement in the area of breast atypia, even among experts, makes ADH one of the more challenging categories in breast pathology.[14,19]

Study Findings and Review of Literature

Given the diagnostic challenges and the clinical need for better risk stratification, we focused exclusively on pathologic-radiologic concordant cases to ensure diagnostic accuracy. In our study, which focused solely on pathologic-radiologic concordant cases, the upgrade rate for ADH on subsequent excision was 7%. Overall, upgrades were limited to small, low-to-intermediate nuclear-grade DCIS without invasion. The upgrade rate was higher in core biopsies targeting calcifications than those targeting mass-forming or NME targets, especially when the core biopsy procedure removed ≤50% of the imaging target. This finding contrasts with a prior study where core biopsies targeting a mass were more likely to be associated with an upgrade.[20] Notably, concordant core biopsies that removed >50% of the mass target, where benign histologies (e.g., fibroadenoma, intraductal papilloma, and radial scar) were identified alongside ADH, showed no upgrades to carcinoma.

Our findings align with emerging evidence supporting risk stratification approaches. Complete ADH and ≤50% removal of imaging target were significantly associated with higher upgrade rates. When applying established low-risk criteria defined by Nguyen *et al.* (≤2 foci and ≥95% target removal) to our study population, no upgrades were identified in this subgroup, accounting for 23% of cases.[21] When we applied low-risk criteria characterized in our study, we found no upgrades in cases of incomplete ADH where the core biopsy removed >50% of the imaging target. These two features delineated our institutional low-risk subgroup, encompassing 48% of our patients. These findings are consistent with other single-institution studies demonstrating that focal ADH with adequate sampling has lower upgrade rates than extensive lesions with incomplete removal.[13,26,27]

Features associated with a low-risk of upgrade have been defined in several studies using combinations of criteria, including the percentage of targeted lesion(s) removed, absence of a mass on imaging, the number of foci of ADH identified in the biopsy, and the absence of single-cell necrosis.[11,13,17,21-26] Peña *et al*. separately defined low-risk lesions as ADH without necrosis and either one focus with ≥50% removal or 2–3 foci with ≥90% removal.[11] In contrast, complete ADH, >3 foci, and punctate necrosis are features associated with a higher upgrade rate.[17,21] Grabenstetter *et al*. reported that focal ADH (one focus ≤2 mm) had a lower upgrade rate than non-focal ADH.[13] While further validation through larger studies is needed, our current findings suggest that a significant proportion of patients diagnosed with ADH on biopsy may be assigned to a low-risk subgroup.

Clinical Implications and Management

These results have important implications for evolving management of ADH. While the current standard of care for patients with DCIS diagnosed on core biopsy is surgery with post-lumpectomy radiation therapy, the management of ADH is increasingly nuanced.[26,27] Single-institutional studies suggest that observation and surveillance may be appropriate for carefully selected patients with low-risk ADH features.[11,12,25]

Our follow-up data demonstrated 5 subsequent breast lesions (one ADH, three DCIS, and one IDC), all in the non-upgraded group, underscoring that the long-term increased risk of carcinoma development in either breast necessitates ongoing follow-up and consideration of risk-reducing endocrine therapy.[28] A multidisciplinary approach is essential for managing ADH diagnosed on biopsy, emphasizing careful correlation between pathologic and imaging findings for effective planning and discussions in multidisciplinary conferences.

Study Limitations and Future Directions

While this single-institution cross-sectional study using historical data has limitations, our findings provide valuable insights into ADH risk stratification. These limitations include a small sample size that limits statistical power, potential selection bias from including only patients who underwent surgical excision, exclusion of histologic-radiologic discordant cases which may limit generalizability to all ADH cases encountered in clinical practice, and lack of long-term outcome data. Inherent inter-

observer variability in ADH diagnosis remains a limitation despite review by experienced breast pathologists.

Despite these limitations, our findings contribute to evidence supporting personalized ADH management approaches. Future multi-institutional prospective studies with larger populations, standardized protocols, and long-term follow-up are needed to validate these risk stratification criteria and establish definitive management guidelines for low-risk ADH patients.

CONCLUSION

In our cohort, upgrades of ADH diagnosed on core biopsy are limited to small, low-to-intermediate grade DCIS. Factors associated with a low risk of upgrade include incomplete ADH and >50% removal of the imaging target. A multidisciplinary approach, including careful correlation between histologic and radiologic findings, the adequacy of sampling, and the qualitative and quantitative extent of ADH in a core biopsy, can be utilized to select patients with ADH who may benefit from active surveillance and risk-reducing strategies rather than surgical excision.

CONFLICTS OF INTEREST

The authors have no conflict of interest to disclose.

ETHICAL CONSIDERATIONS

All procedures performed in this retrospective data analysis involving human participants were in accordance with the ethical standards of the institutional review board, which did not require informed consent. This study was approved by the University of Miami Institutional Review Board (IRB #00000738).

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DATA AVAILABILITY

All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author. All data generated or analyzed during this study are included in this article. Further inquiries can be directed to the corresponding author.

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None.

AI DISCLOSURE

No AI tools used in preparing this manuscript.

AUTHOR CONTRIBUTIONS

YT, NCM, and BS contributed to the study's conception and design. Material preparation, data collection, and analysis were performed by YT and NCM. YT wrote the first draft of the manuscript. YT, JS, LMB, NCM, SBK, and BS reviewed and revised it. All authors have read and approved of the final manuscript.

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