Histopathologic Correlates of Nonmass Enhancement Detected by Breast Magnetic Resonance Imaging

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Dynamic, contrast-enhanced magnetic resonance imaging (MRI) is a highly sensitive imaging modality for the detection of breast cancer.1–8 Over the past decade, the clinical role of breast MRI has greatly expanded and it has become an important tool. Currently, the more common clinical indications include preoperative staging of patients, high-risk screening in women with high genetic, familial, or personal risk factors, analysis of indeterminate breast lesions that were detected with other imaging modalities, such as mammography or ultrasound, and evaluation of neoadjuvant chemotherapy response.9–15

However, although MRI has proven to be a highly sensitive imaging modality, with studies showing a sensitivity rate of approximately 90% or higher, the specificity is lower, often ranging between 75% to 85% depending on the study.16–22

Lesions detected by MRI can be categorized broadly by morphology into mass, nonmass, or focus, with nonmass enhancement (NME) being defined as an area of enhancement without an associated space-occupying mass that is distinct from the surrounding background parenchyma. NME has been associated with a wide spectrum of benign and malignant lesions23–34 and more than half of nonpalpable invasive carcinomas according to 1 study.35 Given the overlap between benign and malignant lesions, there may be resultant uncertainty with regard to the concordance between MRI and pathologic findings in such cases. This uncertainty may cause the need for additional procedures.

Context.—Dynamic, contrast-enhanced magnetic resonance imaging (MRI) is a highly sensitive imaging modality for screening and diagnostic purposes. Nonmass enhancement (NME) is commonly seen on MRI of the breast. However, the pathologic correlates of NME have not been extensively explored. Consequently, concordance between MRI and pathologic findings in such cases may be uncertain and this uncertainty may cause the need for additional procedures.

Objective.—To examine the histologic alterations that correspond to NME on MRI.

Design.—We performed a retrospective search for women who underwent breast MRI between March 2014 and December 2016 and identified 130 NME lesions resulting in biopsy. The MRI findings and pathology slides for all cases were reviewed. The follow-up findings on any subsequent excisions were also noted.

Results.—Among the 130 cases, the core needle biopsy showed 1 or more benign lesions without atypia in 80 cases (62%), atypical lesions in 21 (16%), ductal carcinoma in situ in 22 (17%), and invasive carcinoma in 7 (5%). Review of the imaging features demonstrated some statistically significant differences in lesions that corresponded to malignant lesions as compared with benign alterations, including homogeneous or clumped internal enhancement, type 3 kinetics, and T2 dark signal; however, there was considerable overlap of features between benign and malignant lesions overall. Of 130 cases, 54 (41.5%) underwent subsequent excision with only 6 cases showing a worse lesion on excision.

Conclusions.—This study illustrates that NME can be associated with benign, atypical, and/or malignant pathology and biopsy remains indicated given the overlap of radiologic features.

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tively searched from March 1, 2014 to December 31, 2016 for core needle biopsy cases with a radiologic indication of NME. Additional information collected from the pathology archive included patient sex and age at time of biopsy. A total of 164 core needle biopsies were initially identified. Of these, 10 cases were excluded because imaging was performed at outside institutions and was not available for review. An additional 24 cases were excluded because the area of NME was within the same quadrant as an area of known concurrent carcinoma and thus judged (after review of imaging) to be a possible area of satellite extension from the mass lesion and not a distinct area of NME.

All hematoxylin-eosin–stained slides for the remaining 130 cases were reviewed by 2 board-certified pathologists with fellowship training in breast pathology (VFT and GMB) without knowledge of the previous interpretation. A detailed morphologic analysis was performed and all histologic alterations were identified and recorded for each case. The worst/most severe (highest risk) lesion in each case was noted. For example, if a case had both pseudoangiomatous stromal hyperplasia and ductal carcinoma in situ (DCIS), then DCIS was counted as the most severe finding; if the case had both DCIS and invasive carcinoma, then the invasive carcinoma was counted as the most severe finding. In addition, assessed was the dominant lesion for each case, which was defined as the most prevalent alteration and not necessarily the worst alteration (ie, the most quantitatively prominent lesion on the biopsy for each case). Each case potentially could have more than 1 dominant alteration if 2 histologic alterations were both equally prominent; alternatively, a case could have no dominant alteration.

The MRI reports were reviewed by 2 radiologists with subspecialty expertise in breast imaging (NAR and JP) for overall background enhancement pattern as well as the distribution, internal enhancement pattern, T1/T2 characteristics, and kinetic pattern of the NME. To capture data for all cases, the MRIs were reviewed by 1 of 2 breast imaging radiologists for cases with incomplete imaging descriptions of the NME. The descriptors of NME adhered to the breast imaging-reporting and data system (BI-RADS) and every NME had a complete description with 3 terms as follows: type, distribution, and kinetic curve.

Imaging features of malignant (DCIS or invasive carcinoma) and benign lesions were compared using $X^2$ and Fisher exact tests as appropriate. $P$ values < .05 were considered statistically significant.

### RESULTS

A total of 130 biopsies were obtained from 121 patients (median age 50 years, mean 50.1 years, range 27–78 years) that fit the inclusion criteria during the specified time period. Of 130 cases, 66 patients (50.8%) had NME identified as part of high-risk screening, 37 (28.5%) were undergoing evaluation for extent of disease, and 27 (20.8%) had a clinical finding with no corresponding imaging finding using other modalities.

The “worst” or most severe histologic alteration was documented for each case, with the general categorization outlined in Table 1 and the detailed catalogue of findings documented in Table 2. The majority of cases (80 of 130; 61.5%) showed benign findings without atypia. Of the benign alterations, the most common findings included usual ductal hyperplasia (UDH) (34 of 130; 26.2%), papillomas (11 of 130; 8.5%), and pseudoangiomatous stromal hyperplasia (6 of 130; 4.6%). The benign findings are further divided into those that are not generally actionable in-and-of-themselves (such as UDHP or apocrine metaplasia; 60 of 80 benign cases) and those that may be actionable (such as radial scars/complex sclerosing lesions and papillomas; 20 of 80 benign cases) (Table 1).

Of 130 cases, 7 (5.4%) showed invasive carcinoma as their worst histologic lesion. The invasive carcinomas included 5

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| Table 1. Most Severe (Worst) Histologic Finding for All Nonmass Enhancement Cases |
|---------------------------------------------------------------|
| **Histologic Finding**                                      | **N (%)** |
|---------------------------------------------------------------|
| Benign findings (without atypia)                             | 80 (61.5) |
| Benign findings, not actionable                              | 60 |
| (usual ductal hyperplasia, columnar cell change/hyperplasia, | 60 |
| apocrine metaplasia, adenosis, ectatic ducts, fibroadenomatous | 60 |
| change, pseudoangiomatous stromal hyperplasia)              | 60 |
| Benign findings, actionable                                 | 20 |
| (radial scar, complex sclerosing lesion, papilloma, fibroadema, | 20 |
| benign spindle cell/vascular lesion)                        | 20 |
| Atypical lesions (flat epithelial atypia, atypical lobular    | 21 (16.2) |
| hyperplasia/lobular carcinoma in situ, atypical ductal       | 21 |
| hyperplasia)                                                 | 21 |
| Ductal carcinoma in situ                                     | 22 (16.9) |
| Invasive carcinoma                                           | 7 (5.4) |
| **Total**                                                    | 130 |

| Table 2. Worst (Most Severe) Histologic Finding for All Nonmass Enhancement Cases |
|---------------------------------------------------------------|
| **Histologic Finding**                                      | **N (%)** |
|---------------------------------------------------------------|
| Benign/without atypia (n = 80)                               |          |
| Usual ductal hyperplasia                                     | 34 (26.2) |
| Papilloma                                                    | 11 (8.5)  |
| Pseudoangiomatous stromal hyperplasia                        | 6 (4.6)   |
| Sclerosing adenosis                                          | 5 (3.8)   |
| Columnar cell change/columnar cell hyperplasia               | 4 (3.1)   |
| Vascular lesion                                              | 4 (3.1)   |
| Fat necrosis                                                 | 3 (2.3)   |
| Radial scar/complex sclerosing lesion                        | 3 (2.3)   |
| Apocrine cysts                                               | 2 (1.5)   |
| Ectatic ducts                                                | 2 (1.5)   |
| Adenosis                                                     | 1 (0.8)   |
| Fibroadenoma/fibroadenomatous change                         | 1 (0.8)   |
| Lymphocytic mastopathathy                                    | 1 (0.8)   |
| Mammary duct ectasia                                         | 1 (0.8)   |
| Spindle cell lesion                                          | 1 (0.8)   |
| None (breast tissue only)                                    | 1 (0.8)   |
| **Atypical lesions (n = 21)**                                |          |
| Flat epithelial atypia                                       | 2 (1.5)   |
| Atypical lobular hyperplasia                                 | 4 (3.1)   |
| Atypical ductal hyperplasia                                  | 12 (9.2)  |
| Lobular carcinoma in situ                                    | 3 (2.3)   |
| Ductal carcinoma in situ (n = 22)                            | 22 (16.9) |
| Invasive carcinoma (n = 7)                                   |          |
| Invasive ductal carcinoma                                    | 5 (3.8)   |
| Invasive lobular carcinoma                                   | 1 (0.8)   |
| Invasive carcinoma with ductal and lobular features          | 1 (0.8)   |
| **Total**                                                    | 130 |
invasive ductal carcinomas, 1 invasive lobular carcinoma, and 1 invasive carcinoma with ductal and lobular features. DCIS was the most severe lesion in 22 (16.9%) cases. Atypical lesions, which included flat epithelial atypia, atypical lobular hyperplasia, atypical ductal hyperplasia (ADH), and lobular carcinoma in situ, comprised 21 (16.2%) cases. The atypical lesions included 2 cases of flat epithelial atypia, 4 cases of atypical lobular hyperplasia, 12 cases of ADH, and 3 cases of lobular carcinoma in situ.

All histologic findings for all cases were also catalogued (Table 3). Of 130 cases, 119 (91.5%) demonstrated multiple histologic alterations. The most frequent findings included benign cysts (found in 75 of 130 cases), UD (74 cases), apocrine metaplasia (53 cases), and pseudoangiomatous stromal hyperplasia (47 cases). With regard to the imaging findings, there were some statistically significant differences between malignant and benign lesions, with malignant lesions demonstrating more often homogenous (P = .01) or clumped (P = .04) internal enhancement, type 3 kinetics (P = .01), and T2 dark signals (P < .001) as compared with the benign group and benign lesions demonstrating more often T1 bright (P = .02) and T2 bright (P = .02) or intermediate (P = .02) signals. However, there was considerable overlap between the imaging features of the 2 groups overall (Table 5).

Follow-up data were also collected and is outlined in Table 6. Of 130 cases, 54 (41.5%) had a subsequent excision specimen available in our system for review. Of these, 14 were from those with benign (non-atypical) lesions on the biopsy and 40 were from cases with atypical or higher lesions noted on the biopsy. A total of 6 cases (6 of 54; 11.1%) had a worse lesion identified on the subsequent excision, which included 1 case of complex sclerosing lesion found to have ADH on excision, 1 case of UDH found to

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**Table 3. All Histologic Alterations Catalogued for All Nonmass Enhancement Cases**

| Histologic Finding | N (%) |
|--------------------|-------|
| Benign/without atypia |       |
| Cysts | 75 (57.7) |
| Usual ductal hyperplasia | 74 (56.9) |
| Apocrine metaplasia | 53 (40.8) |
| Pseudoangiomatous stromal hyperplasia | 47 (36.2) |
| Columnar cell change/columnar cell hyperplasia | 42 (32.3) |
| Apocrine cysts | 32 (24.6) |
| Sclerosing adenosis | 23 (17.7) |
| Adenosis | 16 (12.3) |
| Papilloma | 14 (10.8) |
| Ectatic ducts | 11 (8.5) |
| Fibroadenoma/fibroadenomatous change | 10 (7.7) |
| Ducts with inspissated material | 9 (6.9) |
| Radial scar/complex sclerosing lesion | 8 (6.2) |
| Cyst/duct wall lining | 7 (5.4) |
| Vascular lesion | 4 (3.1) |
| Fat necrosis | 3 (2.3) |
| Lymphocytic mastopathy | 1 (0.8) |
| Mammary duct ectasia | 1 (0.8) |
| Spindle cell lesion | 1 (0.8) |
| None (breast tissue only) | 1 (0.8) |
| Atypical or higher |       |
| Flat epithelial atypia | 14 (10.8) |
| Atypical lobular hyperplasia | 9 (6.9) |
| Atypical ductal hyperplasia | 19 (14.6) |
| Lobular carcinoma in situ | 8 (6.2) |
| Ductal carcinoma in situ | 24 (18.5) |
| Invasive carcinoma | 7 (5.4) |

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**Table 4. Dominant (Most Prevalent) Histologic Finding for All Nonmass Enhancement Cases**

| Histologic Finding | N (%) |
|--------------------|-------|
| No dominant finding | 10 (7.8) |
| Benign/without atypia (94/130; 72.3%)* |       |
| Pseudoangiomatous stromal hyperplasia | 19 (14.6) |
| Usual ductal hyperplasia | 14 (10.8) |
| Apocrine cysts | 14 (10.8) |
| Sclerosing adenosis | 12 (9.2) |
| Papilloma | 6 (4.6) |
| Ectatic ducts | 5 (3.8) |
| Radial scar/complex sclerosing lesion | 5 (3.8) |
| Columnar cell change/columnar cell hyperplasia | 4 (3.1) |
| Vascular lesion | 4 (3.1) |
| Adenosis | 3 (2.3) |
| Apocrine metaplasia | 1 (0.8) |
| Cysts | 1 (0.8) |
| Fat necrosis | 1 (0.8) |
| Fibroadenoma/fibroadenomatous change | 1 (0.8) |
| Cyst/duct wall lining | 1 (0.8) |
| Lymphocytic mastopathy | 1 (0.8) |
| Mammary duct ectasia | 1 (0.8) |
| Spindle cell lesion | 1 (0.8) |
| Ducts with inspissated material | 0 (0.0) |
| Atypical or higher (40/130; 30.8%)* |       |
| Flat epithelial atypia | 2 (1.5) |
| Atypical lobular hyperplasia | 0 (0) |
| Atypical ductal hyperplasia | 6 (4.6) |
| Lobular carcinoma in situ | 2 (1.5) |
| Ductal carcinoma in situ | 24 (18.5) |
| Invasive carcinoma | 6 (4.6) |
| Total | 134* |

* Total of dominant lesions (134) is more than the total of cases (130) because some cases showed more than 1 dominant lesion. Some cases also showed no dominant finding (as noted above). Percentages are derived from dividing by the total number of cases (130).
### Table 5. Imaging Features of Malignant (Ductal Carcinoma In Situ or Invasive Carcinoma) Versus Benign (All Else) Lesions

| Imaging Characteristic                  | Benign Lesions, n = 101 (%) | Malignant Lesions, n = 29 (%) | P Value (If Significant) |
|----------------------------------------|-----------------------------|-------------------------------|--------------------------|
| Background enhancement pattern         |                             |                               |                          |
| Minimal                                | 11 (10.9)                   | 6 (20.7)                      |                          |
| Mild                                   | 42 (41.6)                   | 11 (37.9)                     |                          |
| Moderate                               | 35 (34.7)                   | 11 (37.9)                     |                          |
| Marked                                 | 13 (12.9)                   | 1 (3.4)                       |                          |
| Distribution                           |                             |                               |                          |
| Focal                                  | 56 (55.4)                   | 12 (41.4)                     |                          |
| Linear                                 | 32 (31.7)                   | 9 (31.0)                      |                          |
| Segmental                              | 6 (5.9)                     | 5 (17.2)                      |                          |
| Regional                               | 7 (6.9)                     | 3 (10.3)                      |                          |
| Multiple                               | 0 (0)                       | 0 (0)                         |                          |
| Diffuse                                | 0 (0)                       | 0 (0)                         |                          |
| Internal enhancement                   |                             |                               |                          |
| Homogenous                             | 6 (5.9)                     | 7 (24.1)                      | .01                      |
| Heterogeneous                          | 86 (85.1)                   | 14 (48.2)                     |                          |
| Clumped                                | 8 (7.9)                     | 7 (24.1)                      | .04                      |
| Clustered Ring                         | 1 (0.99)                    | 1 (3.4)                       |                          |
| Kinetics                               |                             |                               |                          |
| 0/No enhancement                       | 15 (14.9)                   | 0 (0)                         |                          |
| 1                                       | 26 (25.7)                   | 3 (10.3)                      |                          |
| 2                                       | 21 (20.8)                   | 3 (10.3)                      |                          |
| 3                                       | 9 (8.9)                     | 9 (31.0)                      | .01                      |
| Mixed                                   | 30 (29.7)                   | 14 (48.2)                     |                          |
| T1                                      |                             |                               |                          |
| Bright                                 | 34 (33.7)                   | 2 (6.9)                       | .02                      |
| Intermediate                            | 58 (57.4)                   | 23 (79.3)                     |                          |
| Dark                                    | 9 (8.9)                     | 4 (13.8)                      |                          |
| T2                                      |                             |                               |                          |
| Bright                                 | 40 (39.6)                   | 3 (10.3)                      | .02                      |
| Intermediate                            | 43 (42.6)                   | 3 (10.3)                      | .02                      |
| Dark                                    | 18 (17.8)                   | 23 (79.3)                     | <.001                    |
| Abbreviations: ADH, atypical ductal hyperplasia; DCIS, ductal carcinoma in situ. |

### Table 6. Cases With Follow-Up Excisions: Worst (Most Severe) Finding on Original Biopsy and If a Worse Lesion Was Found on the Excision

| Worst Histologic Finding on Original Biopsy | N (%) | Worse Lesion Present on Excision? |
|--------------------------------------------|-------|-----------------------------------|
| Benign (n = 14; 25.9%)                     |       |                                   |
| None                                       | 1 (1.9)|                                   |
| Pseudoangiomatous stromal hyperplasia      | 1 (1.9)|                                   |
| Vascular lesion                            | 1 (1.9)|                                   |
| Complex sclerosing lesion                  | 2 (3.7)| 1 (ADH)                           |
| Usual ductal hyperplasia                   | 4 (7.4)| 1 (DCIS)                          |
| Papilloma                                  | 5 (9.2)|                                   |
| Atypical or higher (n = 40; 74.1%)         |       |                                   |
| Flat epithelial atypia                     | 1 (1.9)|                                   |
| Atypical lobular hyperplasia               | 3 (5.5)|                                   |
| Atypical ductal hyperplasia                | 10 (18.5)| 1 (DCIS)                         |
| Lobular carcinoma in situ                  | 3 (5.5)|                                   |
| Ductal carcinoma in situ                   | 17 (31.5)| 3 (invasive carcinoma)          |
| Invasive carcinoma                         | 6 (11.1)|                                   |
| Total                                      | 54   | 6                                 |

Abbreviations: ADH, atypical ductal hyperplasia; DCIS, ductal carcinoma in situ.
have DCIS on excision, 1 case of ADH found to have DCIS on excision, and 3 cases of DCIS found to have invasive carcinoma on excision (1 of which was microinvasive).

DISCUSSION

In current practice, breast MRI is being used for a variety of indications including extent of disease evaluation in patients with newly diagnosed breast carcinoma, high-risk patient screening, and additional evaluation of indeterminate lesions. This is in part owing to its excellent depiction of lesion morphology and high sensitivity in detecting breast carcinomas. However, the specificity of breast MRI for detecting invasive carcinomas has been found to be only moderate as a substantial portion of enhancing lesions are found on histology to be benign. The pathologic correlates of enhancing breast lesions that are detected by MRI have not been extensively explored, particularly those of nonmass enhancing lesions, which comprise only approximately 15% of all MRI enhancing lesions. Consequently, the concordance between MRI and pathologic findings can be uncertain and this uncertainty may cause the need for additional procedures. Thus, the objective of this study was to investigate and catalogue the histologic alterations that correspond to nonmass enhancement detected by breast MRI.

Our results showed that the “worst” lesion in the majority of cases corresponding to nonmass enhancement were benign alterations or lesions (61.5% of the total cases). In contrast, 22.3% of cases (29 of 130) had DCIS or invasive carcinoma as their worst lesion with invasive cancer as the worst lesion in only 7 cases (5.4%). ADH accounted for another 9.2% (12 of 130) of cases. These findings are similar to those of 2 recent studies.33,34 Jabbar et al33 found that only a minority of their nonmass enhancing cases were found on histologic examination to show malignant lesions (14%, 11 of 76) or atypical epithelial hyperplasia (10%, 7 of 76). Yang34 reported that 22% (25 of 113) of his cases were associated with invasive carcinoma or DCIS and 66% (75 of 113) carried a benign diagnosis that included most commonly fibrocystic change and UDH. In contrast, in a study by Jansen et al,7 the majority of nonmass lesions correlated with malignant lesions (81.2%, 212 of 261). A study by Ballesio et al32 also demonstrated that the majority of their nonmass cases corresponded to malignant lesions (73.4%, 69 of 94) or atypical hyperplasia (4.3%, 4 of 94). In addition, a study by Bartella et al35 showed that 57% (39 of 68) of their invasive carcinomas showed nonmass enhancement by MRI. Thus, our study, in conjunction with the findings of other studies, illustrates the wide variety of benign to malignant lesions that can correspond with NME.

An additional finding in our study was that the majority of cases (91.5%; 119 of 130) demonstrated multiple histologic alterations. Yang also found that the majority of his cases (65%) demonstrated more than 1 pathologic finding.34 This highlights a complicating factor in evaluation of these cases. As multiple histologic alterations may be seen on a biopsy corresponding to NME detected by imaging, it is not entirely clear what histologic alteration or combination of alterations is “causing” that enhancement pattern. This is because enhancement detected on breast MRI reflects more the physiology and kinetics of the detected lesion rather than lesion morphology. Consequently, without an understanding of the physiology of these lesions, correlation of the histologic findings with the radiologic findings remains problematic. In an attempt to delineate the underlying “cause” for the NME in our cases, we had also subdivided the findings based on what was the most prevalent (or dominant) histologic alteration on each biopsy. However, no distinct trend was identified. Investigations into the biologic factors underlying enhancement of these lesions (such as microvessel density and distribution) could be of interest.36

With regard to the specific imaging findings, malignant lesions were significantly more often associated with homogeneous or clumped internal enhancement, type 3 kinetics, and T2 dark signal as compared with cases with benign lesions/alterations. Conversely, benign lesions were significantly more likely to have T1 bright, T2 bright, or T2 intermediate signals. However, there was considerable overlap between the MRI findings of benign and malignant cases. Other studies have found similar difficulties identifying radiologic characteristics that could reproducibly distinguish between benign and malignant lesions among cases presenting with NME. In particular, it has been noted that kinetic patterns of DCIS versus benign lesions can show considerable overlap.37–40 In addition, we found that radiologic-pathologic concordance, arguably one of the most important considerations, was difficult to assess in these cases. In all of our cases the histologic findings in the biopsies were ultimately considered to be “concordant” with imaging findings and were followed-up either clinically or with surgical biopsy depending on the histologic findings.

There are some notable limitations to this study. As stated above, radiologic-pathologic concordance was difficult to assess, which reflects the real-life difficulty of these cases in practice. At our institution, many of these cases are presented at our weekly breast radiology-pathology correlation conference. However, while radiologic-pathologic correlation to assess concordance remains prudent, the overlap between the imaging features between benign and malignant lesions complicates determination of the concordance. We have included the follow-up data for our cohort to at least partially address this, although this is also limited in that likely some patients received their follow-up care outside of our institution. Another limitation of this study is that there is likely a patient selection bias. While we have included all cases of NME that were biopsied during the study period, not all patients with NME are necessarily biopsied as other variables are considered (both clinical and imaging characteristics).

In conclusion, although MRI is a highly sensitive imaging modality, benign and malignant lesions demonstrate considerable radiologic overlap. Thus, in our view biopsy remains indicated for all suspicious NME lesions detected by MRI. Additional studies are needed to identify features of NME that can more reliably distinguish malignant from benign lesions.

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