Stratification of B3 lesions following radiology and pathology audit

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Purpose

There is an increasing focus on B3 lesions within the breast and their appropriate management. The heterogeneity of lesions within the B3 group means that the risk of malignancy varies widely between subgroups (1). It is known that underestimation of malignancy in this group is similar between 11G and 14G cores (2). However, with the availability of large bore, 8G vacuum assisted biopsy, the larger tissue samples produced have the potential to allow increased pre-operative pathological certainty. As such, wider sampling can both reduce the level of suspicion for malignancy and provide reassurance that the lesion sampled is benign, or allow appropriate first line cancer surgery if the lesion is upgraded to a B5 diagnosis pre-operatively.

With the aim of increasing pre-operative diagnosis rates for malignancy, whilst safely reducing unnecessary excision biopsies for benign disease, and following an internal audit of final pathology of all B3 lesions, we devised a new management pathway to stratify B3 lesions depending on their risk of malignancy.

Methods and Materials

A retrospective audit of all B3 lesions in the screened and symptomatic population, between September 2009 and August 2011 was performed. B3 lesions were identified following initial 11G/14G core biopsy. Lesions were subdivided into 6 pathological categories and followed to final pathological diagnosis to assess risk of upgrade to invasive or in situ carcinoma (B5 diagnosis).

The pathological categories were; radial scar/complex sclerosing lesion(RS/CSL); papillary lesions; atypical fibro-epithelial lesions; Atypical intra-ductal proliferations (AIDP)/ flat epithelial atypia (FEA); incidental lobular in situ neoplasia(LCIS) and other pathology(includes mucocoele/ spindle cell lesions/ infarction).

Within each pathological category, final excision biopsy pathology was reviewed to ascertain whether a subsequent B5 diagnosis was made. This information was used to create a risk profile for each pathological category upon which we based our proposed future management model.
Results

171 B3 lesions were identified over two years. This comprised 23 radial scars/complex sclerosing lesions (RS/CSL), 54 papillary lesions, 40 lesions comprising atypical intraductal proliferations (AIDP), flat epithelial atypia, or both, 15 lobular in situ neoplasia, 24 atypical fibroepithelial lesions and 15 cases of other pathology (mucocoele, spindle cell lesion etc).

Lesions were considered by type, allowing the initial development of lesion specific management flow charts (Fig. 1 on page 4, Fig. 2 on page 4, Fig. 3 on page 5, Fig. 4 on page 6). For example, of the 23 RS/CSL (Fig. 4 on page 6), there was only one which had ductal atypia and 2 were upgraded to B5 diagnosis following excision biopsy. This gave a risk of malignancy of 9%. As this is a small group size, our agreed radiologicopathological management for this group is large bore vacuum assisted biopsy for B3 with out ductal atypia and radiological surveillance if the lesion remains B3 with out ductal atypia or less. For lesions in this group with B3 with ductal atypia, either on first or second sampling, these are surgical excised.

A similar pathway was devised for papillary lesions (Fig. 2 on page 4). However, because of the larger group size, and the presence of lesions with atypical changes in conjunction with the papillary lesion, we were able to risk stratify these lesions depending on the presence or absence of ductal atypia. 54 papillary lesions were reviewed, of which 42 had no ductal atypia, and 12 had co-existing ductal atypia. Only 2 of the 42 lesions without ductal atypia, progressed to a malignant diagnosis (risk of 5%) compared to 7 of the 12 lesions with ductal atypia (59%). In addition, review of the pathological group containing purely atypical changes (AIDP/FEA), demonstrated that 18/40 patients with AIDP and/or FEA had a final malignant diagnosis, a risk of 45%. Within this group, 4 cases of FEA alone were not upgraded at final histology; however, the sample size is too small to treat FEA alone as a separate group.

Considering all lesion types with respect to the presence or absence of ductal atypia, there were 72 lesions with ductal atypia, of which 38% were upgraded, compared to 5% in those lesions without ductal atypia. Of lesions purely with ductal atypia present (AIDP, FEA or both), a 45% rate of upgrade to B5 on final pathology was observed.

Atypical fibroepithelial lesions have been excluded from our new management pathway and will continue to have surgical excision biopsy. This is because pathological diagnosis is dependent on sampling of the total lesion, and wide bore vacuum assisted biopsy would not further assist pathological diagnosis.
Fig. 1: Pathway for Calcification as the radiological finding; B3 with and without ductal atypia
Fig. 2: Pathway for Papillary lesions; B3 with and without ductal atypia
Fig. 3: Pathway for Mass Lesions (excluding papillary lesions); B3 with and without ductal atypia
Fig. 4: Pathway for deformity as the radiological lesion; B3 with and without ductal atypia
Fig. 5: Management of B3 lesions, with and without ductal atypia.
Conclusion

From this collaboration between radiology and pathology, and retrospective review of the previous management of B3 lesions in our institution, we have proposed an all encompassing management pathway (Fig. 5 on page 9), separating lesions with or without ductal atypia, and managing them in different ways. This is shown in our overall management of B3 lesions flow chart (Fig. 5 on page 9). Put simply, the low risk of malignant findings in patients with B3 lesions without ductal atypia, has reassured us that wider sampling should allow return of the screened patient to routine recall, provided that the vacuum assisted biopsy does not raise any further concern for malignancy or ductal atypia. In radial scars and papillary lesions with ductal atypia, we propose to continue straight to surgical excision in these patients. The rationale in this group is that pathological diagnosis requires sampling of the whole lesion, and a significant rate of upgrade to a B5 diagnosis (59%) was found. For all other cases with ductal atypia, a large bore vacuum assisted biopsy is proposed, with the aim of upgrading around 50% to B5 diagnosis, allowing appropriate first line cancer surgery. In those cases not upgraded by wider sampling, either 5 years annual surveillance will be instituted (in patients where atypical changes are found incidentally, and not in association with the initial radiological abnormality), or the patient will proceed to excision biopsy initially. It is hoped that future audit of excision biopsy in comparison to large bore vacuum assisted biopsy will clarify the accuracy of wider sampling, hopefully allowing us to safely omit excision biopsy of these lesions in this group of women in the future. Initial results from other units suggest that this management protocol would be an acceptable step to progress our future management and understanding of these complex breast lesions (3). This will be of particular importance with the introduction of digital mammography in our unit, which has been reported to increase the B3 biopsy rate (4).

In conclusion, B3 lesions remain a management challenge. However, large bore vacuum biopsy has the potential to clarify the management of subgroups of lesions within this group, and with time reduce the need for excision biopsy of lesions of uncertain malignant potential.

Images for this section:
Fig. 5: Management of B3 lesions, with and without ductal atypia.
References


