29 Common Consultation Conundrums in Breast Pathology

Sandra Shin MD

2011 Annual Meeting – Las Vegas, NV

AMERICAN SOCIETY FOR CLINICAL PATHOLOGY
33 W. Monroe, Ste. 1600
Chicago, IL 60603
29 Common Consultation Conundrums in Breast Pathology

Difficult or problematic breast lesions are commonly submitted to breast pathology consultants for a second opinion because of: 1) unusual, unexpected or ambiguous histological features and/or immunohistochemical staining results; 2) lack of consensus in lesion classification; 3) unclear reporting guidelines; or 4) rarity of the lesion. The purpose of this session is to provide pathologists with a practical approach to such problematic breast lesions that they will be able to apply in their daily practice. Discussion topics will include diagnostic problems with papillary, fibroepithelial, columnar cell and in situ lesions, microinvasion, small glandular proliferations, spindle cell lesions, and vascular lesions. Emphasis will be placed on the application and pitfalls of adjunctive immunohistochemical studies to resolve these differential diagnostic dilemmas.

- Correctly classify and diagnose problematic breast lesions.
- Understand the uses and limitations of immunostains in resolving diagnostic dilemmas in breast pathology.

FACULTY:

Sandra Shin MD
Practicing Pathologists
Surgical Pathology
Surgical Pathology (Derm, Gyn, Etc.)
2.0 CME/CMLE Credits

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COMMON CONSULTATION CONUNDRUMS IN BREAST PATHOLOGY

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ASSOCIATE PROFESSOR OF PATHOLOGY
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NEW YORK PRESBYTERIAN HOSPITAL-WEILL CORNELL MEDICAL COLLEGE

DISCLOSURE
• NONE

CONSULTATION CONUNDRUMS
THE VAST MAJORITY OF CONSULTATION CASES REPRESENTS THE PROBLEMATIC AREAS IN BREAST PATHOLOGY COMMON TO ALL PATHOLOGISTS
CONSULTATION CONUNDRUMS

- UNUSUAL, UNEXPECTED OR AMBIGUOUS HISTOLOGIC FEATURES AND/OR IMMUNOHISTOCHEMICAL RESULTS
- UNCLEAR REPORTING GUIDELINES
- LACK OF INTRADEPARTMENTAL CONSENSUS OF DIAGNOSIS
- RARITY OF LESION

CHALLENGING AREAS IN BREAST PATHOLOGY

- PAPILLARY LESIONS
- FIBROEPITHELIAL LESIONS
- SMALL GLANDULAR PROLIFERATIONS
- SPINDLE CELL LESIONS (includes VASCULAR)
- LOBULAR CARCINOMA IN-SITU (LCIS)
  - COLUMNAR CELL LESIONS
  - INVASIVE CARCINOMA
TOPIC 1 OF 5
PAPILLARY LESIONS

NO ONE’S FAVORITE BREAST LESION
• COMPLEX AND HETEROGENEOUS
• DO NOT FOLLOW A STEPWISE PROGRESSION OF INCREASING PROLIFERATIVE CHANGES AND “ATYPIA”
• GROSS TUMOR SIZE / CLINICAL SIGNS/SX NOT HELPFUL
• IMMUNOHISTOCHEMISTRY LESS HELPFUL IN CLASSIFYING THESE LESIONS (i.e. INVASIVE VS. IN-SITU)

HISTOLOGIC COMMONALITY OF PAPILLARY LESIONS
PAPILLARY, ARBORSCENT EPITHELIAL PROLIFERATION SUPPORTED BY FIBROVASCULAR STALKS WITH OR WITHOUT AN INTERVENING MYOEPITHELIAL CELL LAYER
SPECTRUM OF PAPILLARY LESIONS

- Papilloma with/without proliferative changes
- Papilloma with atypia
  - AKA... Atypical papilloma, papilloma with ADH, atypical papillary lesion
- Papilloma with DCIS
- Papillary (in-situ) carcinoma
  - Papillary DCIS
  - Encapsulated (intracystic) papillary carcinoma
  - Solid papillary carcinoma
- Invasive papillary carcinoma

PROBLEM AREAS

- Papilloma vs. papillary DCIS
- Papilloma with (florid DH vs. ADH vs. DCIS)
- Papilloma with florid DH vs. solid papillary carcinoma
- Encapsulated (intracystic) papillary CA
- Papillary lesions in needle core BXS
- In-situ vs. invasive papillary CA
PAPILLOMA VS. PAPILLARY DCIS

- Histologic overlap
  - Well-formed papillae with fibrovascular cores throughout lesion
  - Low nuclear grade
  - Intraductal/intracystic growth pattern with point of attachment
  - Variable size
INTRADUCTAL PAPILLOMA | PAPILLARY DCIS
---|---
CELL TYPE | EPITHELIAL AND MYOEPITHELIAL | EPITHELIAL
CELL ORIENTATION | HAPHAZARD | UNIFORM, ↑ TO FVC
NUCLEI | NORMOCHROMATIC | HYPERCHROMATIC
STROMA OF PAPILLAE | PROMINENT: FIBROSIS PRESENT | DELICATE
APOCRINE MET PROLIFERATION IN ADJ DUCTS | HYPERPLASIA | ABSENT

OTHER “SOFT” FINDINGS OF PAPILLOMAS
- MYOEPITHELIAL HYPERPLASIA
- APOCRINE METAPLASIA
- INFARCTION
- SQUAMOUS METAPLASIA
**DISTRIBUTION OF MYOEPITHELIAL CELLS**

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<thead>
<tr>
<th></th>
<th>WITHIN PAPILLAE</th>
<th>PERIPHERY</th>
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<tbody>
<tr>
<td>PAPILLOMA</td>
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<tr>
<td>PAPILLOMA WITH ADH/DCIS</td>
<td>PRESENT/ABSENT</td>
<td>PRESENT</td>
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<tr>
<td>PAPILLARY DCIS</td>
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<td>PRESENT</td>
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<tr>
<td>ENCAPSULATED PAPILLARY CA</td>
<td>ABSENT</td>
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<tr>
<td>SOLID PAPILLARY CA</td>
<td>ABSENT</td>
<td>PRESENT OR ABSENT</td>
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Collins LC and Schnitt SJ, Histopathol 2008
POSSIBLE PITFALLS OF MYOEPIHELIAL CELL MARKERS

- MEC MARKERS VARY IN SENSITIVITY AND SPECIFICITY - PANEL
- MISTAKEN FOR MEC
- PERICYTES
- MYOFIBROBLASTS IN THE STROMA
- NEOPLASTIC CELLS
- DISPLACED CARCINOMA CELLS CAN BE NEGATIVE FOR ME MARKERS
PROBLEM AREAS
- PAPILLOMA VS. PAPILLARY DCIS
- PAPILLOMA WITH (FLORID DH VS. ADH VS. DCIS)
- PAPILLOMA WITH FLORID DH VS. SOLID PAPILLARY CARCINOMA
- ENCAPSULATED (INTRACYSTIC) PAPILLARY CA
- PAPILLARY LESIONS IN NEEDLE CORE BXs
- IN-SITU VS. INVASIVE PAPILLARY CA
SOLID PAPILLARY CARCINOMA
**PAPILLOMA WITH FLORID DUCT HYPERPLASIA**

**SOLID PAPILLARY CARCINOMA**

**DISTRIBUTION OF MYOEPITHELIAL CELLS**

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<td>Papilloma (+/-FDH)</td>
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<tr>
<td>Papilloma with ADH/DCIS</td>
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<td>Papillary DCIS</td>
<td>Absent</td>
<td>Present</td>
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<tr>
<td>Encapsulated Papillary CA</td>
<td>Absent</td>
<td>Absent</td>
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<tr>
<td>Solid Papillary Carcinoma</td>
<td>Absent</td>
<td>Present or Absent</td>
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**NORMAL DUCT**

**ATTENUATED & ABSENT STAINING**

**p63**
### OTHER USEFUL IMMUNOSTAINS

<table>
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<tr>
<th></th>
<th>Papilloma</th>
<th>Papilloma Solid Pap</th>
<th>Solid Papilloma Adenosis/DCIS</th>
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<tr>
<td>HMWCK (CK5/6; K903)</td>
<td>+ FDH</td>
<td>+ Adenosis/DCIS</td>
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<tr>
<td>ER</td>
<td>FEW POS</td>
<td>NEG/POS</td>
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<td>NE Markers (SYNAPTO, CD56)</td>
<td>NEG</td>
<td>NE/POS</td>
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### FLORID DUCT HYPERPLASIA

- **CK 5/6**

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### SOLID PAPILLARY CARCINOMA

- **CK 5/6**

**Note:**

OTHER USEFUL IMMUNOSTAINS

<table>
<thead>
<tr>
<th></th>
<th>PAPILLOMA + FDH</th>
<th>PAPILLOMA + ADH/DCIS</th>
<th>SOLID PAP CARCINOMA</th>
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<td>HMWCK (CK5/6; K903)</td>
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**NEEDLE CORE BIOPSY**
NEUROENDOCRINE MARKERS

- Most commonly positive in solid papillary carcinoma
- Also positive in papillary DCIS and encapsulated papillary carcinoma (lesser extent)
- Synaptophysin, chromogranin, CD56
- Neuron specific enolase – not recommended

PROBLEM AREAS

- Papilloma vs. papillary DCIS
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- Papilloma with florid DH vs. solid papillary carcinoma
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NEEDLE CORE BIOPSY

DISTRIBUTION OF MYOEPITHELIAL CELLS

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Collins LC and Schnitt SJ, Histopathol 2008
ENCAPSULATED (INTRACYSTIC) PAPILLARY CARCINOMA
- ? IN-SITU OR LG INVASIVE CA
- MORPHOLOGICALLY SIMILAR METASTATIC FOCI
- PRECURSOR TO MUCINOUS CARCINOMA BUT ALSO OTHER TYPES OF INVASIVE DUCT CA
- INDOLENT BEHAVIOR WITHOUT CONCURRENT INVASIVE COMPONENT
- EVEN WITH INVASION, PROGNOSIS IS FAfavorable compared to invasive duct CA of comparable size

PROBLEM AREAS
- PAPILLOMA VS. PAPILLARY DCIS
- PAPILLOMA WITH (FLORID DH VS. ADH VS. DCIS)
- PAPILLOMA WITH FLORID DH VS. SOLID PAPILLARY CARCINOMA
- ENCAPSULATED (INTRACYSTIC) PAPILLARY CA
- PAPILLARY LESIONS IN NEEDLE CORE BXES
- IN-SITU VS. INVASIVE PAPILLARY CA
DIAGNOSTIC PROBLEMS MAGNIFIED IN NEEDLE CORE BXs

- Morphologic pitfalls due to small sample size
  - Entrapped glands of a sclerosing papillary lesion mimic invasive duct carcinoma
- Atypia, if present, can be focal
- Tissue fragmentation
- Rendering a final dx of a papillary lesion on NCB is virtually impossible

ALL PAPILLARY LESIONS

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<th>CNB diagnostic category</th>
<th>No. of cases</th>
<th>No. excised (%)</th>
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<tr>
<td>B2 BENIGN</td>
<td>49</td>
<td>40 (81.6)</td>
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<tr>
<td>B1 FDHUS/ADH</td>
<td>10</td>
<td>10 (100)</td>
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<tr>
<td>B4 SUSP DCIS</td>
<td>3</td>
<td>2 (66.7)</td>
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<tr>
<td>B5 DCIS/INV CA</td>
<td>67</td>
<td>56 (83.6)</td>
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<tr>
<td>Total</td>
<td>129</td>
<td>108 (83.7)</td>
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Pathologic upgrade on EXBX = 4%

CONCLUSIONS - SHAH ET AL STUDY

- With IHC, accuracy of diagnosis increased for all 4 observers
  - PPV 78-88%
  - NPV 100%
  - Accuracy 91-95%
- IHC most helpful to least experienced observer
- IHC permitted reclassification of all B3a cases into one of two more clinically useful categories (B2 or B3b/4/5)
ER-high/CK5-low immunoprofile identifies atypical papillary lesions at a sensitivity of 93% and specificity of 100%

ER-low/CK5-high immunoprofile identified non-atypical papillary lesions at a sensitivity of 100% and specificity of 93%

PROBLEM AREAS
- PAPILLOMA VS. PAPILLARY DCIS
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- PAPILLOMA WITH FLORID DH VS. SOLID PAPILLARY CARCINOMA
- ENCAPSULATED (INTRACYSTIC) PAPILLARY CA
- PAPILLARY LESIONS IN NEEDLE CORE BXs
- IN-SITU VS. INVASIVE PAPILLARY CA
TOPIC 2 OF 5
SMALL GLANDULAR PROLIFERATIONS

HISTORY
- 59 YEAR-OLD FEMALE
- BREAST NODULE - RETROAREOLAR
- NO OTHER SIGNIFICANT CLINICAL HX
- NEEDLE CORE BIOPSY PERFORMED

2011 ASCP Annual Meeting
DIFFERENTIAL DIAGNOSIS

- INVASIVE WELL-DIFFENTIATED DUCT CARCINOMA
- TUBULAR FEATURES
- SYRINGOMATOUS ADENOMA
- LOW-GRADE ADENOSQUAMOUS CARCINOMA
- RADIAL SCLEROSING LESION
- COMPLEX SCLEROSING LESION
- SCLEROSING ADENOSIS
- SKIN ADNEXAL CARCINOMA
INVASIVE TUBULAR CARCINOMA?

ADENOSIS?

RADIAL SCLEROSING LESION?

INVASIVE TUBULAR CARCINOMA?

INVASIVE TUBULAR CARCINOMA ARISING IN RSL OR ENTRAPPED/PARTICIPATING BENIGN GLANDS

RADIAL SCLEROSING LESION
HOW WE LEARN PATHOLOGY

- Most didactic lectures are focused around specific entities
- Pattern – based learning may be more useful, esp in certain settings such as core needle biopsies
  - Pathologic findings are fragmented
  - Diagnostic features are missing
  - The spectrum of benign, atypical and malignant entities can have the same pattern

ARRIVING AT THE CORRECT DX

- Pattern recognition - DDX
- Algorithm
- When we get stuck…
  - Use adjunctive tools (IHC)
  - Consult a colleague
  - Both

ADJUNCTIVE DIAGNOSTIC TOOLS

- Immunohistochemistry
- Special stains
- Molecular characterization
  - FISH
  - Gene profiling
- Electron microscopy
SMALL GLANDULAR PROLIFERATIONS OF THE BREAST (SGPB)

- MALIGNANT-APPEARING BUT BENIGN
- BENIGN-APPEARING BUT MALIGNANT
- MALIGNANT-APPEARING BUT MALIGNANT OF A DIFFERENT TYPE

CASE 1 continued…

DIAGNOSIS RENDERED

- INVASIVE DUCT CARCINOMA, WELL-DIFFERENTIATED
- RE-REVIEWED AT INSTITUTION WHERE SURGERY WAS PLANNED
- NEW DIAGNOSIS: SYRINGOMATOUS ADENOMA
- NO SURGERY PERFORMED
5 YEARS LATER…2011
- BREAST MASS GROWN 5 CM LARGE; NEEDS MASTECTOMY IF MALIGNANT
- NEEDLE CORE BIOPSY PERFORMED
2011 ASCP Annual Meeting

LOW-GRADE ADENOSQUAMOUS CARCINOMA

2011 ASCP Annual Meeting

29 CASES OF LGASC
- MYOEPITHELIAL MARKERS (p63, SMM, CD10, CALPONIN, SMA)
- CYTOKERATINS (CKAE1/3, CK7, Cam 5.2, CK5/6, K903)
- GLANDULAR EPITHELIUM AND ADJACENT STROMA STUDIED
RESULTS- MYOEPITHELIAL MARKERS

GLANDULAR EPITHELIUM

CIRCUMFERENTIAL STAINING IN MOST (>80%) CASES WITH EITHER COMPLETE (~75%) OR WEAK, DISCONTINUOUS (~35%) STAINING USING ANY ONE STAIN. OCCASIONALLY GLANDS ARE NEGATIVE

ADJACENT STROMA

LAMELLAR STAINING ~45% OF CASES USING ANY ONE STAIN

DIFFUSE STROMAL POSITIVITY IN >50% (CALPONIN, CD10, SMA); ~12.5% (SMM); 0 (p63)

LUMINAL CELL STAINING

p63

VARIABLY POSITIVE IN BASALLY LOCATED CELLS

SMOOTH MUSCLE MYOSIN

VARIABLY POSITIVE IN BASALLY LOCATED CELLS

“LAMELLAR” STAINING PATTERN
RESULTS - CYTOKERATIN MARKERS

GLANDULAR EPITHELIUM
POSITIVE FOR ONE OR MORE STAINS IN ALL CASES
DIFFUSE WITH EITHER UNIFORM OR VARIABLY
INTENSE INTENSITY IN >75% OF CASES

CORE STAINING IN 25% TO 67% USING ANY ONE STAIN

ADJACENT STROMA
UNIFORMLY NEGATIVE IN ALMOST ALL CASES

“CORE” STAINING PATTERN

HMW-CK
K903
OTHER SGPB TO CONSIDER IN CORE NEEDLE BIOPSIES

- INVASIVE DUCT CARCINOMA (WELL-DIFFERENTIATED, TUBULAR TYPE)
- RADIAL SCLEROSING LESION
- ADENOSIS/SCLEROSING ADENOSIS/TUMOR
- MICROGLANDULAR ADENOSIS

MORPHOLOGIC FEATURES

IFDC (TUBULAR) VS LGASC

- WELL-FORMED GLANDS
- HAPHAZARD, INFILTRATIVE ARRANGEMENT
- ROUND, OVAL, OR TEAR DROP-SHAPED GLANDS
- LOW NUCLEAR GRADE
- ELASTOTIC STROMA

- WELL-FORMED GLANDS
- HAPHAZARD, INFILTRATIVE ARRANGEMENT
- ANGULATED GLANDS +/- SQUAMOUS DIFFERENTIATION
- LOW NUCLEAR GRADE
- PERI-GLANDULAR SPINDLE CELL METAPLASIA IN STROMA
- LYMPHOCYTIC AGGREGATES
IMMUNOHISTOCHEMICAL FEATURES

IFDC VS LGASC

- NEGATIVE FOR MYOEPIHELIAL MARKERS (P63, SMM, CALPONIN)
- POSITIVE FOR ER/PR (STRONG, DIFFUSE)
- STROMA - NEGATIVE
- CIRCUMFERENTIALLY POSITIVE (VARIABLE) FOR MYOEPIHELIAL MARKERS; OCCASIONAL NEGATIVE GLAND(S)
- "LAMELLAR" POSITIVITY FOR MYOEPIHELIAL MARKERS (SMM) IN SPINDLE CELL STROMA
- P63 ALSO POSITIVE IN EPITHELIUM WITH SQUAMOUS DIFFERENTIATION
- POSITIVE (DIFFUSE, VARIABLE INTENSITIES) FOR MOST CYTOKERATINS
- CORE STAINING FOR SOME CYTOKERATINS (HMWCK)
- ER/PR NEGATIVE
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<th>MORPHOLOGIC FEATURES</th>
<th>IFDC (TUBULAR)</th>
<th>VS</th>
<th>RSL</th>
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<td>LOW NUCLEAR GRADE</td>
<td>CO-EXISTING CCL, ADH, LOBULAR LESIONS</td>
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<td>PARTICIPATING</td>
<td>ELASTOTIC STROMA</td>
<td>+/- HEAVY SCLEROSIS IN NIDUS</td>
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MORPHOLOGIC FEATURES

IFDC (TUBULAR) VS SA

- WELL-FORMED GLANDS
- HAPHAZARD ARRANGEMENT
- ROUND, OVAL, OR TEAR-DROP SHAPED GLANDS
- ELASTOTIC STROMA
- WELL-FORMED GLANDS
- LOBULOCENTRIC BUT MAY NOT BE APPARENT ON CNB
- PSEUDINFILTRATIVE GROWTH PATTERN WITH INCREASING SCLEROSIS
- CAN BE SECONDARILY INVOLVED BY ALH, LCIS, ADH, DCIS
- ASSOCIATED CALCS; MASS FORMING IF ADENOSIS TUMOR
IS THIS TUBULAR CARCINOMA ARISING IN A RADIAL SCLEROSING LESION?

IMMUNOHISTOCHEMICAL FEATURES

IFDC VS RSL/SA

- NEGATIVE FOR MYOEPITHELIAL MARKERS (P63, SMM, CALPONIN)
- POSITIVE FOR ER/PR (STRONG, DIFFUSE)

- POSITIVE FOR MYOEPITHELIAL MARKERS (P63, SMM, CALPONIN)
- BUT CAN BE VERY ATTENUATED OR ABSENT IN AREAS OF MARKED SCLEROSIS
- ER/PR POSITIVE BUT NOT DIFFUSELY
NOT ALL MYOEPIHELIAL MARKERS ARE CREATED EQUAL

- DIFFER IN SENSITIVITIES AND SPECIFICITIES
- SOME ALSO STAIN MYOFIBROBLASTS (i.e. SMOOTH MUSCLE ACTIN, CD10)
- SOME ARE LESS ROBUST IN AREAS OF HEAVY SCLEROSIS; FALSE NEGATIVITY
- RECOMMEND USING A PANEL OF MYOEPIHELIAL MARKERS
  - P63, SMOOTH MUSCLE MYOSIN, CALPONIN

SMM-HC

SMOOTH MUSCLE ACTIN
INVASIVE CARCINOMA WITH TUBULAR AND LOBULAR FEATURES
BIOMARKER STAINS PENDING
MICROGLANDULAR ADENOSIS GIVING RISE TO INVASIVE CARCINOMA
MORPHOLOGIC FEATURES
IFDC (TUBULAR) VS MGA

- INFILTRATIVE WELL-FORMED GLANDS
- ROUND, OVAL OR TEAR-DROP SHAPED GLANDS
- LOW NUCLEAR GRADE
- ELASTOTIC STROMA
- IN-SITU COMPONENT IS ABSENT OR ADH/LG DCIS
- CO-EXISTING CCL AND/OR LOBULAR LESIONS

- INFILTRATIVE WELL-FORMED GLANDS
- ROUND GLANDS
- LOW NUCLEAR GRADE
- BRIGHT EOSINOPHILIC LUMINAL SECRETIONS
- STROMA IS UNALTERED
- NO IN-SITU COMPONENT UNLESS ATYPICAL
IMMUNOHISTOCHEMICAL FEATURES

IFDC VS MGA

- NEGATIVE FOR MYOEPITHELIAL MARKERS (P63, SMM, CALPONIN)
- POSITIVE FOR ER/PR (STRONG, DIFFUSE)

- NEGATIVE FOR MYOEPITHELIAL MARKERS (P63, SMM, CALPONIN)
- NEGATIVE FOR ER/PR
- POSITIVE FOR BASEMENT MEMBRANE MARKERS (RETICULIN, LAMININ, COLLAGEN TYPE IV)
- POSITIVE FOR S-100 PROTEIN
INVASIVE LOBULAR CARCINOMA FOCALLY INVOLVING A RADIAL SCLEROSING LESION
RADIAL SCLEROSING Lesion (RSL)

- Occurrence of invasive or in-situ carcinoma involving RSL or in close proximity of RSL is well known
- Rates 0-34%
- Easily underdiagnosed on NCB since involvement by carcinoma of a RSL is focal or peripheral

TOPIC 3 OF 5
FIBROEPITHELIAL Lesions

- Fibroadenoma (FA)
  - Variants (cellular, juvenile, complex, giant)
- Phyllodes Tumor (PT)
  - Grades (benign, borderline, malignant)
- Fibroadenomatoid Mastopathy
  (Sclerosing Lobular Hyperplasia)
FIBROADENOMATOID MASTOPATHY

MANAGEMENT

- FIBROADENOMAS
  - Simple enucleation
  - Clinical follow-up if small

- PHYLLODES TUMOR
  - Wide local excision (1cm or more?) for PT (all grades?) compliance by surgeons?
  - Residual PT at margins is a strong predictor of local recurrence; re-excision is recommended

BENIGN PT CALLED FA ON CNB

- Clinical F/U only
- Enucleation (unoriented specimen)
- EXBX if clinically worsening
- Potentially higher grade PT in unsampled areas; less favorable cosmetic result if large
- Positive undesignated margins in the EXBX
- Re-excision of undesignated margin(s)
- Potentially more tissue excised

Psychological +/- physical morbidity of pt
PHYLLODES TUMORS

- Rare compared to FA (2-3% of FEL tumors of the breast)
- Age 35-55 females
- Breast mass +/- rapid growth
- Size at presentation larger than that of FA but difference is shrinking due to screening
  - 1.3cm (PT) vs. 1.0cm (FA) [Komenaka et al. Arch Surg 2003]
- Clinically and radiologically indistinguishable from FA
- Local recurrence rates: Benign (8%), BL (29%), HG (36%)
- Metastatic potential: Benign (0%), BL (<4%), HG (<22%)

MORPHOLOGIC FEATURES

- Leaf-like growth pattern
- Stroma (cellularity, atypia, overgrowth)
- Interfield variability of gland to stroma ratio
- Tumor border (invasive)
- Stromal mitotic figures
- Pseudoangiomatous stromal hyperplasia
- Excessive epithelial hyperplasia (micropapillary)

Pseudopapillary Growth Pattern in Phyllodes Tumor

Fibroadenoma-intracanalicular type
BENGIN PHYLLODES TUMOR
MYXOID STROMA

FIBROADENOMA
WITH
MYXOID STROMA

Stromal Mitoses
INFILTRATIVE TUMOR BORDER

MICROPAPILLARY DUCT HYPERPLASIA IN PHYLLODES TUMOR

FA or Fibroadenomatous areas in PT?
### Initial and Final Diagnoses

<table>
<thead>
<tr>
<th>Core Biopsy Result</th>
<th>No. of Patients</th>
<th>Fibroadenoma</th>
<th>Phyllodes Tumor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Favor fibroadenoma</td>
<td>25</td>
<td>23</td>
<td>2</td>
</tr>
<tr>
<td>Favor phyllodes tumor</td>
<td>23</td>
<td>4</td>
<td>19</td>
</tr>
<tr>
<td>Equivocal</td>
<td>9</td>
<td>5</td>
<td>4</td>
</tr>
<tr>
<td>Total</td>
<td>57</td>
<td>32</td>
<td>25</td>
</tr>
</tbody>
</table>

NPV 93%
PPV 83%


### Jacobs, et al.
AJCP 2005;124:342-354
- 29 pts with NCB containing FEL with cellular stroma who had subsequent EXBX
- "cellular stroma" = 2x normal perilobular stroma
- Surgical outcome:
  - 16 (55%) were FA
  - 12 (41%) were PT (5-benign,6-LGM,1-HG)

### 10 HISTOLOGIC FEATURES

- Stromal cellularity
- Stromal nuclear atypia
- Stromal mitotic count
- Stromal proportion to epithelium
- Stromal overgrowth
- Enhancement of stromal cellularity adjacent to epithelium
- Infiltrative tumor border
- Epithelial hyperplasia
- Overall growth pattern
- Multinucleated giant cells
10 HISTOLOGIC FEATURES

- Stromal cellularity
- Stromal nuclear atypia
- Stromal mitotic count
- Stromal proportion to epithelium
- Stromal overgrowth
- Enhancement of stromal cellularity adjacent to epithelium
- Infiltrative tumor border
- Epithelial hyperplasia
- Overall growth pattern
- Multinucleated giant cells

Histopathology 2007;51:336-344

Histological features useful in the distinction of phyllodes tumour and fibroadenoma on needle core biopsy of the breast

A H H Lee, P Folee, E S Wells, C W Elston
Anatomical Pathology, Massachusetts General Hospital, Boston, MA

Histological features useful in the distinction of phyllodes tumour and fibroadenoma on needle core biopsy of the breast

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Anatomical Pathology, Massachusetts General Hospital, Boston, MA

Seven histologic features significantly different b/t FA and PT

- Stromal cellularity >50% of core
- Marked stromal pleomorphism
- Stromal overgrowth (x10 field)
- Edge-infiltrative (only when edge can be adequately assessed in ncb)
- Fragmentation
- Adipose tissue in stroma (invasive tumor border vs lipomatous metaplasia)
- Stromal mitoses
Seven histologic features significantly different b/t FA and PT

- Stromal cellularity >50% of core
- Marked stromal pleomorphism
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- Edge-infiltrative (only when edge can be adequately assessed in ncb)
- Fragmentation
- Adipose tissue in stroma (invasive tumor border vs lipomatous metaplasia)
- Stromal mitoses

Analysis of histological features in needle core biopsy of breast useful in preoperative distinction between fibroadenoma and phyllodes tumour

J Mike Morgan, Anthony O Douglas-Jones & Sajidee E Gupta

Department of Histopathology, University Hospital of Wales, Cardiff. "Cardiff University, Health Park Campus, Cardiff, UK, and Institute of Medical Sciences, Bhum, Varsan, India"

112 NCB of FEL
(21 PT AND 91 FA; ALL WITH EXBX)
RECEIVER-OPERATING CHARACTERISTIC (ROC) ANALYSIS

- Objective method for determining optimal cut-off values in frequency distributions for 2 datasets with one variable (tumor type).

- CUT OFF VALUES FOR PT OVER FA
  - AGE (50-55)
  - PERCENT STROMA (85-90)
  - MITOSES (≥ 1 PER 2.2 MM²)

-Confirmed findings of Lee, et al.
Predictors of phyllodes tumours on core biopsy specimens of fibroepithelial neoplasms

Ana Rochello Jave Lezama, Meenakshi Adbineth, Aye Aye Thike, Philip Ch-Wai Lee
Gary Mao-Ki Teo, & Ping Hoon Tan
Department of Pathology, St John’s General Hospital, Singapore, and Institute of Analytical and Cellular Pathology, Chinese University of Hong Kong, Prince of Wales Hospital, Hong Kong, SAR, China

261 NCB of FEL
98 (37%) FEL CANNOT EXCLUDE PT
57/98 (58%) UNDERWENT EXBX

MORPHOLOGIC FEATURES
• EXCLUSIVE FEATURES OF PT
  • MARKED STROMAL CELLULARITY
  • MARKED STROMAL ATYPIA
  • MITOSES ≥ 2 / 10 HPF
  • ILL-DEFINED LESIONAL BORDER
• HIGH CORRELATION WITH PT ON EXBX
  • MODERATE STROMAL CELLULARITY
  • STROMAL OVERGROWTH
  • MODERATE STROMAL ATYPIA
  • PASH

SUMMARY OF STUDIES
• MORPHOLOGIC FEATURES OF PT
  • STROMAL CELLULARITY (MODERATE TO MARKED)
  • STROMAL ATYPIA
  • STROMAL MITOSES (AT LEAST 1)
  • STROMAL OVERGROWTH
  • INFILTRATIVE TUMOR BORDER
SPINDLE CELL LESIONS

SIMILAR TO SMALL GLANDULAR PROLIFERATIONS, SPINDLE CELL LESIONS CONSISTS OF A SPECTRUM OF BENIGN TO MALIGNANT ENTITIES WHICH SHARE AN OVERALL SIMILAR MORPHOLOGIC APPEARANCE.

FORMULATING AN ACCURATE DX ON CORE NEEDLE BIOPSIES CAN BE DIFFICULT IF NOT IMPOSSIBLE.

CELLS OF ORIGIN

- FIBROBLASTS, MYOFIBROBLASTS
  - INTRA- AND INTERLOBULAR MAMMARY STROMA
  - POSITIVE VIMENTIN, BCL-2, CD-99
  - MYOFIBROBLASTS ALSO α-SMOOTH MUSCLE ACTIN, DESMIN, HORMONE RECEPTORS
- EPITHELIAL
- MYOEPIHELIAL
- HISTIOCYTES
- ENDOTHELIAL CELLS

SPINDLE CELL LESIONS

CYTOLOGICALLY BLAND-APPEARING LESIONS
  - BENIGN
  - MALIGNANT

CYTOLOGICALLY ATYPICAL LESIONS
  - MALIGNANT
CYTOLOGICALLY BLAND SPINDLE CELL LESIONS DIFFERENTIAL DIAGNOSIS
- PASH
- FASCICULAR
- TUMOROUS
- MYOFIBROBLASTOMA
- BENIGN PHYLLODES TUMOR (STROMA ONLY)
- SCAR
- REACTIVE SPINDLE CELL NODULE
- ADENOMYOEPITHELIOMA
- SPINDLE CELL LIPOMA
- NEUROFIBROMA
- LEIOMYOMA

CYTOLOGICALLY ATYPICAL SPINDLE CELL LESIONS: DIFFERENTIAL DIAGNOSIS
- NODULAR FASCIITIS
- PHYLLODES TUMOR (STROMA ONLY)
  - BORDERLINE, HIGH GRADE
- METASTASES
  - MELANOMA
  - SARCOMA
    - PRIMARY (HIGH-GRADE ANGIOSARCOMA)
    - METASTATIC

OTHER CO-EXISTING CELLS
- RED BLOOD CELLS - ANGIOSARCOMA
- MIXED INFLAMMATORY INFILTRATE & RED BLOOD CELLS – NODULAR FASCIITIS
- LYMPHOCYTES
  - PERIPHERAL AGGREGATES – FIBROMATOSIS
  - DISPERSED IN BACKGROUND – SPINDLE CELL METAPLASTIC CARCINOMA, REACTIVE SPINDLE CELL NODULE
- UNINVOLVED BREAST TISSUE OR ADIPOSE TISSUE PRESENT?
CLINICAL AND RADIOLOGIC CORRELATION

PAST CLINICAL HISTORY
- RECENT HISTORY OF TRAUMA/NEEDLE BX (SPINDLE CELL NODULE)
- BREAST CARCINOMA AND STATUS POST RADIATION THERAPY (POST-RADIATION ANGIOSARCOMA)
- OTHER MALIGNANCIES (I.E. MELANOMA)
- FAMILIAL ADENOMATOUS POLYPOSIS OR GARDNER’S SYNDROMES (FIBROMATOSIS)
- "BRUISE" ON BREAST SKIN (ANGIOMATOSIS)

TUMOR CHARACTERISTICS
- SOLITARY (PHYLODES TUMOR, MYOFIBROBLASTOMA, MYOEPITHELIOMA, TUMOROUS PASH)
- WELL-CIRCUMSCRIBED (MYOFIBROBLASTOMA, ADENOMYOEPITHELIOMA)
- INFILTRATIVE BORDERS (FIBROMATOSIS, SOME PHYLODES TUMORS)
- SMALL SIZE (NODULAR FASCITIS, REACTIVE SPINDLE CELL NODULE)
- SUPERFICIAL LOCATION (SPINDLE CELL LIPOMA, DFSP, DF)
- NIPPLE LOCATION (LEIOMYOMA)
MYOFIBROBLASTOMA

- Circumscribed +/- lipomatous differentiation
- Short fascicles of uniform spindle-shaped cells with round to oval nuclei; mitoses very rare
- Hyalinized collagen bands
- Mast cells, myxoid change, chondroid or smooth muscle metaplasia
- Variants exist...most impt, epithelioid confuse with infiltrating carcinoma
- CD 34 POSITIVE +/- SMOOTH MUSCLE ACTIN, DESMIN; S-100 NEG (unlike spindle cell lipoma)
- ER, PR, BCL-2, VIMENTIN

MYOFIBROBLASTOMA-EPITHELIOID VARIANT
FIBROMATOSIS
- Painless, slowly growing mass
- Stroma vary from keloidal to myxoid/fasciitis like
- SMA positive; +/- desmin, S-100
- High rate of local recurrence
- Difficult or impossible to distinguish between fibromatosis and scar in re-excision
β-catenin/Wnt signalling pathway in fibromatoses, metaplastic carcinomas, and phylloides tumours of the breast
Magzil Saqiri, PhD,*†‡, Fabio C Genoni,* Maria B Lanzino,* Kay Serag,* Ian O Ellis,‡ and Jamie C. East*§

*Biological Pathology: Mechanisms Department, Institute of Cancer Research, London, UK
†The Breakthrough Breast Cancer Research Centre, Institute of Cancer Research, London, UK
‡Biological Pathology: Mechanisms Department, Institute of Cancer Research, London, UK
§Department of Pathology, School of Medicine, University of Washington, Seattle, WA, USA

100% FIBROMATOSIS

BUT ALSO...
23% METAPLASTIC CARCINOMA
94% BENIGN PT
57% MALIGNANT PT
<table>
<thead>
<tr>
<th>CASE 1</th>
<th>CK7</th>
<th>CAM5.2</th>
<th>K903</th>
<th>P63</th>
</tr>
</thead>
<tbody>
<tr>
<td>+</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>CASE 2</td>
<td>+</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>+</td>
<td></td>
<td>+</td>
<td>+</td>
<td></td>
</tr>
<tr>
<td>CASE 3</td>
<td>+</td>
<td></td>
<td>-</td>
<td>+</td>
</tr>
</tbody>
</table>

*Positive staining can be very focal
*p63 is diffusely positive if one CK is also
*Always perform a panel of CK markers to rule in/out this entity
*A negative immunopanel on a CNB does not exclude this dx
SPINDLE CELL METAPLASTIC CARCINOMA

- Morphologic clues before IHC
  - Admixed invasive duct carcinoma, NOS
  - Presence of DCIS
  - Spindle cells that aggregate and contain more cytoplasm/larger nuclei ("epithelioid")
  - Areas of squamous differentiation
  - Scattered chronic inflammation in the background ("dirty background")
LOW-GRADE FIBROMATOSIS-LIKE SCMC
- Composed of bland spindle cells similar to those seen in fibromatosis
- Can see areas that are more epithelioid +/- squamous differentiation
- High rate of local recurrence
- Metastasis is rare

What do the findings in the next slides have in common?
THEY REPRESENT DIFFERENT AREAS OF THE SAME CASE
Hemangiomas and Angiosarcomas of the Breast
Diagnostic Utility of Cell Cycle Markers With Emphasis on Ki-67

Sandra J. Yiu, M.D.; Martin Isaac, Ph.D.; Hal Peris, M.D.

Contact—Lobular lesions comprise a minor subgroup of tumors arising in the breast and represent variable subsets of hemangiomas and angiosarcomas. Diagnostic challenges may arise when differentiating hemangiomas from type-I angiosarcomas, which may be difficult to distinguish. This study aimed to evaluate Ki-67 and other cell cycle markers in a series of hemangiomas, angiosarcomas, and lobular lesions.

Objective—To investigate the utility of Ki-67 and other cell cycle markers in the differential diagnosis of hemangiomas, angiosarcomas, and lobular lesions.

Methods—Hemangiomas (21), angiosarcomas (11), and lobular lesions (20) were studied. Ki-67, p53, p21, and p16 expression were evaluated. The mean values of Ki-67 at four different nuclear levels were determined.

Results—The mean value of Ki-67 at each level was statistically different when comparing hemangiomas and angiosarcomas (p < 0.001). Ki-67 expression was negative in hemangiomas (n = 21), whereas hemangiomas were negative (p < 0.001). Sensitivity and specificity values for Ki-67 were 100% and 95%, respectively, for the diagnosis of hemangiomas. The mean values of Ki-67 at four different nuclear levels were compared among the three groups.

Conclusions—Ki-67 may be used as a diagnostic tool to distinguish between hemangiomas and angiosarcomas. The mean values of Ki-67 at four different nuclear levels were compared among the three groups.
Loss of E-cadherin Expression
- LOH at 16q22 (site of E-cad gene)
- Mutations
- Transcriptional silencing by epigenetic mechanisms (e.g. promoter methylation)

Adjunctive Immunostains To Discern LCIS (particularly variants of) from DCIS
- E-cadherin
- P120 catenin
- High molecular weight cytokeratin

DCIS
E-cadherin Positive

Low grade  High grade
LCIS: Loss of E-cadherin Expression

E-Cadherin Staining in LCIS

- Myoepithelial cells (weak, fragmented)
- Residual ductal epithelial cells
- LCIS cells (weak, fragmented)
Rat Mammary Lobule

E-Cadherin Staining in LCIS

- Myoepithelial cells (weak, fragmented)
- Residual ductal epithelial cells
- LCIS cells (weak, fragmented)
E-Cadherin Staining in LCIS

- Myoepithelial cells (weak, fragmented)
- Residual ductal epithelial cells
- LCIS cells (weak, fragmented)

Aberrant Expression of E-cadherin in Lobular Carcinomas of the Breast

- Aberrant E-cadherin “positivity” (incomplete membrane, cytoplasmic or Golgi staining) seen in 4 of 25 ILC, despite presence of genetic alterations commonly seen in ILC (16q loss, 1q gain)
- Evidence to suggest that E-cadherin protein in these cases was dysfunctional
- Presence and pattern of E-cadherin expression may be related to molecular mechanism of E-cadherin inactivation

P120 Catenin

- Present at junction of cell membrane and cytoplasm
- Involved in linking E-cadherin to actin cytoskeleton
- Normal cells/ductal lesions:
  - membrane distribution
- Lobular lesions:
  - cytoplasmic distribution

Dabbs, AJSP, 2007
E-cadherin and P120 Catenin in DCIS and LCIS

<table>
<thead>
<tr>
<th></th>
<th>DCIS</th>
<th>LCIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>E-cadherin</td>
<td>positive</td>
<td>negative</td>
</tr>
<tr>
<td>P120 catenin</td>
<td>membranous</td>
<td>cytoplasmic</td>
</tr>
</tbody>
</table>

Adjunctive Immunostains To Discern LCIS (particularly variants of) from DCIS

- E-cadherin
- P120 catenin
- High molecular weight cytokeratin
Heterogeneity of LCIS

- Like DCIS, the term “LCIS” encompasses a heterogeneous group of lesions that differ in morphology, immunophenotype, genetic alterations and, possibly, clinical behavior.
- Heterogeneity of LCIS has until recently been largely under-appreciated.

VARIANTS OF LCIS

- Common feature – E cadherin negative
- Clinico-pathologic characteristics
- Biomarker expression by IHC
- Molecular signature – high res aCGH

FLÓRID LCIS
Differences Between CLCIS and FLCIS/PLCIS

<table>
<thead>
<tr>
<th>Feature</th>
<th>Classical LCIS</th>
<th>FLCIS/PLCIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Younger (premenopausal)</td>
<td>Older (postmenopausal)</td>
</tr>
<tr>
<td>Presentation</td>
<td>Incidental</td>
<td>Mammographic</td>
</tr>
<tr>
<td>ER</td>
<td>Pos</td>
<td>Pos</td>
</tr>
<tr>
<td>HER2</td>
<td>Neg</td>
<td>Neg or Pos</td>
</tr>
<tr>
<td>Ki67</td>
<td>Low</td>
<td>High</td>
</tr>
<tr>
<td>Genomic changes (aCGH)</td>
<td>Fewer</td>
<td>More numerous</td>
</tr>
</tbody>
</table>

Histologic Distinction Between LCIS Variants and DCIS

<table>
<thead>
<tr>
<th>Feature</th>
<th>LCIS Variants</th>
<th>DCIS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loss of cohesion</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>Intracytoplasmic vacuoles</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Pagetoid ductal involvement</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Associated classical LCIS</td>
<td>More common</td>
<td>Less common</td>
</tr>
<tr>
<td>Microacini</td>
<td>Absent</td>
<td>Present</td>
</tr>
<tr>
<td>Polarization of cells at periphery</td>
<td>Absent</td>
<td>Present</td>
</tr>
</tbody>
</table>
USCAP 2002

21 FLORID LCIS (FLCIS); 5 LOW-GRADE SOLID DCIS
- AGE 42-81 (MEAN 61.5)
- 17 LUMINAL NECROSIS
- 15 CALCIFICATIONS
- 12/21 CONCURRENT INVASIVE CARCINOMA
  - 10/12 LOBULAR TYPE
- 21/21 E-CADHERIN NEGATIVE
- 15/16 STUDIED SHOWED LOH OF MS MARKER NEAR E-CADHERIN GENE

LCIS VARIANTS (FLCIS, PLCIS)
CLINICAL IMPORTANCE

Problem for Pathologist
Distinction from DCIS

Problem for Clinician
Manage like classical LCIS or like DCIS?

FLCIS / PLCIS

- E-cadherin neg
- Concurrent inv ca
- Comedo necrosis
- "Bad" biomarker profile

- +/- Nuclear pleomorphism
- Direct ductal extension

- LCIS variant
- More like DCIS than LCIS
- Treat like LCIS

- Treat like DCIS
What Does “Treat Like DCIS” Really Mean?

- Excision to negative margins?
- Radiation therapy?
- In the absence of data, is it better to over-treat or under-treat?

Florid and Pleomorphic LCIS
Clinical Significance

- No clinical follow-up studies akin to those available for classical LCIS and DCIS
- Natural history/biologic behavior unknown
- Appropriate management uncertain

Lobular breast carcinoma and its variants

However, due to their aggressive behavior and frequent association with invasive tumors mainly of high-grade lesions, there is at present an international consensus to regard Pf LCIS as an established form of in situ carcinoma equivalent to high-grade DCIS and should be managed in similar fashion to DCIS. Therefore, distinguishing Pf LCIS from DCIS becomes
26 pts with PLCIS at or close to surgical margin
- 16 received chemoprevention and/or radiation
- 10 received no adjuvant treatment
- 1/26 (3.8%) had recurrent PLCIS 18 mo later (F/U range 1-104 mo; mean 46 mo)
- At margin with no truncation

Columnar cell lesions
(in the context of LCIS)

Lobular carcinoma

Low-grade DCIS

High-grade DCIS

Invasive carcinoma
- Well-differentiated +1q, -16q
- Poorly-differentiated +17q
NOMENCLATURE

- COLUMNAR CELL CHANGE
- COLUMNAR CELL CHANGE WITH ATYPIA (FLAT EPITHELIAL ATYPIA)
- COLUMNAR CELL HYPERPLASIA
- COLUMNAR CELL HYPERPLASIA WITH ATYPIA (FLAT EPITHELIAL ATYPIA)
- ATYPICAL COLUMNAR CELL HYPERPLASIA = ATYPICAL DUCT HYPERPLASIA

INVASIVE CARCINOMA (IN THE CONTEXT OF LCIS)

Histologic Type of Cancer Following CLCIS
Most (up to 75%) are invasive ductal carcinomas

Frequency of Invasive Lobular Carcinoma Following CLCIS

<table>
<thead>
<tr>
<th>Study</th>
<th>% Invasive Lobular Ca</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chuba (SEER)</td>
<td>23.1%</td>
</tr>
<tr>
<td>Wheeler</td>
<td>25.0%</td>
</tr>
<tr>
<td>Haagensen</td>
<td>25.5%</td>
</tr>
<tr>
<td>Rosen</td>
<td>36.0%</td>
</tr>
<tr>
<td>Ottesen</td>
<td>37.0%</td>
</tr>
<tr>
<td>Page</td>
<td>70.0%</td>
</tr>
</tbody>
</table>
Tumor Recurrence (TR) in cases of CLCIS treated by Local Excision

- 180 pts exbx only for LCIS
- 12 year follow-up
- 26 (14.4%) ipsilat TR; 14 (7.8%) contralat TR
- Cases with slides for review: 8/9 (89%) ipsilat TR and 6/8 (75%) contralat TR – IFLC type
- 96% ipsilat and 100% contralat TRs occurred within the same site as the index LCIS


CLCIS AND MICRO-INVASIVE LOBULAR CARCINOMA, CLASSICAL TYPE

ROSS DS AND HODA SA
AM J SURG PATHOL 2011; 35(5):750-756

- 16/75,250 (0.02%) BREAST CASES
- AVG AGE 52 YRS
- PRESENTATION
  - RADIOGRAPHIC ABNORMALITY 13/16
  - PALPABLE 3/16
  - U/L R=L
- ALL WITH ASSOCIATED CLCIS=11, FLCIS=4, PLCIS=1
- NEG SLN 16/16
- SYNCHRONOUS IFDC =2, DCIS =2
CLCIS ON NEEDLE CORE BIOPSY

- CLASSICAL LCIS IS RARELY THE TARGET OF SCREEN DETECTED NEEDLE CORE BIOPSY
- RARELY ASSOCIATED WITH CALCIFICATIONS
- NOT A MASS FORMING LESION
- NO SPECIFIC MRI FEATURES
- AS A RESULT, MOST CLCIS FOUND IN THIS TYPE OF SURGICAL SPECIMEN IS INCIDENTAL IN NATURE

LCIS/ALH in NCB

Literature Review of 27 Studies
Cangiarella, 2008

- Worse lesion found on excision in 112 of 700 cases (16%)
  - 61/307 (20%) with LCIS (range, 0-50%)
  - 51/393 (13%) with ALH (range, 0-67%)
- Caveats
  - Studies differ with regard to details of needle biopsy procedure, extent of lesion removal
  - Not all patients with LCIS/ALH underwent excision
  - Some cases were associated with mass lesions or consisted of non-classical forms of LCIS

OUTCOMES OF PROSPECTIVE EXCISION FOR CLASSIC LCIS AND ALH ON PERCUTANEOUS BREAST CORE BIOPSY (ABSTRACT) LUEDTKE C, ET AL. MSKCC, NY

- PROSPECTIVE STUDY 2004-2009
- 69 PTS / 71 NCBS
- AGE RANGE 31-73 YEARS
- CLINICAL PRESENTATION
  - CALCIFICATIONS 47/71 (66%)
  - MASS 7/71 (10%)
  - MRI ENHANCEMENT 17/71 (24%)
- RESULTS: 2/71 (3%) PATHOLOGIC UPGRADE ON EXBX
  - ONE HAD 2.3 MM TUBULAR CARCINOMA AND 2 MM DCIS; OTHER HAD 2 MM DCIS
MANAGEMENT OF LCIS ON NCB

- Excision probably not necessary if CLCIS/ALH is completely incidental finding and there is radiologic-pathologic concordance.
- Excision recommended if:
  - another lesion present which by itself would be an indication for excision (e.g., ADH)
  - histologic features raise question of LCIS vs DCIS or non-classical LCIS (e.g., PLCIS)
  - radiologic-pathologic discordance
- Excision recommended if FLCIS or PLCIS

THANK YOU!