UTILITY OF URINE CYTOLOGY IN CONJUNCTION WITH ImmunoCyt TESTING FOR UROTHELIAL CARCINOMA

Los Angeles Society of Pathologists  
November 11, 2008

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PATIENT

- 69-year-old male
- Past medical history
  - Coronary artery disease, hypertension
- Hematuria
  - Persistent microscopic (1 year)
  - Acute gross hematuria
- Urine culture positive for Enterococcus spp.
- Cystoscopy
  - Bladder wall lesion, tumor vs. inflammatory
  - Urethral stricture
- Transurethral resection, bladder and prostate
**Immunostains:**
+ CK 7
+ CK 20
- PAP, S100, CEA

**DIAGNOSIS**

- Urothelial carcinoma in-situ
  - Pagetoid spread
  - Involving bladder and prostatic urethra
  - No invasive carcinoma
TWO MONTHS LATER …

- Cystoscopy
  - Frondular lesion, bladder neck
- Cytology
  - Bladder irrigation
  - ImmunoCyt
    - Cellular antigens specific for UC using fluorescent monoclonal antibodies
  - UroVysion FISH
    - Chromosomal abnormalities in UC
- TURBT
**Immunostains:**
- + CK7
- + Pankeratin
- + EMA
- - CD31

**DIAGNOSIS**

- Microscopic foci of invasive urothelial carcinoma
  - Invading superficial lamina propria
  - Microcystic variant

  **Microcystic variant:**
  - Variable size cysts
    - Lining → absent, flat, urothelial, mucinous differentiation
    - Pink secretions, necrotic material

  **Differential diagnosis:**
  - Urothelial carcinoma with glandular differentiation
    (apical cytoplasm, basal nuclei)
  - Adenocarcinoma (goblet cells)
  - Vascular neoplasm (CD31-positive)
  - Nephrogenic adenoma (no atypia)

  No prognostic significance
DIAGNOSIS

- Atypical cells

- ImmunoCyt
  - Positive
  - 8 green
  - > 20 red

- UroVysion FISH
  - Normal signal pattern
  - Single cell with abnormal pattern
    (need ≥ 4 cells per FDA criteria)
**FOLLOW-UP CYTOLOGY**

**Diagnosis:**
Urothelial carcinoma
High grade/CIS

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**ImmunoCyt**
(Diagnostix)
ASSAY

- Qualitative direct immunofluorescent assay
- Patented monoclonal antibody cocktail
  - M344 and LDQ10 react with mucin glycoprotein in cytoplasmic vacuoles (*green*)
  - 19A211 specific to glycosylated form of CEA (*red*)
    - *Expressed in cancer cells, not normal urothelium*
- FDA-approved for monitoring bladder carcinoma in combination with urine cytology and cystoscopy
COMPARISON OF CYTOLOGY, ImmunoCyt & UroVysion

- 100 specimens (11/2006 → 03/2007)
  - Patients undergoing monitoring for UC recurrence
  - Low grade and high grade cases
  - Multiple sites in Canada
- Voided urine samples collected
  - Immediately before cystoscopy
  - 41% positive cystoscopy
- Cytology, ImmunoCyt and UroVysion analysis
  - Performed blindly at three different labs
  - Cystoscopy with histology as gold standard

Unpublished data, UCLA Department of Pathology, 2008.
**ImmunoCyt vs. UroVysion**

- Cytology + ImmunoCyt
  - Significantly increases sensitivity of cytology alone (73% vs. 20%)
- ImmunoCyt more sensitive than UroVysion
  - 75% vs. 10%
- UroVysion more specific than ImmunoCyt
  - 92% vs. 60%

*Unpublished data, UCLA Department of Pathology, 2008.*

**CONCLUSIONS**

- Urine cytology is important for diagnosing and monitoring urothelial carcinoma
- Carcinoma may present with positive cytology, but denuded urothelium on biopsy
  - Critical to examine the denuded areas to r/o CIS or micro-invasive component
- ImmunoCyt may provide additional diagnostic value
  - Appears to be more sensitive (but less specific) than UroVysion in detecting early lesions
THANK YOU

Dr. Jian Yu Rao
Clinical history

- 10 month old female with right groin abscess, thrombocytopenia, anemia and jaundice
- 6 weeks prior to admission, the patient had a rash and right groin mass which was lanced
- 5 weeks later, the rash improved but the patient spiked fevers and was diagnosed with otitis media
- The patient became jaundiced and had swelling of the right leg and was sent to UCLA for admission
- On exam, a seborrheic rash was noted
Laboratory studies

- CBC: Hgb 5.7, Hct 19.7%, MCV 88.8, WBC 7.46, Platelets 60,000, Reticulocyte count 10.24%
- LFTs: Albumin 2.0, Alk Phos 968, ALT 26, AST 20, Total Bilirubin 9.1, Direct Bilirubin 5.1, LDH 157
- PT 10.6, PTT 33.8, INR 1.1

Radiologic studies

- Ultrasound – Hepatosplenomegaly, no evidence of bile duct obstruction
- MRCP – Hepatosplenomegaly with no evidence of liver lesions. Left intrahepatic biliary dilatation with marked irregularity of the left intrahepatic bile ducts.
Liver – Preliminary diagnosis

- Changes consistent with bile duct obstruction
- Causes:
  - Stones
  - Neoplasm
    - Bile ducts
    - Pancreas
  - Choledochal cyst
  - Trauma – Surgery
  - Inflammation
  - Autoimmune
  - Ischemia
  - Infection
  - Enlarged lymph nodes
**Langerhans Cell Histiocytosis**

- Abnormal proliferation of Langerhans cells
- Affects all age groups
- Incidence: 4 per million, predominantly males
- Unifocal - Usually involves the bone
- Hans-Schuller-Christian disease: Diabetes insipidus, Exophthalmos, Bony defects
  - May involve lungs and/or mucocutaneous sites
- Disseminated: Fever, anemia, thrombocytopenia, and involvement of skin, lung, liver, spleen and other organs
  - Skin shows petechiae and crusted papules involving scalp, face, trunk, buttocks, intertriginous areas

**LCH in the liver**

- Clinical findings
  - Hepatosplomegaly
  - Jaundice
  - Ascites
  - Abnormal liver enzyme levels
- Gross pathology
  - May form nodules
  - Biliary fibrosis
  - Large duct involvement
    - Dilation and rupture
**LCH in the liver - histology**

- Most cases show infiltration and injury of bile ducts
- Rupture of large ducts may invoke xanthogranulomatous response
- Periductal fibrosis
- Secondary sclerosing cholangitis

**LCH in the liver - histology**

- Langerhans cells
  - Abundant cytoplasm
  - Kidney bean shaped nuclei
  - Fine chromatin without nucleoli
  - CD1a+, S100+, CD68-, CD163-, CD31-
  - EM – Birbeck granules
- Accompanying inflammation
  - Eosinophils, lymphocytes, neutrophils, plasma cells, histiocytes, giant cells
LCH in the liver – secondary changes

- Ductular reaction
- Ductopenia
- Peri-portal cholestasis
- Copper deposition
Other histiocytic proliferative lesions of the liver

- **Xanthoma disseminatum**
  - Disseminated juvenile xanthogranulomas
  - S100-, CD1a-, CD68+, Factor 13a+, fascin+

- **Rosai Dorfman disease**
  - Involves portal areas and sinusoids
  - Emperipolesis
  - S100+, CD1a-, CD68+

- **Erdheim Chester disease**
  - Rare case reports
  - S100-, CD1a-, CD68+
Bibliography

Steven Ohsie MD

- 2003-2007 - AP/CP residency
- AP/CP Board Certified - 2007
- 2007-2008 - Klaus Lewin GI/Liver Pathology Fellowship
- 2008-2009 – Dermatopathology Fellowship
- July 2009 - ?????
Case #4
Seong Ra
UCLA Medical Center

CLINICAL HISTORY

- A 76-year-old male presented with an incidentally detected asymptomatic 3 cm mass in the region of the uncinate process of his pancreas.
- A fine needle aspiration was performed at an outside institution which was diagnosed as a pancreatic endocrine neoplasm.
CLINICAL

- After a surgical consultation, a pancreaticoduodenectomy procedure was recommended and performed.

GROSS

- At frozen section a 3.0 x 2.9 x 2.7 cm well circumscribed white to brown ovoid mass was seen.
- Pancreatic, bile duct, and small bowel margins were negative.
DIFFERENTIAL DIAGNOSIS

- Adenocarcinoma with neuroendocrine differentiation.
- Mixed ductal-endocrine carcinoma.
- Pancreatic endocrine neoplasm with entrapped ductules.

ADENOCARCINOMA WITH NEUROENDOCRINE DIFFERENTIATION

- Ductal adenocarcinoma is the predominant component with focal neuroendocrine cells, typically seen by immunohistochemistry.
- Prognosis is the same for typical ductal adenocarcinoma.
MIXED DUCTAL-ENDOCRINE CARCINOMA

- Malignant epithelial neoplasm with separate, morphologically recognizable ductal adenocarcinoma and endocrine elements, each constituting at least 25% of the neoplasm.
  - In most of these cases, the endocrine elements have the appearance of a poorly differentiated endocrine carcinoma rather than a well-differentiated PEN.
  - The ductal adenocarcinoma component is typically moderately to poorly differentiated.

PANCREATIC ENDOCRINE NEOPLASM WITH ENTRAPPED DUCTULES

- PEN with a proliferation of ductules present within multiple foci within the tumor displaying tight intermingling and merging of the tumor-associated ductules with endocrine elements.
- The endocrine and ductal components are usually well differentiated.
- Behaves similar to a typical nonfunctional PEN.
  - 5 year survival: 65%.
  - 10 year survival: 45%
FINAL DIAGNOSIS

- Well differentiated pancreatic endocrine neoplasm with entrapped ductules.
- Size: 3.0 cm
- Four of fifteen lymph nodes are positive for metastases (4/15)
- Lymphovascular and perineural invasion is identified
- Surgical resection margins are negative for carcinoma

COMMENT

- This case was sent for the opinion of Dr. Ralph Hruban.
- He also interpreted the ductal component to be benign and concurred with our diagnosis.
PANCREATIC ENDOCRINE NEOPLASM WITH ENTRAPPED DUCTULES

- There are scattered reports of PENs and they are rare.
- This neoplasm has been defined as:
  - Proliferation of small-caliber ductules present within multiple foci within the tumor, including the central portion of the tumor.
  - Tight intermingling and merging of the tumor-associated ductules with endocrine elements.

Patients ranged in age from 9-80 years (mean 54 and 59 years) similar to typical PENs.

- A slight majority was seen in females which is similar to typical PENs.
- Insulinomas: 8 of 31 cases
- Glucagonomas: 2 of 31 cases
- Remaining cases nonfunctional or clinical information unavailable.
Follow up information (Deshpande et al.)
- Alive and free of disease: 10 of 12 patients with a median follow up of 70 months.
- Died of disease: 2 patients at 81 and 158 months.

Controversy exists concerning the origin of the ductules:
- Deshpande et al. (15 cases) believed that the admixed elements arose from a multipotent stem cell.
- van Eeden et al. (16 cases) believes that the ducts are entrapped within the endocrine neoplasm.
van Eeden et al.

- van Eeden et al showed that molecular genetic changes often present in ductal pancreatic neoplasms were not found in these lesions by immunohistochemistry for *DPC4*, *p53*, and *ERBB2* and by sequence analysis of *KRAS* codon 12.

- van Eeden et al. performed an X-chromosome inactivation clonality assay of one such tumor from a female patient.
  - This revealed that the endocrine component was monoclonal, contrasting with the ductular component that was polyclonal.
van Eeden et al.

- The lymph node and liver metastases from 3 patients only contained the neuroendocrine component, and no ductules were observed.

van Eeden et al.

- Certain morphologic features of ductule containing endocrine tumors are reminiscent of the embryonic development of the human pancreas.
- But none of the tumors expressed PDX-1, a transcription factor essential in pancreatic organ development.
CONCLUSION

- Van Eeden et al. suggest that based on their evidence that:
  - The ductular component in these lesions are the result of entrapment of preexisting non-neoplastic ductules.
  - The neoplasms are otherwise not distinctive from conventional PENs.

CONCLUSION

- Although these neoplasms are rare, it is important to recognize and to distinguish them from a true mixed ductal-endocrine neoplasm, which are biologically similar to ductal adenocarcinoma and carry a grim prognosis.
References


THANK YOU FOR YOUR SUPPORT

- Dr. Galen Cortina
- Dr. Sarah Dry
- Dr. David Cassarino
- Dr. Charles Lassman
Case Presentation of a Soft Tissue Neoplasm

Sachiv Sheth, M.D.
UCLA Department of Pathology
11/11/2008

Case

- 2 year old female
- Slowly growing buttock mass
- Imaging
  - 10 cm well-circumscribed pre-sacral mass
  - Arising from pelvic floor
  - Mass effect on bladder and rectum
Case - MRI

T1

T2 fat suppressed

Radiology DDx: Sacrococcygeal teratoma, fatty lesion

Histology
Diagnosis:
Lipoblastoma
Lipoblastoma

- Jaffe 1926 – “lipoblastoma”
- Chung and Enzinger 1973
  - “Benign tumor of fetal or embryonal adipose tissue in infants and children”
  - Lipoblastoma: well-circumscribed
  - Lipoblastomatosis: infiltrative
- 250 reported cases (October 2008)

Lipoblastoma

- Most patients under 3; oldest 48 years old
- Since 2000, increase in reports in older children/adults

![Pie chart showing age distribution of lipoblastoma cases](chart.png)
**Lipoblastoma**

- Classic: subcutaneous, extremity
- 2/3 circumscribed, 1/3 infiltrative

**Behavior**

- In the largest seven reported series, 16% local recurrence (22/137)
  - Easily controlled with surgery
- NO distant metastases
- Local recurrence more common in deep seated tumors or those with positive margins
Histology
Zonation

Mixture of adipocytic stages
Myxoid areas

- May show plexiform capillaries
- Typically focal
- Rarely extensive (>50%)
Lipoblastoma - molecular

- 1993: Chromosome 8 rearrangements in pediatric adipose tumors (J. Fletcher)
- 2000: Involvement of the PLAG1 gene in lipoblastoma
  - Rearrangements of 8q11-13 (80%)
    - Leads to promoter swapping
    - Results in gain of activation of PLAG1
  - Gains of chromosome 8 (10%)
- Molecular alterations seen in all adipocytic stages

Lipoblastoma - molecular

- Cytogenetics/FISH
- 8q alterations rare in other adipocytic tumors
  - Lipomas, hibernoma, ALT
  - Not reported in myxoid LPS

FISH image courtesy of N. de Saint Aubain Somerhausen
Lipoblastoma - differential

- Predominant myxoid component
  - Myxoid liposarcoma
- Predominant spindle cell component
  - Infantile fibromatosis
- Predominant mature adipocytic component
  - Lipoma
  - Atypical lipomatous tumor/well-differentiated liposarcoma
- Predominant brown fat component
  - Hibernoma

Myxoid liposarcoma

- Rare <10 y.o.
- Peak 25-45 y.o
- Usually deep
- Plexiform vessels
- Myxoid matrix
- Signet-ring lipoblasts
- Mucin pooling
- Nuclear atypia
- $t(12;16)$ (FUS-CHOP)
- $t(12;22)$ (EWS-CHOP)
Lipofibromatosis (aka infantile fibromatosis)

- Birth – 8 y.o. (<2 y.o.)
- Deep seated
  - H&N, extremities
- Spindled or ovoid fibroblasts within adipocytic component
- Fascicles, bundles
- Cellularity may be high
- Mitoses can be seen
- NO lobulation, zonation, or primitive adipocytes

Lipoma

- Rare in children
- Peak 40-60 y.o.
- Uniform mature adipocytes
- No prominent fibrous septae
- HMGIC rearrangements at 12q13-15
Atypical Lipomatous Tumor/Well-differentiated Liposarcoma

- Peak 50-60 y.o.
- Occasional fibrous bands
- Markedly enlarged, hyperchromatic cells seen in adipocytic and fibrous areas
- Lipoblasts rare
- No zonation
- 12q13-15 amplification of MDM2 or CDK4 genes

Hibernoma

- Peak 20-30 y.o.
- Eosinophilic granular cells (EGC) +/- multivacuolated lipid droplets
- Occasionally myxoid
- No fibrous septae
- No zonation
- Distinct mutation of 10q or 11q
Adipocytic Tumors - Molecular

- Lipoblastoma
  - 8q11-13 rearrangements or polysomy
- Myxoid liposarcoma
  - t(12;16) or t(12;22)
- Lipoma
  - HMGIC rearrangements at 12q
- Atypical lipomatous tumors/Well-differentiated liposarcoma
  - Giant marker and ring chromosomes with 12q amplification
- Hibernoma
  - 10q or 11q alterations

Lipoblastoma - summary

- Benign adipocytic tumor usually in children
  - Lobulated with fibrous septae
  - Zonation
  - Adipocytes of varying stages
  - Atypical cells NOT seen
  - Rearrangements involving the PLAG1 gene on 8q or gains of chromosome 8
- Unusual features
  - Adolescents and adults
  - Single predominant component (myxoid, primitive mesenchymal cells, mature fat, brown fat)
    - Molecular studies (cytogenetics, FISH) very useful
References

- Special thanks to Dr. Sarah Dry, M.D.

62 year old female with polypoid uterine mass

November 11, 2008
Peggy S. Sullivan, MD
UCLA Geffen School of Medicine

Clinical History: 62 year old female

• MRI 2006:
  – Enlarging non-enhancing complex endometrial mass, 3.4 cm

• 2008: Post-menopausal bleeding, no HRT
  – EMB: complex hyperplasia with atypia

• TAH+BSO with staging
Gross description

- UTERUS, 84 grams with attached adnexa:
- Distorted by leiomyomata up to 1.6 cm
- 4.5 cm smooth gray-tan mass, endometrial cavity
- Cut surface: firm tan-white solid, cystic (up to 1 cm) filled with clear viscous fluid
- Remaining specimen unremarkable
Differential diagnosis

• Poorly-Differentiated Endometrioid Carcinoma

• Carcinosarcoma (Malignant Mixed Mullerian Tumor)

• Arising in an Adenosarcoma?

Why is this distinction important?

• Management issue: Carcinosarcoma distinct entity and should not be included in high-grade EMC trials
  – Amant et al 2005 – G3 EMC, UPSC, CCC, CS (137): CS worse outcome & higher incidence of pulmonary metastases
  – Bansal et al 2008 – SEER data (~9000); mortality 45% less in G3 EMC

• MMMT arising in polyp somewhat better outcome:
  – Barwick et al 1979, Kahner et al 1975 – disease-free 1-11 yr f/u, few cases
Carcinosarcoma (MMMT)

- Admixture of malignant epithelial and mesenchymal components
- Epithelial: glandular (endometrioid or non-endometrioid), rarely squamous or undifferentiated; usually high grade but not always
- Sarcomatous: homologous (undifferentiated, smooth muscle, endometrial stromal) or heterologous (cartilage, skeletal muscle)
- Some cases: minor component following extensive sampling
- Most cases: sharply demarcated; some appear to merge with transitional forms
- Immunohistochemistry: (+) keratins
  - Mesenchymal component: (+) vimentin and focal keratin, p53 concordance
  - Heterologous component: (+) S100, myoglobin

WHO 2003

Carcinosarcoma histogenensis

- Metaplastic carcinomas rather than true mixed tumors (WHO 2003)
  - Clinical, IHC, ultrastructural, molecular studies
  - Pattern spread via lymphatics; similar to high grade endometrial carcinoma rather than sarcoma
  - Metastases are carcinomatous
  - Heterologous vs homologous: no difference
    - Except in stage I: Ferguson et al 2007
Why should we consider endometrioid carcinoma?

Review Article

Endometrioid Carcinoma of the Uterine Corpus: A Review of Its Pathology With Emphasis on Recent Advances and Problematic Aspects

*Philip B. Clement and †Robert H. Young

Advances in Anatomic Pathology 2002; 9(3):145–184

ENDOMETRIOID CARCINOMAS WITH SPINDLED EPITHELIAL CELLS

The spindle cells are almost always less atypical (Fig. 12) than the sarcomatous spindle cells of a malignant müllerian mixed tumor (MMMT), and a biphasic pattern (so typical of the latter tumor), although seen, is generally not as striking.

Though the MMMT is the best known biphasic tumor, it is worth noting that pure endometrioid carcinomas of various subtypes (Table 4) may descriptively be biphasic, a feature that can result in a misdiagnosis of MMMT.
Endometrioid Carcinomas of the Uterine Corpus
With Sex Cord-like Formations, Hyalinization,
and Other Unusual Morphologic Features

A Report of 31 Cases of a Neoplasm That May Be Confused
With Carcinosarcoma and Other Uterine Neoplasms

Shawn K. Murray, MD,* Philip B. Clement, MD,† and Robert H. Young, MD‡

“Corded and hyalinized endometrioid carcinoma”

**Abstract:** We describe a series of unusual endometrioid carcinomas (ECs) of the uterine corpus characterized in significant part by cords of epithelioid cells, spindle cells, and a hyalinized stroma that sometimes formed osteoid. These features, particularly when prominent, produced an appearance strikingly different from that of conventional EC, sometimes resulting in problems in differential diagnosis, especially with a malignant mullerian mixed tumor (carcinosarcoma).

• “resulted in a diagnosis of MMMT being favored or considered in 70% of the cases”
• “... less atypical and less mitotic than the sarcomatous spindle cells of MMMTs.
• almost imperceptible merging with the obvious glandular or squamous carcinomatous cells.”
• Low p53 expression
“Corded and hyalinized endometrioid carcinoma”

• “…the age of the patients, the low stage…, and the generally favorable prognosis of the tumors support that they are a variant of EC rather than MMMT…”

• 52 yo, most stage II, FIGO grade 2 or 1, 83% NED f/u 34 months

Is this Endometrioid Carcinoma?

A final argument for Carcinosarcoma:

Evaluation of the Relationship Between Adenosarcoma and Carcinosarcoma and a Hypothesis of the Histogenesis of Uterine Sarcomas

Jeffrey D. Seidman, M.D., and Suman Chauhan, M.D.

### TABLE 1. Clinical and pathologic features of uterine carcinosarcomas

<table>
<thead>
<tr>
<th>Group</th>
<th>n</th>
<th>Mean age (yr)</th>
<th>Mean size (cm)</th>
<th>FIGO stage</th>
<th>Myometrial invasion</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adenosarcoma-like component present</td>
<td>4</td>
<td>64</td>
<td>3.8</td>
<td>I-1</td>
<td>0</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>III-3</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>III-2</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>U-1</td>
<td>3</td>
</tr>
<tr>
<td>Leaf-like processes lined by malignant epithelium</td>
<td>4</td>
<td>67</td>
<td>4.8</td>
<td>II-1</td>
<td>1</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>III-2</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>U-1</td>
<td>3</td>
</tr>
<tr>
<td>Ordinary carcinosarcoma</td>
<td>18</td>
<td>64</td>
<td>4.6</td>
<td>I-5</td>
<td>8</td>
</tr>
<tr>
<td></td>
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<td>II-2</td>
<td>8</td>
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<td></td>
<td></td>
<td></td>
<td>III-3</td>
<td>7</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td>IV-4</td>
<td>7</td>
</tr>
</tbody>
</table>

Seidman and Chauhan 2003

### TABLE 3. Reported carcinosarcomas with a low-grade stromal component

<table>
<thead>
<tr>
<th>Series</th>
<th>n</th>
<th>No. with low-grade stroma</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Gagne (29)</td>
<td>58</td>
<td>9</td>
<td>16%</td>
</tr>
<tr>
<td>Bitterman (4)</td>
<td>22</td>
<td>6</td>
<td>27%</td>
</tr>
<tr>
<td>Silverberg (3)</td>
<td>175</td>
<td>29</td>
<td>17%</td>
</tr>
<tr>
<td>Major (28)</td>
<td>267</td>
<td>41</td>
<td>15%</td>
</tr>
<tr>
<td>Costa (10)</td>
<td>56</td>
<td>6</td>
<td>11%</td>
</tr>
<tr>
<td>Totals</td>
<td>578</td>
<td>91</td>
<td>16% weighted</td>
</tr>
</tbody>
</table>

Seidman and Chauhan 2003
Proposed histogenetic schema for Carcinosarcoma (CS)

- 85-90% metaplastic carcinomas
- 10-15% biphasic or collision-type neoplasm with independently arising carcinomatous and sarcomatous components
  - Reflected in cases of CS with an adenosarcomatous or low-grade stromal sarcomatous component
  - Other studies: 8% cases CS with biclonality established by molecular data

Seidman and Chauhan 2003

Final diagnosis??
• Carcinosarcoma arising in an adenosarcoma

• 0/34 LN, omentum, washings (-)

• Additional comments:
  • “Although some would interpret this tumor as entirely poorly differentiated endometrioid adenocarcinoma, we feel that the adjacent more low-grade adenosarcoma appearing areas and the pattern of undifferentiated tumor growing around benign endometrial glands are diagnostic for carcinosarcoma.”

Final Comments

• A case that highlights problematic issues in gynecologic neoplasms and raises more questions than answers

• Further studies:
  – Neoplasms with low grade sarcomatous, ‘collision’, and adenosarcomatous component
  – Molecular characterization and follow-up
  – Include endometrioid carcinomas with atypical features
Acknowledgements

- Tyler Youngkin
- Teri Longacre
- Richard Kempson
- Kirsten Woolf
- Amy Ly
- Ankur Sangoi