Loyola University Medical Center
Department of Pathology

IRAP Case Presentation Handouts

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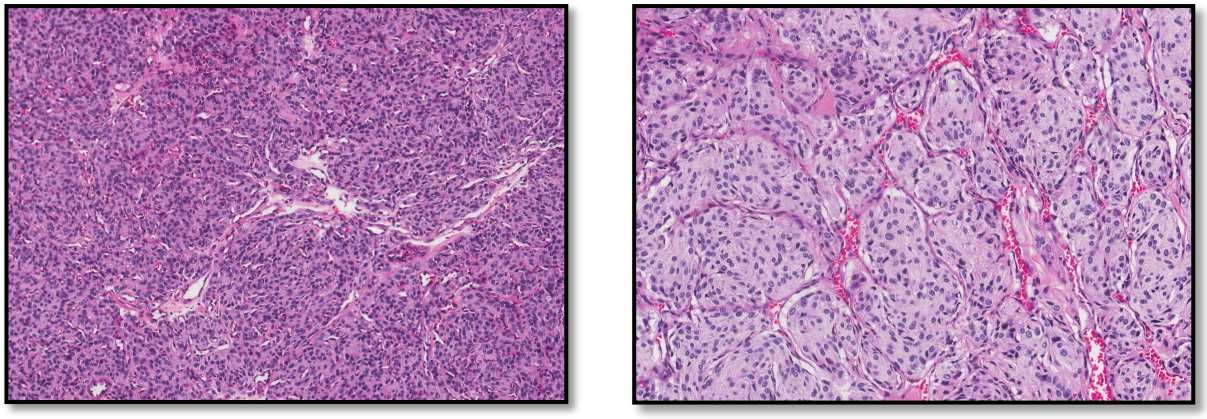
Case 1
Presenter: Michael Moravek MD, PGY II
Attendings: Stefan Pambuccian, MD and Ewa Borys, MD

Clinical History: A 53-year-old man presented with a pain in his thigh. One year previously, he had suffered a subtrochanteric femur fracture after slipping on ice. Workup for pathologic fracture, including a bone scan and blood work was negative at that time. He was treated with an intramedullary (IM) nail insertion, and the fracture healed uneventfully. On admission, imaging studies (plain X-rays and CT scan) showed an aggressive lesion in the proximal diaphysis of the femur surrounding the intramedullary nail and extending through the cortex into the adjacent soft tissues. A core needle biopsy followed by surgical resection of the proximal femur was performed.

Final Diagnosis: METASTATIC MENINGIOMA

Differential Diagnosis:
- Clear Cell Sarcoma
- Malignant Melanoma
- Metastatic Meningioma
- Myoepithelial carcinoma of soft tissue
- Paraganglioma
- Solitary Fibrous Tumor

Histology:



The tumor is composed of lobules and nests of fairly uniform cells, separated by fibrotic bands of varying thickness with focally prominent vascular spaces. The tumor cells exhibit abundant pale eosinophilic cytoplasm, indistinct cell borders, round to oval nuclei with mild pleomorphism and hyperchromasia, and inconspicuous nucleoli. Other areas show sheet-like (solid) growth of tumor cells with higher nucleocytoplasmic ratio and more hyperchromatic nuclei. Overall, mitotic activity is low.

Positive IHC:	**Negative IHC:**
Keratin AE1/AE3	EMA
S100	P63
Vimentin	Desmin, SMA
CD117	CK7, CK20
PR (focal)	CD31, CD34
	HMB45, MART1, MITF
	Synaptophysin, Chromogranin, CD56

Discussion:

Meningiomas are the most common primary intracranial neoplasms, constituting more than 30% of all CNS tumors with a prevalence of approximately 97.5 in 100,000 (1). Metastases from meningiomas are rare, reportedly occurring in 0.1-0.7% of cases, although these figures are likely overestimates reflecting publication bias and the inclusion of meningeal hemangiopericytoma/solitary fibrous tumors (previously referred to angioblastic meningioma), which are much more likely to metastasize. The incidence of meningioma metastases increases with increasing tumor grade from 0.1% in WHO grade I tumors to up to 5% in WHO grade II (atypical) meningiomas and up to 30% in WHO grade III (anaplastic) meningiomas (2). Metastases tend to occur late, after multiple local recurrences and multiple resections, and have been reported up to 30 years after the initial diagnosis (3). The most common sites of metastasis are lungs, followed by bones, liver, and lymph nodes(4). 50% of bone metastases of meningiomas are solitary and occur in patients without visceral metastases. Bones affected by metastatic meningiomas are more commonly part of the axial skeleton (spine, ribs, pelvis); long bones are rarely involved (5).

Metastases from meningiomas tend to reflect the histologic features of the primary tumor and therefore be composed of a variety of cell types, including spindle cells (fibroblastic), epithelioid (meningothelial), clear or rhabdoid cells, growing in nested, whorled, fascicular, papillary or solid arrangements. Due to their higher grade, unusual histologic types may be overrepresented in metastases, but most reported metastatic meningiomas have meningothelial nested histology.

The immunohistochemical staining pattern of metastatic meningiomas has not been studied in depth; however, it is likely to parallel that of intracranial meningiomas. In general diagnostic immunostains are rarely performed in intracranial meningiomas, both due to the fact that their diagnosis is usually straightforward and because they do not have a very specific immunostaining pattern. Nonetheless, meningiomas are usually positive for EMA (50-90%), PR (the percentage of staining cells is inversely proportional to the tumor grade), and may be weakly, focally positive for S100 and cytokeratin. Dual D2-40 and E-cadherin staining has been reported to be useful in the diagnosis of meningiomas (6), and a recent study showed that somatostatin receptor 2A (SSTR2A) was the single most sensitive and specific marker for the diagnosis of meningioma, with high sensitivity (95.2%) and specificity (92%) (7). However, as also seen in this case, the staining pattern of metastatic meningioma may be atypical and it is important to carefully review the patient's history and any available pathologic material, despite the rarity of metastases in meningiomas.

Although meningioma is an unlikely differential diagnosis for a bone tumor, metastatic meningioma should be considered when the histologic pattern is reminiscent of meningioma even in patients without a known prior diagnosis of meningioma to prevent a misdiagnosis as metastatic carcinoma(8).

Take home messages:

Meningioma metastases are rare and may involve the lungs, bones, and lymph nodes, in descending order. EMA and PR, the most commonly used diagnostic immunostains for meningiomas, can be negative in aggressive and metastatic meningiomas. Therefore, in cases with unusual morphology, the pathologist should review the patient's history and consider the possibility of metastatic meningioma in the appropriate clinical context. Finally, if it looks like meningioma on H&E it is probably meningioma and stains may only complicate the diagnosis.

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Case 2
Presenter: 'Tobi Odetola MD, PGY II
Attendings: Swati Mehrotra, MD

Clinical History: The patient is a 49-year-old woman who is transferred to our hospital for management of recurrent ascites and pleural effusion(s). Her past medical history is significant for latent tuberculosis (treated in 2012), a right ovarian mass (diagnosed on ultrasound in 2015) and hypothyroidism. CT imaging demonstrates moderate ascites with no adnexal masses, multiple hypodense liver lesions, and a right-sided pleural effusion associated with atelectasis. Work-up for metabolic etiologies and heavy metal toxicities is negative. A QuantiFERON-TB test was reportedly positive at the referring institution; otherwise an infectious work up is negative. A work up for TB includes pleural fluid cytology and lung wedge biopsy. A section from the wedge lung biopsy is submitted for your review.

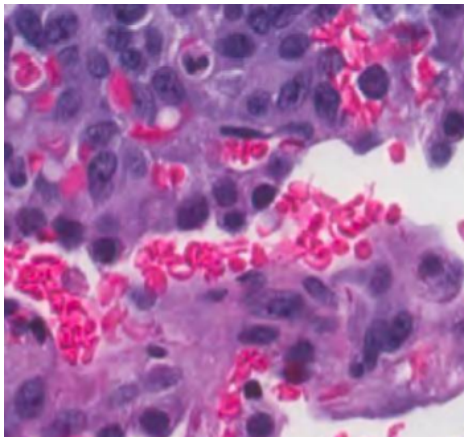
Final Diagnosis: ANGIOSARCOMA INVOLVING THE PLEURA

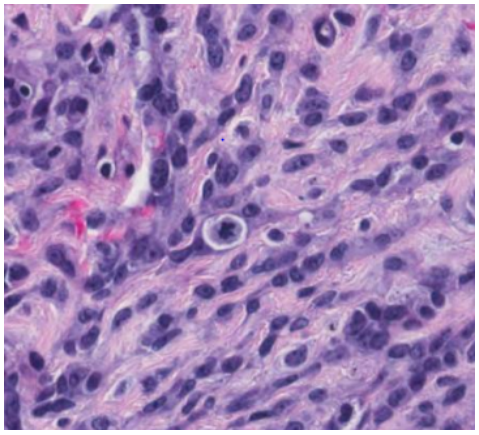
Differential Diagnosis:
- Benign mesothelial hyperplasia
- Mesothelioma
- "Pseudomesotheliomatous" adenocarcinoma
- Metastatic carcinoma (e.g. breast)
- "Pseudomesotheliomatous" epithelioid hemangioendothelioma
- "Pseudomesotheliomatous" epithelioid angiosarcoma

Key Features:
Histology:

Diffuse pleural thickening with bland epithelioid cells with moderate-to-abundant eosinophilic cytoplasm, centrally-placed nuclei with irregular borders, vesicular chromatin, binucleation and prominent nucleoli.

Two main growth patterns are seen: vasoformative (vascular lakes, poorly formed vascular spaces lined by tumor cells and RBC-containing intracytoplasmic lumen) and solid (strand-like and infiltrative) patterns. Abundant mitotic figures are also identified.





Immunohistochemistry:

Positive Stains	Negative Stains
CD31	CD34
FLI-1	WT-1
D2-40	Calretinin
	Pankeratin
vWF	Cytokeratin 5/7
	EMA
	TTF-1
	CD15
	CEA
	ER & PR
	TAG-72
	Special stains for AFB, CMV, GMS, Adenovirus, HSV

Discussion:

- Mesotheliomas are the main diagnostic consideration for an epithelioid pleural-based lesion; when mesothelial markers are negative, a malignant vascular tumor should be considered.
- Malignant pleural vascular tumors (either primary or metastatic) are very rare and have been associated with tuberculous pyothorax(1), radiation therapy(2) and asbestos exposure. Both pleural epithelioid hemangioendothelioma (EHE) and epithelioid angiosarcoma (EAS) have been described(3, 4). Such tumors may arise from the pleural vessels or from transdifferentiation ("metaplasia") of mesotheliomas(5).
- EHE has a characteristic stroma (myxoid, hyaline or chondroid) with variable atypia while the EAS often consists of vasoformative and solid areas frequently showing atypical cells.
- The tumor cells in EAS are usually positive for at least one vascular marker (CD31, CD34, FLI-1, vWF, D2-40, ERG). Negativity for epithelial markers is typical although 30% of cases may show positivity for at least one keratin (usually AE1/AE3 in EAS and CK 8/18 in EHE)(4).
- Although pleural effusions are usually present, pleural fluid cytology is rarely helpful in the diagnosis of EAS, because malignant cells rarely shed into the fluid. However, performing CD31 and CD34 immunostains may occasionally show rare malignant cells(6, 7).
- Cytogenetic studies for the WWRT-1-CAMTA-1 gene fusion can be helpful in differentiating intermediate grade EHE (positive) from EAS(4).
- Primary pleural EAS is very aggressive and rapidly fatal malignancy with about 70% of patients dying of disease within 7 months.

Take-Home Messages:

- The diagnosis of a pleural vascular malignancy is not easily reached and is usually preceded by the consideration of more common entities.
- Immunohistochemical studies are key to the diagnosis of these entities, particularly EAS. Molecular studies may aid the differentiation between EHE and EAS.

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Case 3
Presenter: Shaun Boyes MD, PGY II
Attendings: Dariusz Borys, MD and Swati Mehrotra, MD

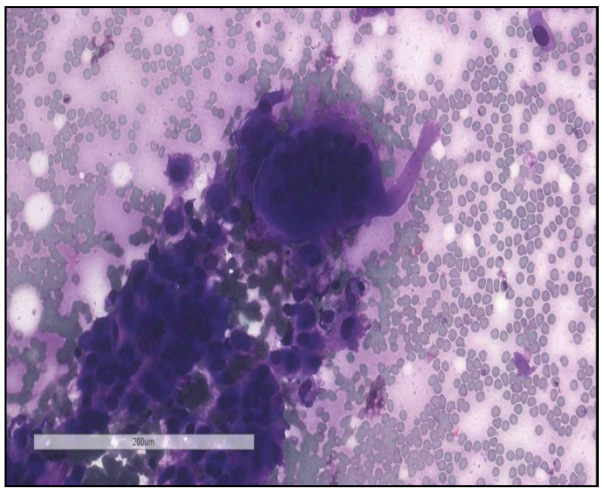
Clinical History: The patient is a 58-year-old man who presents with a right parotid mass for about 4-6 months. Physical examination reveals a firm and fixed, 4 cm mass in the pre-auricular region with no skin involvement. CT imaging of the neck demonstrates an "intensely enhancing lesion in the right parotid gland that is well circumscribed with no invasion of adjacent structures". Based on cytological interpretation of prior FNA of this mass at an outside institution, the patient undergoes an excisional biopsy. A section from this resection is submitted for your review.

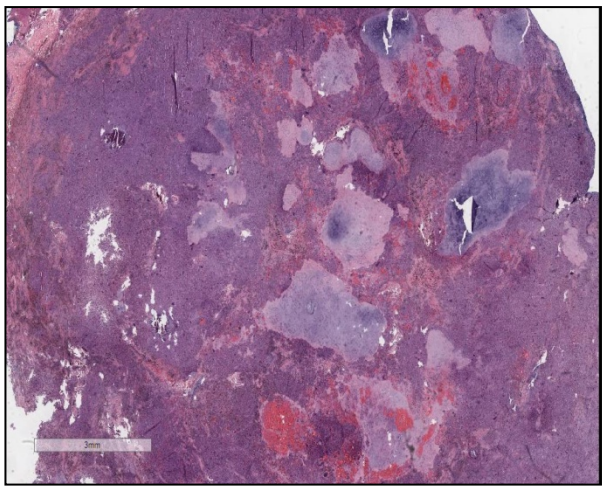
Final Diagnosis: TENOSYNOVIAL GIANT CELL TUMOR WITH CHONDROID METAPLASIA

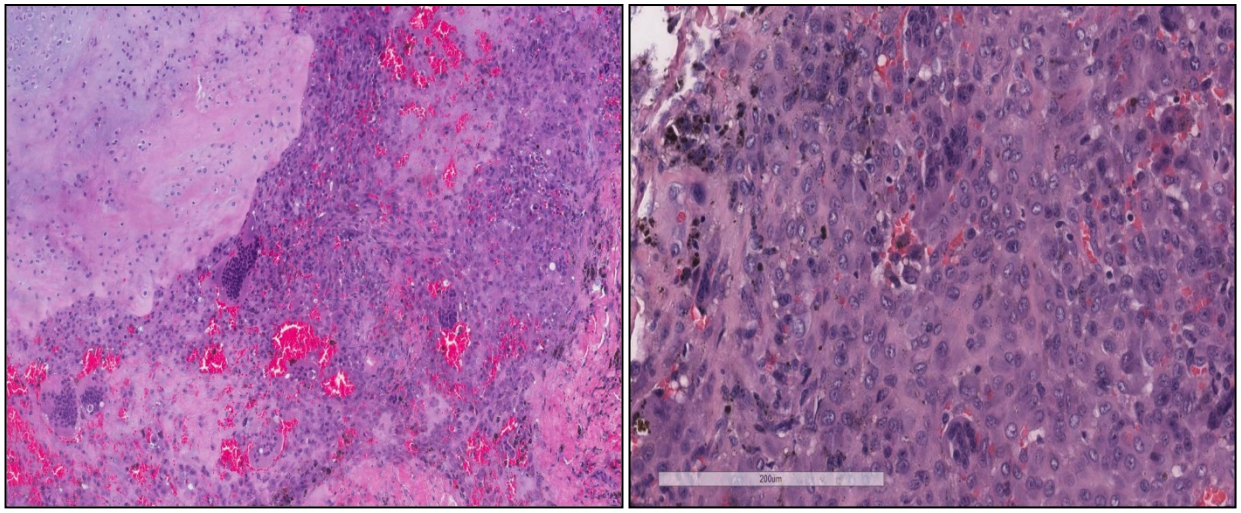
Differential Diagnosis:
- Chondroblastoma
- Pleomorphic Adenoma
- Tenosynovial Giant Cell Tumor (TGCT)
- Giant Cell Reparative Granuloma
- Dedifferentiated Chondrosarcoma

Key Features:
Cytology/Histology:

Fine needle aspiration cytology revealed abundant osteoclast-like giant cells in a background of mononuclear plasmacytoid appearing cells. There is focal hemosiderin pigment within some cells. Low power examination of the excision specimen shows a cellular proliferation with a cartilaginous component and pseudovascular spaces. On high power, the cellular areas consist of scattered multinucleated giant cells surrounded by spindled to epithelioid histiocyte-like cells, some of which contain abundant eosinophilic cytoplasm and nuclear grooves. Focal hemosiderin deposition is present. The cartilaginous component appears mature with no atypia and is well demarcated from the cellular areas.







Immunohistochemistry:
Positive IHC:
CD 68 (Giant Cells)

Negative IHC:
S100
p63

Discussion:

Tenosynovial giant cell tumors (TGCT), previously known as pigmented villonodular synovitis, frequently affect the hands in the localized form and larger joints such as the knee, hip, or shoulder in the diffuse form (1). TGCT involving the temporomandibular joint (TMJ) is rare, with around 70 cases reported in the literature (2). The mean age is 45 years with an gender distribution. The most common presenting symptom is pain. Similar to TGCT involving the knee and hip, TGCT of the TMJ typically is more often diffuse and requires wide local resection. The localized form can present as a parotid, middle ear or intracranial mass, and can be diagnosed cytologically on fine needle aspirates of the parotid gland (3).

The etiology of TGCT is unclear, but the majority of studied cases have rearrangements involving the colony stimulating factor 1 (*CSF1*) gene on chromosome 1 (1p13), frequently as part of a specific t(1;2) translocation involving *COL6A3* (on 2q35) and *CSF1*. These translocations lead to elevated CSF1 levels, which may lead to giant cell formation (4). TGCT of the TMJ may display chondroid metaplasia. In a recently reported case series from the Mayo Clinic spanning 45 years, 7 of 11 cases showed cartilaginous metaplasia; 9 of 11 cases had originally been diagnosed as chondroblastoma. There are few other case reports of TGCT with chondroid metaplasia, the earliest of which was reported in 1990 (5). It has been postulated that TGCT with chondroid metaplasia may actually represent TGCT formed as a reactive process to a primary synovial chondromatosis or TGCT occurring simultaneously with synovial chondromatosis (6).

Take Home Messages:

- TGCT with chondroid metaplasia should demonstrate the typical morphologic features of TGCT and well demarcated islands of mature appearing hyaline cartilage
- In the TMJ, the most important differential is chondroblastoma, with which TGCT has been frequently confused in the past

- Many cases of TGCT have rearrangements involving the *CSF1* gene (1p13), frequently as part of a t(1;2) translocation with *COL6A3.*

References:

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Case 4
Presenter: Aaron Muhlbauer M.D. PGY II
Attendings: Dariusz Borys, MD and Saeedeh Masoom, MD

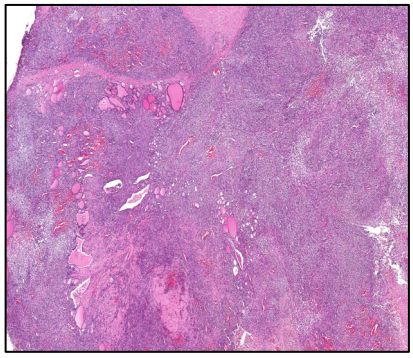
Clinical History: The patient is an 87-year-old male with a rapidly enlarging neck mass was admitted to Hines VA Hospital after a swallow evaluation demonstrated severe dysphagia with visible aspiration. Past medical history was significant for invasive urothelial carcinoma (diagnosed in 2014), multinodular goiter of 1-year duration, and right vocal cord paralysis for the last 2 months. Physical exam showed significant nodularity in the neck bilaterally and severe narrowing of the posterior oropharynx. CT scan of the neck showed a marked increase in thyroid size on the right with mass effect on the trachea, and ultrasonography revealed diffuse heterogeneous enlargement with multiple coarse calcifications. FNA cytology revealed a poorly differentiated malignant neoplasm. The patient underwent a right-sided thyroid lobe exploratory surgery which revealed a firm mass that was adherent to the surrounding tissue. A representative slide from the excisional biopsy from the right thyroid lobe was submitted for review. Grossly, the tissue was white-gray with necrosis and hemorrhage and measured 8.5 cm in greatest dimension.

Final Diagnosis: LEIOMYOSARCOMA OF THE THYROID

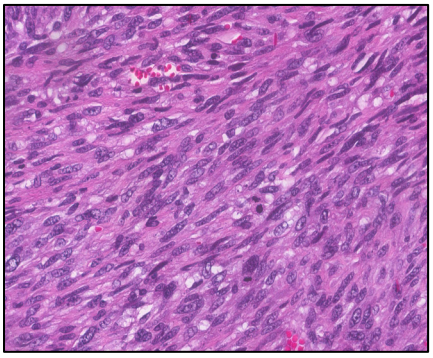
Differential Diagnosis:
- Undifferentiated (anaplastic) thyroid carcinoma
- Thyroid leiomyosarcoma
- Spindle cell squamous cell carcinoma
- Metastatic sarcomatoid urothelial carcinoma
- "Spindle Cell Tumor with Thymus-like Differentiation" (SETTLE)
- Metastatic spindle cell melanoma
- Medullary thyroid carcinoma

Key Features:
Histology:
The thyroid tissue is replaced by a hypercellular tumor composed of spindle cells arranged in long intersecting fascicles. Entrapped benign thyroid follicles are focally seen. The tumor cells are moderately to severely atypical and show elongated hyperchromatic nuclei with round/blunt ends and ill-defined cytoplasm with perinuclear cytoplasmic vacuoles. Tumor necrosis is present. Numerous mitoses are seen.

HE x2



HE x40



Immunohistochemistry:

Positive Stains	Negative Stains
Desmin	AE1/AE3
SMA	CAM5.2
CD99	CK5/6
	P63
	EMA
	S100
	HMB-45
	Melan-A
	TTF-1
	CD34
	Myo-D1
	Myogenin
	PAX-8: Inconclusive

Discussion:

- Leiomyosarcoma of the thyroid is an exceedingly rare tumor. Among all of the tumors of the thyroid gland, leiomyosarcoma accounts for 0.014%(1).
- Leiomyosarcoma of the thyroid has been defined by Kawahara et al(2) as a thyroid malignant neoplasm without any epithelial component lacking immunohistochemical evidence of epithelial differentiation (absence of staining for cytokeratins), the presence of smooth muscle differentiation demonstrated by positivity SMA and desmin and ultrastructural evidence of smooth muscle differentiation. This entity is somewhat controversial, as many authors consider all sarcomatoid neoplasms arising in the thyroid gland are variants of anaplastic carcinoma, irrespective of the immunostaining pattern.
- Both thyroid leiomyosarcomas and anaplastic carcinomas are negative for the usual thyroid markers (TTF1, thyroglobulin), and anaplastic carcinomas can be negative for cytokeratins(3). Therefore, PAX8, if positive, could aid the differential diagnosis, supporting the diagnosis of anaplastic carcinoma(4). However, the literature shows a wide variation of PAX8 positivity in anaplastic carcinoma, possibly due to different antibody clones and interpretation of ambiguous results such as in our case.
- Etiology is controversial. It is thought to arise from the smooth muscle-walled vessels of the thyroid gland periphery. However, others believe it to be a transdifferentiation or metaplasia of an anaplastic thyroid carcinoma(5).
- The clinical presentation overlaps with that of anaplastic thyroid carcinomas. Patients are typically older and present with a rapidly growing neck mass with distant metastases at the time of diagnosis. Prognosis is dismal with a 1-year survival rate of <20%(6).
- Given this grim prognosis and the lack of response to conventional therapeutic modalities of both entities, the differential between anaplastic thyroid carcinoma and leiomyosarcoma of the thyroid is at present more of an academic exercise. However, with the expected development of targeted therapies for both tumors, this distinction may be more clinically relevant in the future.

Take-Home Points:

- Preoperative imaging and gross appearance favored a primary thyroid neoplasm.
- While an undifferentiated (anaplastic) thyroid carcinoma cannot be entirely excluded, the histomorphology showed a purely sarcomatous growth pattern;
- Immunoprofile favored a tumor of mesenchymal origin with no epithelial differentiation identified in any part of the tumor.
- Leiomyosarcomas of the thyroid have a poor prognosis regardless of tumor etiology (smooth muscle origin or transdifferentiation from an anaplastic thyroid carcinoma).

References:

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Case 5

Presenter: Aadil Ahmed, MD PGYIII
Attending: Kamran Mirza, MD, PhD

Clinical History: The patient is a 17-year-old boy who presents with mild abdominal pain, early satiety and night sweat for two weeks. Physical examination reveals conjunctival pallor, abdominal fullness and cervical lymphadenopathy. Laboratory data demonstrates pancytopenia. The patient undergoes an excisional biopsy of an enlarged cervical lymph node, a section of which is provided for your review.

Final Diagnosis: T-CELL/HISTIOCYTE RICH LARGE B-CELL LYMPHOMA

Differential Diagnosis:

Reactive Adenopathy

- Dermatopathic Lymphadenpathy
- Rosai-Dorfman Disease

Infectious Adenopathies

Malignant Adenopathy

- Classic Hodgkin Lymphoma
- Nodular Lymphocyte Predominant Hodgkin Lymphoma
- T-cell/Histiocyte Rich Large B-cell Lymphoma
- Peripheral T-cell Lymphoma (lymphoepithelioid variant, Lennert lymphoma)

Morphology:

Histologic sections demonstrated an effacement of lymph node architecture with a vaguely nodular expansion of cortical and medullary zones. The effacement was due to a proliferation of small, mature appearing lymphocytes and histiocytes with occasional larger cells with a spectrum of morphologic appearances. Focal necrosis was evident.

Immunohistochemistry:

Positive IHC:

- Malignant cells: CD20, PAX5 (strong), BCL-6

Negative IHC:

- Malignant cells: CD30, CD15, CD10, EBER
- Background lacks CD21/CD23 follicular dendritic meshworks
- No CD3/CD4/CD57 T-cell rosettes around large B-cells

Discussion:

The differentiation of T-cell/Histiocyte rich large B-cell lymphoma and Nodular lymphocyte predominant Hodgkin lymphoma is based on a series of clinical,

morphologic, and phenotypic findings. Biologically, some studies have shown both entities to arise from germinal center B-cells. It is hypothesized that early divergence in the evolution of the neoplastic processes, such as loss of PU.1, may be the underlying cause for the pathogenesis of one lymphoma vs. the other. Clinically, low-stage NLPHL is treated differently from T/HR-LBCL and thus the often painstaking patohologisc deliberation of differentiating these lesions is of clinical relevance.

Take home message:

Adenopathy in children, with nodular effacement, scattered large pleomorphic cells admixed with histiocytes in a background of mature appearing lymphocytes can have a range of differential diagnoses. The differentiation between T-cell/histiocyte rich large B-cell lymphoma and Nodular lymphocyte predominant Hodgkin lymphoma remains essential due to differences in therapy and prognosis. Clinical history, morphological differences including immuno-architectural patterns (see below) must be carefully analyzed and a clinico-pathologic correlation remains essential for a correct diagnosis.

Citation: Hartmann S, Döring C, Jakobus C, Rengstl B, Newrzela S, et al. (2013) Nodular Lymphocyte Predominant Hodgkin Lymphoma and T Cell/Histiocyte Rich Large B Cell Lymphoma - Endpoints of a Spectrum of One Disease? PLoS ONE 8(11): e78812. doi:10.1371/journal.pone.0078812

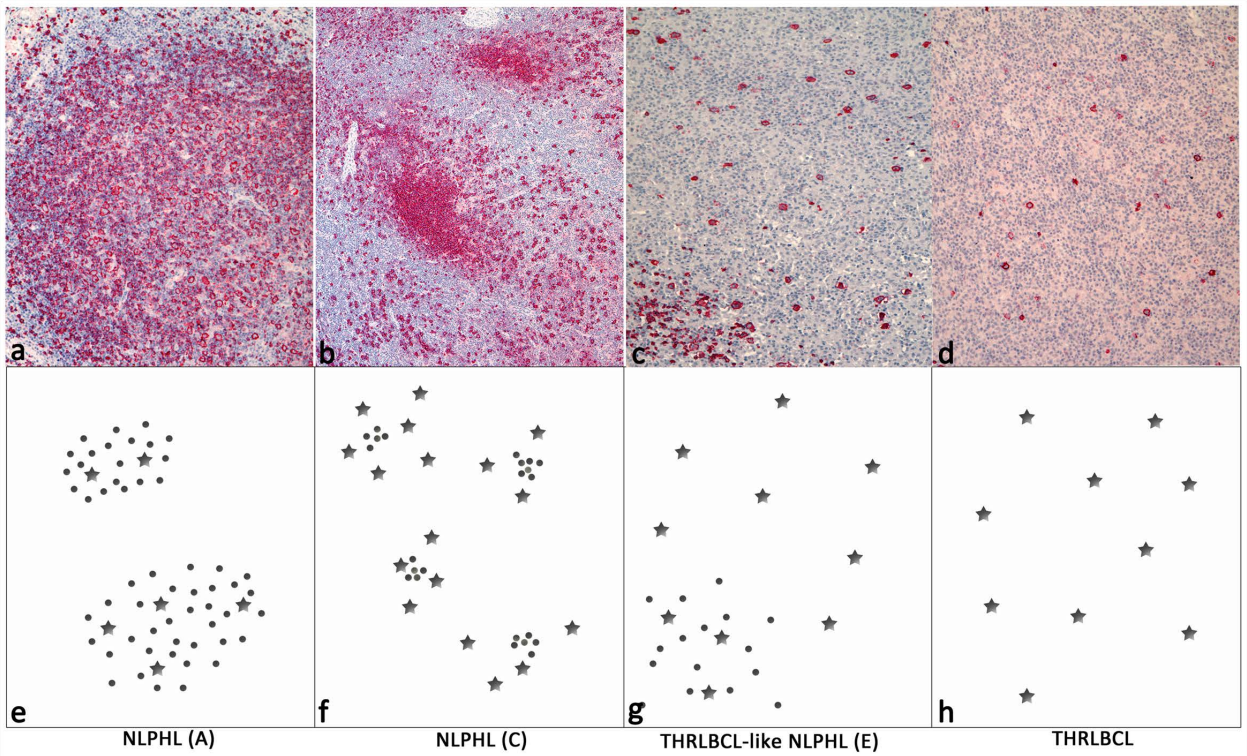

Figure 1. Immunoarchitectural patterns of NLPHL, THRLBCL-like NLPHL and THRLBCL, modified after Fan et al. [5]. a.–d. CD20-immunostainings (100x) of NLPHL patterns A and C, THRLBCL-like NLPHL and THRLBCL. e.–h. Schematic forms of immunoarchitectural patterns. Stars: tumor cells, dots: reactive B cells. a./e. Typical NLPHL Fan pattern A; b./f. NLPHL Fan pattern C; c./g. THRLBCL-like NLPHL (Fan pattern E); d./h. THRLBCL.

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1. Abramson JS. T-cell/histiocyte-rich B-cell lymphoma: biology, diagnosis, and management. Oncologist 2006 Apr;11(4):384-92.
2. Pittaluga S, Jaffe ES. T-cell/histiocyte-rich large B-cell lymphoma. Haematologica. 2010;95(3):352-356
3. The WHO Classification of Tumors of Hematopoeitic and Lymphoid Tissues, 2017

Case 6:
Presenter: Neelima Valluru MD, PGY III
Attendings: Stefan Pambuccian, MD and Xiuzhen Duan, MD

Clinical History: The patient is a peri-menopausal 52 year old, G1 P0 woman with no previous abnormal PAP smears, who presents with abdominal bloating and irregular menstrual bleeding. An endometrial biopsy reveals "benign inactive endometrium". Pelvic ultrasound reveals a solid lesion in the right ovary with internal arterial and venous flow by spectral Doppler. Her CA125 levels are within reference range. Bilateral salpingo-oophorectomy reveals a solid lesion with a tan-grey cut surface measuring 7.5 x 6 x 3.4 cm. A representative section from the lesion is provided for your review.

Final Diagnosis: MICROCYSTIC STROMAL TUMOR

Differential Diagnosis:
Ovarian sex cord/stromal tumors
- Thecoma
- Steroid cell tumor
- Sclerosing stromal tumor
- Signet ring stromal tumor
- Microcystic stromal tumor

Miscellaneous tumors
- Solid pseudopapillary tumor of ovary

Key Features:
Histologic summary:
1) Solid cellular areas
2) Microcysts
3) Paucicellular fibrous stroma

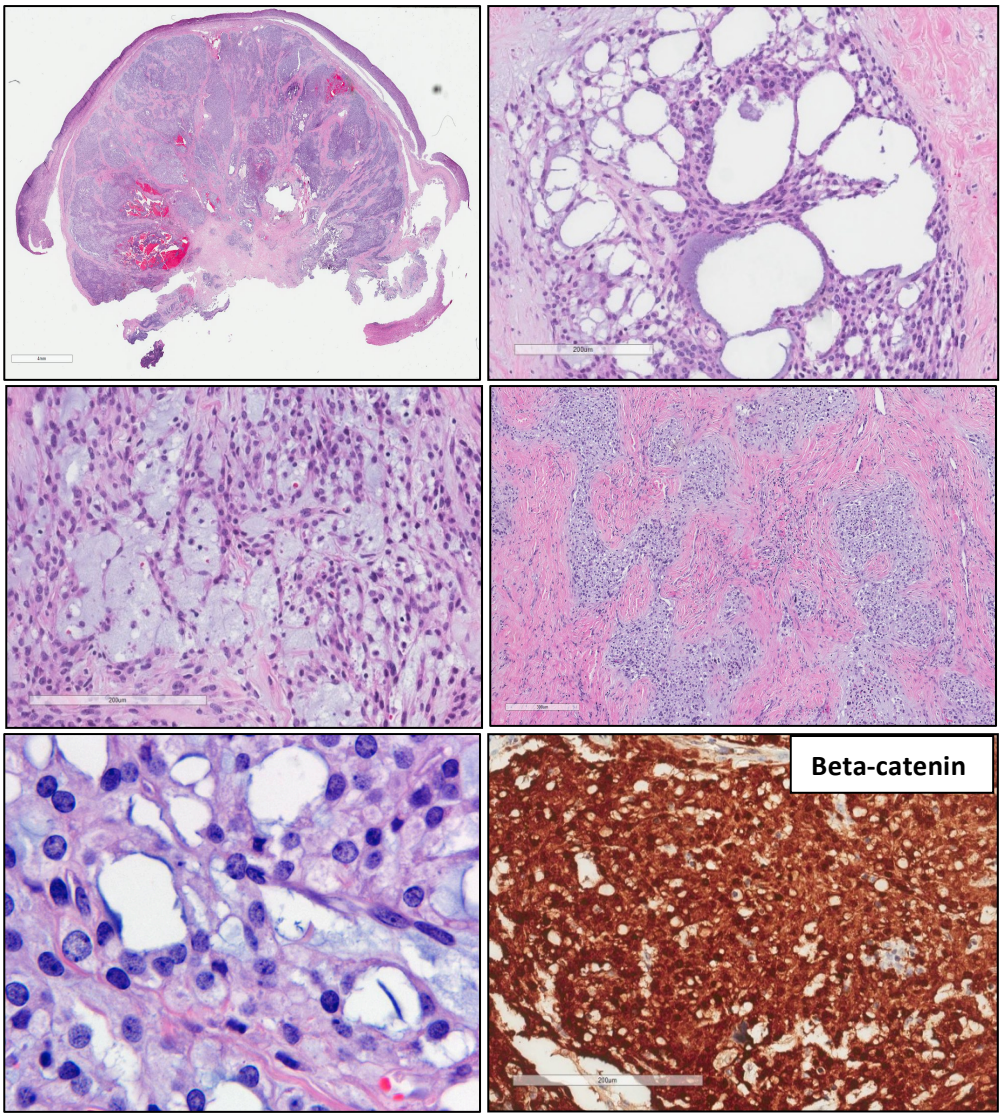
Histology

Histologic sections reveal a well-circumscribed tumor distinct from the adjacent ovarian stroma. The mass is composed of solid cellular areas with focal areas of variably prominent microcysts separated by paucicellular hyalinized stroma, reminiscent of thecoma. The microcysts are characterized by small round to oval cystic spaces filled with clear or slightly basophilic fluid, which coalesce to form larger spaces. The cells have moderate-to-abundant, finely granular, lightly eosinophilic cytoplasm with bland, round-to-oval and spindle-shaped nuclei with fine chromatin and small indistict nucleoli. There is no appreciable mitotic activity.

Immunohistochemically, the tumor cells are negative for cytokeratin (AE1/AE3), inhibin, calretinin and CD99. The tumor cells are positive for CD10 (membranous), beta-catenin (nuclear and cytoplasmic), for cyclin D1, BCL-2 and WT-1.

This morphology and staining profile is characteristic of the microcystic stromal tumor of the ovary, a rare and relatively recently described stromal tumor (Irving and Young, 2009) that has an unusual morphology and IHC staining profile (negative

keratins and usual sex cord/stromal tumor markers – inhibin & calretinin) and positivity for nuclear beta-catenin (corresponding to exon 3 CTNNB1 mutations). β-catenin nuclear positivity is also present in two other ovarian tumors of stromal origin (signet ring stromal tumor and solid pseudopapillary tumor of the ovary), which show some morphologic overlap with microcystic stromal tumor of the ovary.



Take home points:

- Microcystic stromal tumor is a rare tumor entity

- **Histologic characteristics:**
  1) Microcysts
  2) Solid cellular areas
  3) Hyalinized stroma

- **Characteristic immunoprofile for MST include nuclear positivity for:**

    1) **Beta-catenin**
    2) CyclinD1
    3) BCL-2
    4) WT-1
    5) SF-1
    6) FOXL2

- ***CTNNB1* mutation in exon 3**

References:

1. Irving JA, Young RH. Microcystic stromal tumor of the ovary: report of 16 cases of a hitherto uncharacterized distinctive ovarian neoplasm. Am J Surg Pathol. 2009;33(3):367-75.
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3. Irving JA, Lee CH, Yip S, Oliva E, McCluggage WG, Young RH. Microcystic Stromal Tumor: A Distinctive Ovarian Sex Cord-Stromal Neoplasm Characterized by FOXL2, SF-1, WT-1, Cyclin D1, and beta-catenin Nuclear Expression and CTNNB1 Mutations. Am J Surg Pathol. 2015;39(10):1420-6.
4. Podduturi V, Tran T, Champion KJ, Onur N, Shiller SM. Microcystic Stromal Tumor of the Ovary: A Case Report of a Newly Described Ovarian Neoplasm With a beta-catenin (CTNNB1) G34E Mutation. Int J Gynecol Pathol. 2015;34(6):541-5.
5. Meurgey A, Descotes F, Mery-Lamarche E, Devouassoux-Shisheboran M. Lack of mutation of DICER1 and FOXL2 genes in microcystic stromal tumor of the ovary. Virchows Arch. 2017;470(2):225-9.