Conclusion* Assessment of the residual fatty tissue results in the finding of 30% more lymph nodes. This did not result in the detection of more lymph node metastases. We do think it is worthwhile to investigate the residual fatty tissue in women with a high BMI (>30 kg/m2).

213 GENOMIC ALTERATION OF A METASTATIC GASTRIC-TYPE CERVICAL ADENOCARCINOMA: HINTS AND PERSONALIZED TREATMENT OPTIONS

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Introduction/Background* Cervical adenocarcinoma accounts for 20-25% of all uterine cervical cancers with 90% of cases being associated with HPV. Of the remaining, non-HPV associated adenocarcinomas, the most common subtype is the gastric-type (GAS). GAS portends a poorer prognosis irrespective of tumor stage. We report the genomic study of a stage FIGO IVB GAS.

Methodology A 52 years old patient was admitted for atypical glandular cells (AGC) on the cervical smear. Pelvic MRI described a mass infiltrating the proximal third of the vagina, the cervix and the isthmus of the uterus and suggested paraaortic lymphadenopathies. The PET-CT confirmed the nodal involvement and a right iliac bone lesion. Both cervical and bone biopsies were positive for a p53 positive, p16, HER2 and PD-L1 (SP263 clone) negative, GAS. In the absence of standard of care treatment we performed a 52-gene next generation sequencing (NGS) of the cervical biopsies, containing 30% tumor cells

Result(s)* NGS detected the following pathogenic mutations: *TP53* exon 8 (p.Arg280Thr, variant allele frequency (VAF) 18%), *ERBB2* exon 20 (p.Val777Leu, VAF 14%), *KRAS* exons 2 (p.Gly12Ser, VAF 4%) and 4 (p.Ala146Val, VAF 5%).

Conclusion* The most frequently mutations reported in GAS are *TP53*, *STK11*, and *CDKN2A*. The presence of a generally, mutually exclusive, *ERBB2* and *KRAS* in our patient is a rare constellation potentially suggestive of multiple subclones. In the absence of a specific treatment guideline for GAS we initiated a cisplatin, paclitaxel and bevacizumab treatment combination. Based on the NGS results, in case of treatment failure, a MAPK targeted treatment by a MEK inhibitor could be considered.

420 CLINICOPATHOLOGICAL FEATURES OF PATIENTS WITH ENDOMETRIAL STROMAL NODULE AND ENDOMETRIAL STROMAL SARCOMA, SINGLE CENTER EXPERIENCE

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Introduction/Background* To present the clinicopathological features of cases with Endometrial Stromal Nodule (ESN) and Endometrial Stromal Sarcoma (ESS) in our center.

Methodology The cases diagnosed between 2008-2020 were re-examined microscopically. The information for clinical follow-up were retrieved from the hospital archives, retrospectively.

Result(s)* ESN cases consisted of 11 patients between the ages of 41-68 (mean 51,8), Low Grade Endometrial Stromal Sarcoma (LGESS) cases consisted of 13 patients between the ages of 24-53 (mean 44), High Grade Endometrial Stromal Sarcoma (HGESS) cases consisted of 9 patients between the ages of 60-84 (mean 71,2).

Mean tumor size was 3,3 cm (1,7-8 cm) in ESN cases; 6,4 cm (1-13 cm) in LGESS cases; 6,5 cm (4,5-9 cm) in HGESS cases.

In LGESS cases, the number of mitosis in 10 high power fields was 0-7 (average 2,8); In HGESS cases it was counted as 10-45 (average 22). Ki-67 proliferation index was evaluated as 2-5% (mean 3.8%) in LGESS cases; as 30-100% (mean 68%) in HGESS cases.

At the median follow-up of 56,5 months (range, 21-147 months), all of 11 ESN cases are alive. At the median follow-up of 60 months (range, 47-144 months), 1 of 13 case of LGESS have died (%8). At the median follow-up of 8 months (range, 1-53 months), 5 of 9 cases of HGESS have died (% 56). Distant metastasis was seen only in 1 case of 9 HGESS, the location of metastasis is lung.

85% of cases with LGESS were presented in stage I and,% 15 were in stage II. 37.5% of cases with HGESS were presented in stage I, 50% were in stage III and 12.5% were in stage IV.

Conclusion* Endometrial stromal neoplasms are rare lesions of the uterus, usually presenting in the postmenopausal period. The histological type and stage of these lesions are directly related to the clinical course. Average mitotic activity and Ki-67 levels can also provide information in the direction of the clinical course.

496 RELIABLE IDENTIFICATION OF ENDOMETRIAL PRECANCERS THROUGH PAX2, β-CATENIN, AND PTEN IMMUNOHISTOCHEMISTRY

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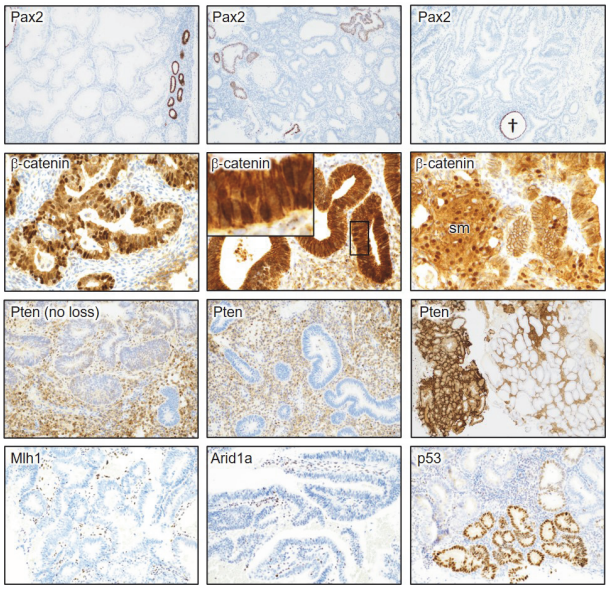
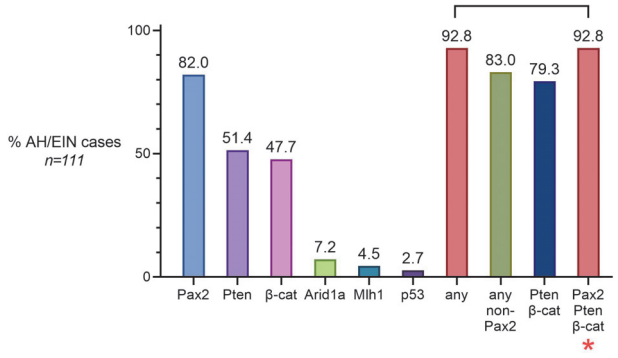
Introduction/Background* The diagnosis of endometrial atypical hyperplasia/endometrioid intraepithelial neoplasia (AH/EIN) remains challenging and subjective in some cases, with variable histologic criteria and differences of opinion among gynecologic pathologists, potentially leading to under/over-treatment. There has been growing interest in the use of specific immunohistochemical markers as adjuncts in AH/EIN diagnosis. For example, the WHO 2020 Classification lists loss of Pten, Pax2, or mismatch repair proteins as desirable diagnostic criteria. Other markers, most notably b-catenin and Arid1a, are also aberrantly expressed in some AH/EIN. However, the performance of these markers individually—and more importantly as a group—has not been rigorously explored, raising critical questions as to which marker(s) or combination(s) thereof is the most efficient and reliable in practice.

Methodology Inclusion criteria was a diagnosis of AH/EIN on an endometrial tissue sample based on histologic features. Formalin-fixed/paraffin-embedded tissue sections from n=111



Abstract 496 Figure 1



Abstract 496 Figure 2

patients were analyzed by immunohistochemistry for 6 markers: Pax2, Pten, Mlh1, b-catenin, Arid1a, and p53. Aberrant expression consistent with an underlying molecular defect was tabulated for each case and marker. An additional set of n=79 normal endometria was also analyzed to define optimal criteria for marker aberrance. The performance characteristics of each marker, the entire panel, and subsets thereof were statistically analyzed.

Result(s)* In order of number of cases detected, the most frequently aberrant markers in AH/EIN were Pax2 (82.0% of cases), Pten (51.4%), b-catenin (47.7%), Arid1a (7.2%), Mlh1 (4.5%), and p53 (2.7%). The great majority of cases showed aberrant expression of ≥2 markers. The 6 markers together identified 92.8% of cases. Arid1a and Mlh1 proved to be robust and readily-scored markers, but all cases showing aberrant expression of either of these two markers was also detected by b-catenin, Pax2, or Pten.

Conclusion* A limited panel of only 3 markers (Pax2, Pten, and b-catenin) showed optimal performance characteristics as a diagnostic adjunct in the histopathologic diagnosis of AH/EIN. Use of this panel was practicable and robust, with at least one of the 3 markers being aberrant in 92.8% of AH/EIN.

672 A CASE REPORT OF BENIGN METASTASIZING LEIOMYOMA

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Introduction/Background* Benign metastasizing leiomyoma (BML) is a rare benign condition involving the extra-uterine spread of smooth muscle cells with histological, immunological, and molecular patterns similar to those of benign uterine leiomyomas. Due to the uncommon nature of the disease, diagnosis and treatment is very challenging. There are no current standardized guidelines for treatment of BML but usually treated with a combination of surgical resection and radiotherapy, followed by hormonal treatment and monitored through radiography.

Methodology This is a case of a 49 year old G0 with an incidental finding of BML in the lungs. Routine pre-employment chest x-ray revealed pulmonary nodules. Computed tomography (CT) scan of the abdomen revealed uterine leiomyoma. Video-assisted thoracic surgery (VATS) with wide resection of pulmonary nodules and frozen section was done. Histopathology revealed BML. Immunostains for p53, Ki-67 and p16 were negative, while immunostain for estrogen and progesterone receptor (ER/PR) were positive. Serial chest radiography monitoring showed no significant interval change in pulmonary nodules and patient remained asymptomatic. Few years later, the patient presented an enlarged abdomen. Transvaginal ultrasound revealed a pedunculated subserous myoma. The patient underwent exploratory laparotomy, extrafascial hysterectomy with bilateral salpingooophorectomy. Histopathology revealed BML.

Result(s)* Currently, patient has been asymptomatic for 6 years with no interval changes in pulmonary nodules size despite having multiple lesions in both lungs.

Conclusion* BML is a monoclonal benign tumor with high metastatic potential. A hormone dependent, which growth depends primarily on estrogen and progesterone. This can metastasize to distant organs commonly in the lung (PBML), hence, confused with leiomyosarcoma. Uterine surgeries such as curettage, hysteromyomectomy and hysterectomy can cause it to spread hematogenously to the lung through venous circulation. Patients are usually asymptomatic at presentation and lesions are only incidentally discovered during routine physical examinations as multiple pulmonary nodules in chest radiography. Thoracoscopic lung biopsy remains the gold standard in the diagnosis for PBML. The most helpful pathologic features that characterize PBML and other BMLs are their low cellularity and mitotic index, and the absence of nuclear atypia and tumor necrosis. Immunohistochemistry staining usually is positive for Desmin, Smooth Muscle Actin (SMA) and ER/PR.