were HPV negative had halo-like cells strongly and uniformly positive for glycogen.
Conclusion: Thus, when combining the PAS/PAS-D plus HPV testing and reevaluation of H&E results, 9/67 (15%) of the cases initially called LGSIL were not, but rather mimics. This underscores the value of ancillary tests in differentiating LGSIL from its mimics.
Supported by a grant from the Lewis Foundation (Gerard J.Nuovo).

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p16 immunostaining pattern helps to distinguish CIN III from atypical immature squamous metaplasia
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Objectives: The role of p16 immunohistochemistry in the differential diagnosis between atypical immature squamous metaplasia (AIM) of the uterine cervix and CIN III was evaluated. Material and Methods:
The total of 128 cervical biopsies was divided into the AIM group (n = 26) and the groups of CIN III (n = 54) and mature squamous metaplasia (n = 48) which served as positive and negative controls. All lesions were analyzed immunohistochemically with p16 antibody. The intensity of immunoreaction (strong; weak) and the horizontal distribution of staining (focal; diffuse) were assessed. Typical staining patterns of positive and negative controls were established and lesions from AIM group were reclassified according to these standards.
Results: The majority of CIN III lesions (96.3%) showed diffuse strong positivity for p16 antibody and the diffuse weak expression was observed in 3.7%. The typical staining pattern of mature squamous metaplasia was negativity (85.7%) or focal weak immunoreaction (14.3%). In AIM group, diffuse strong staining was detected in 30.8% of cases, focal weak positivity in 42.3%, focal strong staining in 7.7%, negativity in 19.2% and diffuse weak immunoreaction was not observed. We conclude that 30.8% cases of AIM may be reclassified as CIN III and 61.5% should be treated as squamous metaplasia. The biologic nature of 7.7% lesions from AIM group remains unclear.
Conclusion: p16 immunohistochemistry is capable to stratify majority of cervical lesions classified as AIM into the groups of CIN III and squamous metaplasia. The category of AIM should therefore not be used for the classification of squamous lesions of the uterine cervix.
Supported by the research projects GAUK 85608 and MZOFNM2005.

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Uterine adenomyolipoma. A case report and review of the literature
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Introduction: Uterine adenomyolipoma also termed adenolipoleiomyoma or lipoadenofibroma is a rare benign, polypoid or less frequently intramural lesion, considered to be of hamartomatous origin or represent an unusual type of benign Müllerian mixed tumour with a heterologous element. We report another case of this uncommon lesion and review the previously documented similar cases.
Patient: A polypoid endometrial mass measuring 28 × 8 × 5 mm was removed during explorative curettage from a 56-year old woman presented with postmenopausal vaginal bleeding. Histologically, it was composed of tubular and cystic glands within a background of connective tissue containing endometrial stroma, smooth muscle and mature adipose tissue. Many glands were of endometrial proliferative type, while elsewhere glands were of tubal type with cilia or occasionally of endocervical type. Neither the epithelial nor the mesenchymal elements showed any cytological atypia, necrosis or mitotic figures. Periglandular stromal condensation was also absent. The glanudular epithelial cells were immunoreactive for cytokeratin, epithelial membrane antigen and vimentin, but negative for carcinoembryonic antigen. The endometrial stromal cells were CD10 positive; smooth muscle cells were desmin and alpha-smooth muscle actin positive, while adipocytes were S100 protein positive. The nuclei of most of the glandular cells and some of the endometrial stromal and smooth muscle cells were both ER and PR positive. The proliferation index (Ki-67) was <1%.
Conclusion: After extensive literature search, the case presented in this article appears to be the ninth report of this unusual uterine lesion occurring most frequently in postmenopausal women and the seventh presenting as an endometrial polypoid mass.

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The p16 protein expression and methylation of p16INK4a gene in patients with high-grade squamous intraepithelial lesion of cervix
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Introduction: Cervical cancer is the second cause of cancer death involving 20–39 year-old women and emerging molecular evidence implies HPV in the carcinogenesis of this lesion. In this process, p16 expression rises as a compensatory mechanism, and methylation of its promoter has been associated with inactivation of its function in cervical cancer as well, although the data is still controversial.
Material and Methods: Seventeen HSIL samples were analyzed by PCR for the presence of HPV genome and p16 promoter methylation, and by immunohistochemistry for p16 expression.

