beam radiotherapy if nodal status unknown and to consider vaginal brachytherapy if node negative. 5) For high risk to consider EBRT vs vaginal brachytherapy.

Methods All stage I endometrial cancer patients registered to our institution from June 2015 to March 2018 were selected from database. Electronic record of case notes, histology, blood results, imaging results and multi-disciplinary team meeting outcomes were retrospectively reviewed.

Results A total of 120 patients, age 32–88 years (median age 65 years). 113 patients underwent surgery (87 had TH + BSO and 26 had TH+BSO+lymphadenectomy). 7 patients were not fit for surgery and treated with hormone. Post op histology showed 76 patients G1, 20 patients G2 and 17 patients G3. 111 patients had FIGO IA and 2 patients had IB. 26 patients were given adjuvant radiotherapy (3 EBRT and 23 Brachytherapy).

Conclusions Rate of adherence with BGSC guidelines for surgery and adjuvant radiotherapy were 90% and 88.5% respectively. Some grade changes between pre and post-op histology, findings in clinical examination and imaging were attributed to the main management reason to treat outside BGCS guidelines. Recurrent rate was 2.5%.

Conclusions Mel-18 was highly expressed in EC and promoted cell proliferation, migration and positively regulated cell cycle progression via PI3K/AKT/mTOR pathway.

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OVEREXPRESSION OF MEL-18 ENHANCES PROLIFERATION, MIGRATION AND POSITIVELY REGULATES CELL CYCLE IN ENDOMETRIAL CANCER VIA PI3K/AKT/mTOR PATHWAY

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Objectives To detect the expression of Melanoma protein 18 (Mel-18) in endometrial carcinoma (EC) and evaluate the biological effects of Mel-18 on the proliferation, immigration and cell cycle of EC cells.

Methods Immunohistochemistry (IHC), Western blotting and RT-qPCR assays were used to examine the expression of Mel-18 in EC. Adenovirus and siRNA were used to regulate Mel-18 gene levels in cells. The MTT dye solution and colony formation assay were used to detect the cell proliferation activity. Transwell migration Assay was used to detect the cell immigration ability. The cell cycle was detected by flow cytometry. Western blotting was used to detect the related proteins expression in PI3K/AKT/mTOR pathway.

Results Mel-18 mRNA and protein were both highly expressed in EC (P < 0.05). The Mel-18 mRNA and protein expression were both significantly increased by transduced with adenovirus encoding Mel-18 cDNA (P <0.05). Meanwhile, The Mel-18 protein and mRNA levels were significantly reduced by transfected with siRNA-Mel-18 (P <0.05). Up-regulation of Mel-18 was significantly promoted the cell viability, clonality and migration capacity (P <0.05). The percentage of cells at S + G2/M phase was significantly increased in Mel-18-over-expressing cells (P < 0.05). We also explored the potential mechanism of Mel-18 in EC cell lines. Overexpression of Mel-18 activated the PI3K/AKT/mTOR pathway, the expression of PI3K p85α, p-AKT, p-mTOR, c-myc, cyclin D1 and bcl-2 proteins were significantly increased, but bax protein was decreased.

Conclusions The suspicion of STUMP is supported by the ultrasound finding of a single or multiple lesion, isoechoic or mixed echogenicity, without shadowing, with regular borders, internal microcystic anechoic areas and vascularization from minimal to high both circumferential and intralesional.

Vulvar and Vaginal Cancer

VAGINAL CANCER WITH UTERINE PROLAPSE: A RARE ENTITY

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