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PS-02-070

Clinicopathological features, immunohistochemical profile and clinical outcomes of 27 primary peritoneal carcinomas: a single institutional study

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Background & objectives: Primary peritoneal carcinomas (PPCs) are rare tumours, with overlapping clinic-pathological features with epithelial ovarian carcinomas(EOCs) and mesotheliomas. We studied clinicopathological features, including immunohistochemical profile and clinical outcomes of PPCs diagnosed at our Institution.
Methods: This was a retrospective study, wherein 27 PPCs, diagnosed between January 2008 and May 2019 were included, after review, as per established criteria. Various clinicopathological features were analysed with Median and average age of patients being 60 and 55 years, respectively.
Results: Microscopically, 93% tumours (25/27) were of high-grade serous type. Sensitivity and specificity of PAX8 was 100% (13/13) and 87.5%, and for oestrogen receptor (ER) was and 100% (7/7) and 100%. Most patients (14/23, 60.9%) were treated with neoadjuvant chemotherapy (NACT), interval debulking surgery (IDS) and adjuvant chemotherapy. Median disease-free survival (DFS) was 32 months. Estimated 3 year-DFS and overall survival was 47.3% and 69.8%. There were lesser recurrences in cases of NACT and IDS (4/14) vs. surgery and adjuvant CT (4/8) (p=0.59).
Conclusion: This constitutes the largest series on clinicopathologic profile of PPCs from our subcontinent. PAX8, ER and calretinin constitute as useful diagnostic immunostains. It is crucial to differentiate these tumours from their mimic, mesotheliomas, in view of associated treatment implications.

PS-02-071

CEACAM1 expression in the normal uterus of rats

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Background & objectives: The carcinoembryonic antigen-related adhesion molecules (CEACAM) play a significant role in numerous physiological processes, such as cell-cell and cell-ECM adhesion, angiogenesis, proliferation, etc.
The objective is characterization of the CEACAM1 expression pattern in normal tissues of rat uterus.
Methods: Uterus sections were taken from intact female wild-type Wistar rats. The immunohistochemical investigation was performed utilizing mAb Be9.2 (α-rat-CEACAM1, N domain binding, mouse IgG1 kappa), mAb 11-1H (α-rat-CEACAM1, not-N domain binding, mouse IgG1 kappa) and isotype matched control antibodies kindly provided by B.B.Singer. Goat anti rat-HRP coupled antibody and a DAB substrate were used for visualization of the CEACAM1 expression.
Results: CEACAM1 expression was found on the apical cellular polarity of the luminal and glandular columnar cells along the surface of normal endometrium and endocervix. This localization was coinciding with the location of the cellular microvilli. Interestingly, the use of mAb 11-1H allowed to detect week CEACAM1 expression only in the luminal and single glandular epithelium, although mAb Be9.2 was expressed by all endometrium. As expected, single leukocytes diffusely scattered in the underlying stroma were CEACAM1-positive as shown by Be9.2 and 11-1H binding and thus served as internal staining control for both antibodies.
Conclusion: The luminal and glandular epithelium of normal endometrium and endocervix express significant amounts of CEACAM1 on the apical cell surface. However, mAb Be9.2 showed a higher sensitivity for CEACAM1 in uterus epithelial cells during immunohistochemical investigation than mAb 11-1H.

PS-02-072

Differential proteomic analysis between low grade, early stage endometrioid endometrial carcinoma and benign endometrium

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Background & objectives: Low grade, early stage endometrioid endometrial carcinomas are the major proportion of endometrial carcinomas. As a previous step to identify diagnostic biomarkers, we aimed to elucidate the differences at the proteomic level between tumour tissues and matched uterine non-tumour tissues.
Methods: Tumour and non-tumour tissue including endometrium and myometrium from 16 patients was analysed. Proteins were extracted from formalin-fixed paraffin-embedded tissue. Quantitative proteomic analysis was done by isobaric labelling with tandem mass tags (TMT). Tryptic peptides were performed using a Q Exactive mass spectrometer coupled to a nEasy-nLC 1000 nano system (ThermoScientific). MS data were analysed with Maxquant using standardised workflows.
Results: A total amount of 3,113 proteins were quantified and 730 were differentially expressed between both conditions. Relevant pathways affected included integrin signalling and inflammation mediated by chemokine and cytokine signalling. The main biological processes altered were cellular and metabolic processes.
Conclusion: Integrin signalling and inflammation mediated by chemokine and cytokine signalling pathways are promising pathways to identify diagnostic biomarkers in low grade, early stage endometrioid endometrial carcinomas.
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PS-02-073

A morphological and immunohistochemical comparison of primary low grade, early stage endometrioid endometrial carcinomas and their relapses

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Background & objectives: Low grade, early stage endometrioid endometrial carcinomas fall into copy number low and microsatellite unstable molecular groups. Our main objective was to analyse concordance between morphological features and immunohistochemical markers used for molecular classifying in primary tumours and their relapses.
Methods: A total of 19 relapse biopsies from 16 patients were identified from a single hospital cohort comprising 258 low grade, early stage endometrioid endometrial carcinomas. Morphological features such as grade, squamous and mucinous differentiation were evaluated in primary and relapses. Tissue microarray were constructed and immunohistochemical markers for mismatch repair proteins (MLH1, PMS2, MSH2 & MSH6) and p53 were performed.
Results: There were 16 biopsies from locoregional relapses and 3 from lung metastases. Concordance was poor for morphological features such as grade (kappa=0.023), squamous (kappa=-0.203) and mucinous differentiation (kappa=0.215). Tumour grade at relapse was the same in 8 cases, upgraded in 8 and downgraded in 3. Microsatellite instability interpretation derived from mismatch repair proteins expression and p53 expression had a perfect concordance (kappa=1).