Poster (O29)

Epithelial Ovarian Cancer including Borderline Tumor

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Clinical outcomes of immunohistochemistry of p53 staining pattern in high-grade serous ovarian carcinoma

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Objective: To investigate the prevalence of p53 mutations and the associated factors between the immunohistochemistry (IHC) of p53 staining pattern among patients with high-grade serous ovarian carcinoma (HGSOC).

Methods: The study was a retrospective study and enrolled a total of 62 patients with HGSOC, who underwent surgery in Srinagarind Hospital, between January 2016 and December 2020. Histological examination was reveal based on the combination of morphology and IHC staining with p53. The p53 immunostaining pattern was interpreted as a missense mutation, nonsense mutation, or wild-type pattern. Missense (p53 overexpression pattern) and nonsense mutation (null expression p53 pattern) were considered to be p53 mutation. A wide-type pattern was defined as p53 non-mutation.

Results: The p53 mutation was identified in 93.55% of patients. When subgroup analysis in p53 mutation group between overexpression p53 pattern and null expression p53 pattern in terms of clinicopathological characteristic and initial treatment was performed. Patients with overexpression p53 pattern had more omental metastasis compared to those with null expression p53 pattern (90.0% vs. 64.7%, $p=0.042$). There were no statistically significant differences in terms of progression free survival (PFS) (9 months vs. 10 months, $p=0.813$) and overall survival (OS) (12 months vs. 17 months, $p=0.526$) were observed between the 2 groups.

Conclusion: The prevalence of p53 mutations in HGSOC in this study was 93%. Omental metastasis is the one significant pathological factor for predicting an overexpression p53 pattern of HGSOC. However, IHC of p53 staining pattern did not impact on OS and PFS among patients with HGSOC.

Poster (O30)

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Efficacy analysis of niraparib using patient-derived xenografts of rare subtypes of ovarian cancer

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Objective: Niraparib, a PARP inhibitor, shows an antitumor effect by synthetic lethality in tumors with homologous recombination deficiency. However, the efficacy of niraparib for rare subtypes of ovarian cancer remains unclear. In this study, we investigated the efficacy of niraparib using patient-derived xenograft (PDX) models with rare subtypes of ovarian cancer.

Methods: We tried to establish PDXs by transplanting patients' tissue specimens subcutaneously into NOG mice. Immunohistochemical (IHC) staining for SLFN11 (D-2 sc-515071X, Santa Cruz), a possible efficacy biomarker of PARP inhibitor, and genetic analysis of 160 cancer-related genes (GeneRead Human Comprehensive Cancer Panel, Qiagen) were performed using the parental tumors. Response to carboplatin (CBDCA) and niraparib was analyzed using the PDX models.

Results: A total of 3 PDXs with rare subtypes of ovarian cancer were established including carcinosarcoma (CS), adenocarcinoma (Adeno) with neuroendocrine carcinoma (NEC), and clear cell carcinoma (CCC). Histologically, PDX tumors generally mimicked their parental tumors. Positive staining for SLFN11 was observed in CS and Adeno with NEC, both of which had TP53 alterations, while only the CS case had BRCA2 pathogenic mutation. In the PDX of CS, both CBDCA and niraparib suppressed tumor growth in a dose-dependent manner. In the PDX of Adeno with NEC, CBDCA significantly suppressed tumor growth, while niraparib did not. Neither CBDCA nor niraparib inhibited tumor growth in the PDX of CCC without BRCA mutation.

Conclusion: The combination of SLFN11 expression in IHC and BRCA status may be an efficacy biomarker for niraparib in rare subtypes of ovarian cancer.

Poster (O31)

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High gradient magnetic separation of circulating endothelial cells as a diagnostic tool for ovarian cancer

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