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335 HOXA9 METHYLATION IN CIRCULATING TUMOR DNA AS A PROGNOSTIC BIOMARKER IN PATIENTS WITH PLATINUM-RESISTANT OVARIAN CANCER

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Objectives Platinum-resistant ovarian cancer (OC) remains a challenge with few or no treatment options. Methylation of the HOXA9 gene has been found in plasma of patients with OC. It does not, however, occur in blood from healthy individuals. The aim of this study was to evaluate if HOXA9 methylation could predict outcome and identify patients who can benefit from palliative chemotherapy.

Methods Plasma from 27 patients with platinum-resistant OC was analyzed by digital PCR with a HOXA9 methylation-specific assay at baseline and before cycle two. The fractional abundance of methylated HOXA9 was calculated and the patients with values increasing above the 95% confidence interval of baseline values was compared with patients having stable or decreasing values. The primary endpoint was progression free survival (PFS).

Results At baseline 22 patients (81.5%) had measurable HOXA9 methylation in plasma. Patients (N=4) with a significant increase in HOXA9 methylation after the first cycle had a median PFS of 1.4 months compared to 5.4 months in patients (N=23) with stable or decreasing HOXA9 (p=0.0019). Nine patients were HOXA9 negative before cycle two. The median PFS in this group was 9 months compared to 2.6 months for patients (N=18) with measurable HOXA9 at second cycle (p=0.001) (figure 1).

Abstract 335 Figure 1 PFS HOXA9 status at 2nd treatment cycle

Conclusions The study demonstrated that an increase in HOXA9 methylated DNA could be used as an early marker to predict poor outcome in platinum-resistant OC. Furthermore, absence of HOXA9 methylation was prognostic favorable indicating the potential to identify patients who can benefit from palliative chemotherapy.

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336 INCREASED CA125 LEVELS AND SEROUS HISTOLOGICAL SUBTYPE ARE ASSOCIATED WITH MICROINVASION IN WOMEN WITH BORDERLINE OVARIAN TUMORS

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Objectives We aimed to assess the following morphological characteristics of serous and mucinous borderline ovarian tumors (BOT) as stratified by Kurman: microinvasion, non-invasive implants or/and invasive implants, linfo-nodal invasion, microinvasive carcinoma, intraepithelial carcinoma and mural nodules. Once they were determined, we assessed the relationship between microinvasion and clinical characteristics and the other morphological features addressed above.

Methods 74 women with serous and mucinous BOT were selected. Two experienced pathologists following Kurman criteria determined morphological characteristics. Disease-free was calculated and compared to presence of microinvasion as well as clinicopathological features. We also calculated the relation between the presence of microinvasion and all other morphological features.

Results A mean follow-up period of 57.6 months was achieved where 48% of patients had serous BOT and 52% had mucinous tumors. Microinvasion was the most frequent morphological feature detected in both subtypes. The patients with microinvasion had higher CA125 levels (172.6 + 255.9U/mL vs. 78.6 + 114.5U/mL; p=0.04) than their counterparts. In addition, in patients with microinvasion, the serous subtype was more prevalent than in those patients without microinvasion (65.4% vs. 38.2%; p=0.02). Microinvasion was not associated with any other clinical characteristic nor the presence of any other morphological feature.

Conclusions Microinvasion is one of the most controversial BOT feature considering management, prognosis and follow-up. In our study, microinvasion was significantly associated with higher CA125 levels and serous subtype. In ovarian cancer, CA125 levels are associated with disease extent. Therefore, higher CA125 levels in women with microinvasion may indicate tumors affecting more aggressively the celomic epithelium.

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337 INCIDENCE AND PATTERNS OF RECURRENCE OF OVARIAN CANCER IN INDIA; A STUDY FROM CANCER CENTER IN NORTHERN INDIA

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Objectives

1. To calculate the disease-free survival (DFS) and overall survival (OS) in ovarian cancer patients with non-metastatic disease and stage IV disease with pleural effusion only.
2. To assess the paclitaxel/carboplatin related toxicity profile in study group cases.