**Abstract 971 Figure 2**

**971**

**NK CELL-MEDIATED ERADICATION OF OVARIAN CANCER CELLS WITH A NOVEL CHIMERIC ANTIGEN RECEPTOR DIRECTED AGAINST CD44**

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**Introduction/Background**

Disease recurrence and chemoresistance are major causes of poor survival rates in ovarian cancer patients. Ovarian cancer stem cells (CSC) were shown to represent a source of tumor recurrence owing to the high resistance to chemotherapy and enhanced tumorigenicity. Chimeric antigen receptor (CAR)-based adoptive immunotherapy represents a promising strategy to reduce the risk for recurrent disease. We developed a third-generation CAR to specifically target CD44, a widely accepted ovarian CSC marker.

**Methodology**

We used ovarian cancer cell lines A2780, SKOV3, and OVCAR3 as well as primary ovarian cancer cells (P1, P2 and P3) harvested from ascites samples of an ovarian cancer patient. An anti-CD44 CAR was generated based on a third-generation CAR design containing CD28 and 41BB as codomains. NK-92 cells were equipped with CAR constructs by lentiviral transduction. IFNγ release assays were performed to demonstrate specific activation of engineered NK cells. Live cell impedance analysis with xCELLigence represents a promising strategy to reduce the risk for recurrent disease. We developed a third-generation CAR to specifically target CD44, a widely accepted ovarian CSC marker.

**Result(s)**

NK92 cells equipped with the anti-CD44 CAR (CD44NK) showed specific cytotoxic activity against CD44-positive ovarian cancer cells and primary ovarian cancer cells (figure 1). Specific activation of engineered NK cells was demonstrated by IFNγ secretion assays. Interestingly, CD44NK cells demonstrated cytotoxic activity under cisplatin treatment. The simultaneous treatment with CD44NK and cisplatin showed higher anti-tumor activity compared to a sequential treatment as shown in figure 2.

**Conclusion**

The new anti-CD44 CAR proved specific killing in ovarian cancer cell lines and primary ovarian cancer cells. We showed that CD44NK retained cytotoxicity during cisplatin incubation. The most potent anti-tumor effect was achieved by simultaneous treatment with CD44NK cells and cisplatin. This study will be the basis for further in vivo studies and future clinical developments.

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**Abstract 977**

**MUCINOUS BORDERLINE OVARIAN TUMORS: PATHOLOGICAL AND PROGNOSTIC STUDY**

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**Introduction/Background**

Mucinous borderline ovarian tumors (MBT) are characterized by an epithelial proliferation similar to those of well-differentiated adenocarcinomas but are distinguished by the absence of stromal invasion. They are often difficult to diagnose histologically. On the one hand, the invasion of the stroma is not always easy to highlight, and on the other hand, the indirect criteria of invasion are not
unanimously accepted. The work aims to specify the pathological and clinical features and to highlight the prognostic factors of these tumors.

Methodology Our study was retrospective and descriptive including 49 cases of primary borderline mucinous tumors of the ovary, diagnosed at the Department of Anatomical Pathology and Cytology of Salah Azaiez Institute, for a period of 27 years, going from 1992 to 2019.

Result(s) The mean age of our patients was 48 years old. Histologically, the cases were divided into 34 cases of pure MBT, 13 cases with intraepithelial carcinoma, and 2 cases associating an intraepithelial carcinoma with microinvasion. The majority of our cases were classified FIGO I and only one case was FIGO III. 14 patients received conservative treatment and 32 received radical treatment. The treatment wasn’t specified in 3 patients. The progress was good in the majority of cases. Only one patient had a contralateral recurrence after a follow-up period of 3 years. There was no significant difference regarding the risk of recurrence and risk factors such as age, gestation, hormonal contraception, hormonal status, FIGO stage, presence of peritoneal pseudomyxoma, intraepithelial carcinoma, and microinvasion.

Conclusion The prognosis of TMBL depends closely on their FIGO stage, stage I tumors have a good prognosis. The presence of intraepithelial carcinoma does not influence their prognosis. However, it is necessary to multiply samples to avoid missing a carcinomatous focus with an anarchic invasion of the stroma which constitutes a poor prognosis factor.