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Personalized medicine and Immunotherapy for Ovarian cancer

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Abstract

The general prognosis for women with advanced ovarian cancer is still dismal, despite advancements in surgery and chemotherapy. Although platinum-based chemotherapy has an initial response rate of roughly 60–80%, the majority of patients will have a recurrence and pass away from the disease. However, a precision medicine approach focused on DNA repair has lately given rise to genuine optimism about increasing survival. Many individuals with BRCA germline-deficient and/or platinum-sensitive epithelial ovarian malignancies have seen life-changing effects as a result of the clinical development of PARP inhibitors. Patients' prognosis could also be improved by intraperitoneal chemotherapeutic methods and antiangiogenic medications. Additionally, developing immunotherapeutic possibilities could benefit patient results.

The effectiveness of immunotherapy in treating ovarian cancer is still limited; however, evaluating sensitive/resistant target treatment subpopulations based on tumour biomarker stratification may increase the predictability of response to immunotherapy. These markers include PD-L1, tumor-infiltrating cells, homologous recombination deficit, tumour mutation burden, and intratumoral heterogeneity of neoantigens. The use of these indicators to choose the best candidates for treatment of ovarian cancer is one of the next prospects in the field. The role of immunotherapy in ovarian cancer is discussed in this paper, along with innovative treatments and research designs including tumour biomarkers that improve the chances of immunotherapy effectiveness in ovarian cancer.

Keywords: Ovarian cancer; Poly-(ADP)-Ribose Polymerase; PARP; Synthetic lethality; Platinum Chemotherapy; Ovarian cancer; Immunotherapy; Biomarker

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Introduction

With 7000 new cases identified each year, ovarian cancer is the seventh most frequent malignancy among women in the UK. Cytoreductive surgery and platinum-based chemotherapy make up the usual management strategy for epithelial ovarian cancer treatment. In order to accomplish the best debulking, cytoreductive surgery seeks to remove malignant tissue from the ovaries and surrounding tissues. Chemotherapy is often used either after cytoreductive surgery or in the neoadjuvant situation (to downstage cancer) (to treat microscopic seedling in optimally debulked tumours or reduce tumour burden in suboptimally debulked cancer). Despite improvements in these treatment options, epithelial ovarian cancer patients continue to have poor overall prognoses; in the UK, over 4000 people pass away from the disease annually. However, new developments in the treatment of ovarian cancer have improved survival rates [1-5].

An ovary growth that is malignant is called ovarian cancer. It may come from the ovary itself or, more frequently, from neighbouring tissues that communicate, including the fallopian tubes or the abdominal wall. Epithelial, germ, and stromal cells are the three types of cells that make up the ovary. These cells have the capacity to divide and become tumours when they develop abnormally. These cells have the capacity to invade and spread throughout the body. There may not be any symptoms at all or only hazy ones when this process starts. The progression of the malignancy increases the severity of the symptoms. Bloating, vaginal bleeding, pelvic pain, stomach swelling, constipation,

and appetite loss are a few examples of these symptoms. Lymph nodes, the lining of the belly, the lungs, and the liver are typical sites where the cancer may metastasize.

Age raises the risk of developing ovarian cancer. The majority of ovarian cancer cases appear after menopause. In addition, it affects women more frequently during the course of their lifespan. This encompasses women who never had children, whose ovulation started earlier in life, and those who go through menopause later in life. Obesity, fertility drugs, and hormone replacement therapy are additional risk factors. Hormonal birth control, tubal ligation, pregnancy, and breastfeeding are factors that lower risk. 10% of cases are attributable to inherited genetic risk; women who have BRCA1 or BRCA2 gene abnormalities have a 50% probability of getting the illness. Some familial cancer disorders, like Peutz-Jeghers syndrome and hereditary nonpolyposis colon cancer, raise the risk of ovarian cancer as well. More than 95% of cases of ovarian cancer are epithelial ovarian carcinoma, which is the most frequent kind. Ovarian carcinoma has five primary subtypes, the most prevalent of which is high-grade serous carcinoma (HGSC). Germ cell tumours and sex cord stromal tumours are less common forms of ovarian cancer. Typically extracted tissue is used for a biopsy to confirm an ovarian cancer diagnosis [6-10].

Discussion

Ovarian cancer is the tenth most prevalent cancer among female patients and the fifth largest cause of cancer-related death in women in the United States, where 22,000 patients are diagnosed with it each year. Currently, debulking surgery combined with platinum-taxane maintenance chemotherapy constitutes the front-line standard of care. Following front-line therapy, the five-year survival rate falls to about 45% in patients with optimum debulking (1 cm residual disease) and 80-85% in patients with inadequate debulking (>1 cm residual disease). Extension of this time frame has been a goal of developments in front-line maintenance therapy. More effective maintenance therapy is required because approved maintenance therapy using bevacizumab or PARP inhibitors has been demonstrated to be successful in prolonging progression-free survival (PFS) but not overall survival (OS). Currently, the majority of clinical studies concentrate on targeted strategies, including more recent initiatives to add immune therapies to the landscape of ovarian cancer treatment.

Through a variety of strategies, including but not limited to immunostimulatory cytokines, tumour antigen vaccines, and monoclonal antibodies targeting immunosuppressive ligands released by tumour cells, immunotherapy improves the anticancer immune response. The latter strategy focuses mostly on immunological checkpoint inhibition (ICI). To identify pathogens from self-cells, immune checkpoints such as cytotoxic T-lymphocyte associated protein 4 and its ligand and programmed death receptor-1 and its ligand (PD-1: PD-L1) are used. A T-lymphocyte searches for epitopes that are compatible with its T-cell receptor (TCR) affinity when it comes into contact with a peripheral cell to identify whether it is a pathogen or a self-cell. T-cells recognise the epitope as a self-cell when immunological checkpoints like PD-L1 are present. The T-cell recognises the target as pathogenic in the absence of immunological checkpoints, which triggers the killing response. Immune checkpoints are upregulated by cancer cells, which reduces the local immune response and allows immune evasion. By tying up, you can stop the tumour and T-cell contact at the immunological checkpoint, restoring T-cell cytotoxicity.

Risk factors

Numerous known risk factors can raise a woman's likelihood of getting ovarian cancer. The length of time a woman spends ovulating has an impact on her chance of acquiring ovarian cancer. Ovarian cancer risk may rise as a result of factors that increase the number of ovulatory cycles a woman has. Cells are stimulated to divide during ovulation. Tumors that have the potential to be cancerous may emerge if this division is improperly controlled. A woman's lifetime ovulatory cycles are increased by early menarche and late menopause, which raises her risk of getting ovarian cancer. Being childless raises the risk of ovarian cancer since pregnancy suppresses ovulation. As a result, women who have never given birth have a twice as high chance of developing ovarian cancer as those who have. The risk is increased by both hormone replacement therapy and obesity.

Women who have fewer menstrual cycles, no menstrual periods, breastfeed, use oral contraceptives, have many pregnancies, and have pregnancies at a young age have a lower risk of developing ovarian cancer. Women who have undergone hysterectomy, both ovaries removed, or tubal ligation (often known as having one's "tubes tied") have a lower risk of developing ovarian cancer (an operation in which the uterus is removed). Another risk factor is age. Ovarian cancer is also at risk from non-genetic factors such diabetes mellitus, a high body mass index, smoking, and alcohol usage.

Prevention

In order to prevent ovarian cancer, women with a high genetic risk may want to have their ovaries surgically removed. This is frequently done once a woman has finished having children. This lowers the likelihood that high-risk women will acquire ovarian cancer (by about 96%) and breast cancer (by around 50%). Since they also have a higher chance of developing Fallopian tube cancer, women with BRCA gene mutations typically also have their Fallopian tubes removed at the same time (salpingo-oophorectomy). Due to the method of study, these data might, however, overstate the risk reduction.

A genetic counsellor is frequently recommended to women with a strong family history of ovarian cancer to determine whether it would be advantageous to test for BRCA mutations. Oral contraceptive usage, the absence of menstrual "periods," and tubal ligation all lower the risk. Ovarian stimulation during infertility therapies and the potential development of ovarian cancer may be related. Ovarian malignancies have been associated with endometriosis. Talc, smoking, and human papillomavirus infection have not been found to raise the chance of getting ovarian cancer.

Conclusion

Ovarian cancer with metastatic illness is still a terrible condition. Numerous ovarian cancer patients' lives have been significantly improved during the past ten years. The main reasons for this are: 1) more patients are receiving care in highly specialised, busy gynecologic oncology centres, and 2) well-conducted clinical studies are looking into precision oncology approaches for ovarian cancer. The use of PARP inhibitor treatment in the maintenance scenario has significantly increased progression-free survival in both BRCA germline-deficient and platinum-sensitive ovarian malignancies, which represents a significant advancement. Inherent or acquired resistance to PARPi treatment is one of the ongoing issues. Precision cancer therapies will have more potential to be improved if actionable predictive biomarkers of resistance are developed. Additionally, the development of novel DNA repair inhibitors in clinical trials—including those that target ATM, ATR, WEE1 and other developing targets in solid tumors—will probably have an influence on ovarian cancer therapy. Improvements in survival are also possible thanks to the development of more modern antiangiogenic drugs and the appearance of immunotherapeutic possibilities.

The need to find clinically useful predictive biomarkers inside tumours, and therefore the best possible candidates for these treatments, comes as a result of developments in these precision methods. The development of validated, tumor-based companion diagnostic tests, such as the FDA-approved Foundation One® test or the NCC Oncopanel® test, is crucial in assisting this shift towards "personalised oncology The former test may evaluate the degree of LOH, HRD, and BRCA1/2 expression, as well as the burden of tumour mutations and PD-L1 expression (predictive of immunotherapy response) (predictive of PARPi response). The treatment of ovarian cancer may alter as a result of expanded access to stratified precision treatments and companion testing.

Early research suggested that ovarian cancer may be immunogenic as a result of a number of causes, including homologous repair inadequacy brought on by widespread BRCA mutation. However, compared to the majority of other immunogenic tumour forms, such NSCLC and melanoma, ovarian cancer immunotherapies have had less effectiveness. To increase the effectiveness of immunotherapy application to ovarian cancer, various strategies are being modified. These include selecting patients based on immune profiling, such as MSI-H/dMMR, HRD, and combining ICI with other therapies. Further study is required to properly characterise immunological features common to ovarian cancer, identify ideal response markers, and improve the patient selection for therapy. To reliably predict response, more than one biomarker might be required due to the complicated immunological landscape of ovarian cancer. It is vital to do deeper investigation of efficacy and risk because combinatorial therapies seem to be an optimistic alternative for optimising therapeutic benefit.

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