Perspective

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Ovarian Cancer Conference on Clinical Research

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Description

Ovarian Carcinoma (OC) is a generic term for a number of distinct diseases (histotypes), each of which has its own unique molecular profile, clinical presentation, and developmental origins. As a result, OC management is moving away from a one-size-fits-all approach and toward management strategies that are more molecularly driven and histotype-specific. Treatments, including the use of PARP inhibitors, have made significant progress as a result of our understanding of the driver events that occur in the most prevalent histotype, high grade serous OC. However, not all patients, particularly those with uncommon OC histotypes, are suitable for these agents. A comprehensive comprehension of the molecular landscape in each OC histotype will be necessary for the development of additional targeted therapeutic strategies. Until recently, there were few studies of tumor profiling in rare histotypes; however, over the past ten years, significant advancements have been made. Our understanding of mutational events in endometrioid, clear cell, mucinous, and low-grade serous OC has been significantly enhanced by reports of genomic characterisation. However, there are still significant knowledge gaps. The current state of our knowledge of each histotype is summarized in this review, which also highlights recent developments in these particular diseases and identifies immediate research priorities for accelerating progress toward improving patient outcomes. In accordance with published consensus guidelines, the sixth Ovarian Cancer Conference on Clinical Research of the Gynecologic Cancer InterGroup (GCIG) was held virtually in October 2021. The consensus meeting was held with the intention of achieving agreement on the design elements of upcoming trials in ovarian cancer, selecting important research questions, and identifying unmet needs. Within four topic groups on clinical research in ovarian cancer, including first-line treatment, recurrent disease, disease subgroups, and future trials, twenty statements were developed, refined, and adopted by all 33 GCIG member groups.

Clinical Research

There was unanimous agreement on 14 of the 20 statements, with more than 90% agreement on the remaining six. After active deliberation among the GCIG groups, the high acceptance rate demonstrated that a consensus procedure could be utilized in a virtual setting. These consensus statements will encourage the harmonisation of international clinical research in ovarian cancer in addition to a detailed categorization of unmet needs. Carriers of the BRCA1 and BRCA2 (BRCA) mutations have a high risk of developing ovarian cancer throughout their lives. In this population, oral contraceptives are protective; however, the effects of other forms of contraception, such as implants, injections, and intrauterine devices, are unknown. We conducted a matched case-control study to investigate the connection between women with BRCA mutations' risk of ovarian cancer and the type of contraception they used. Epithelial Ovarian Cancers (EOCs) are more likely to occur in women with endometriosis. We estimate the genetic correlation, evaluate the causal relationship between genetic liability to endometriosis and EOC histotypes, and identify shared susceptibility loci using data from large endometriosis and EOC genome-wide association meta-analyses. Mendelian randomization analyses support our estimates of a significant genetic correlation (rg) between endometriosis and clear cell, endometrioid, and high-grade serous forms of ovarian cancer (rg=0.49–0.19). In a bivariate meta-analysis, 28 loci were found to be linked to both EOC and endometriosis, with 19 of these loci showing evidence of a shared underlying association signal. The disparity in the shared risk suggests that the connection between endometriosis and the various histotypes may be mediated by distinct underlying pathways.

Several target genes are brought to light through functional annotation by utilizing transcriptomic and epigenomic profiles of relevant tissues and cells. This in-depth analysis reveals significant genetic overlap between EOC and endometriosis histotypes, providing valuable genomic targets for gaining an understanding of the biological mechanisms that link the two conditions. With an average 5-year survival rate of 49.1%, Ovarian Cancer (OC) is the most fatal gynecological cancer. Cytoreduction and chemotherapy are still the most common treatments for advanced OC in clinical practice. On the other hand, the poor overall prognosis compels oncologists to develop new treatments. In the treatment of hematological malignancies, the immunotherapy technique known as chimeric antigen receptor (CAR)-T therapy had achieved success. MNRR1, which is also known as CHCHD2, PARK22, and AAG10, is a bi-organellar protein that can bind to respiratory chain complex IV (COX IV) or Bcl-xL in the mitochondria to boost its anti-apoptotic activity. It can promote the expression of genes involved in

mitochondrial biogenesis, migration, and the cellular stress response in the nucleus by acting as a transcription factor. We hypothesize that MNRR1 can regulate metastatic spread because it can regulate apoptosis and mitochondrial respiration, as well as migration. We demonstrate cell-dependent control of MNRR1's stability and binding partners and heterogeneous protein expression levels of MNRR1 across the tested samples using ovarian cancer models. MNRR1 is necessary and sufficient for a focal adhesion and ECM repertoire that can support spheroid formation in addition to its anti-apoptotic and bioenergetic functions.

Ovarian Cancer

Its ectopic expression is sufficient to activate the type 1 collagen, COL1A1, and the adhesive glycoprotein, THBS4. On the other hand, these genes are significantly downregulated when it is deleted. In addition, in a syngeneic ovarian cancer mouse model, the loss of MNRR1 results in a delay in tumor growth, reduced carcinomatosis, and improved survival. According to these findings, targeting MNRR1 may increase ovarian cancer patients' chances of survival. Germline pathogenic variants are linked to the risk of ovarian, tubal, and peritoneal cancer, and the number of known disease-associated genes has significantly increased over time. From a historical point of view, the literature on the subject is reviewed in this study. The objective is to present a timeline of the advancement of knowledge from the early 1900s to the present day. By comparing the Danish gene panel used for screening for suspected hereditary ovarian cancer to international standards, the findings are put into perspective. The first cases of familial ovarian cancer were reported in 1929, and the first suggestion of a genetic component was made in 1950.

Ovarian cancer was found to be linked to both breast cancer and colorectal cancer in the 1970s, when a number of studies found an increase in the incidence of the disease in particular families. The BRCA genes were discovered in the 1990s as a result of extensive research into the inheritance of cancer susceptibility. In addition, several genes with germline pathogenic variants that raise the risk of ovarian cancer have been identified in new genetic technology-based studies. Women with a genetic predisposition to ovarian cancer can now benefit from specific treatments and preventative measures thanks to the discovery of these pathogenic variants. The screening panel for hereditary ovarian cancer in Denmark is expected to include at least ten genes, and it is likely that additional genes will be added in the future. Numerous tumors employ autophagy, a crucial cellular process, to promote and suppress tumor growth. However, the mechanism and function of abnormal autophagy in ovarian cancer are still unknown. The most significant pathway for specific protein degradation is the ubiquitin-proteasome pathway. In every stage of tumor formation and progression, deubiquitinases (DUBs) play an essential role. In this section, we investigate the DUBs that contribute to ovarian cancer's abnormal autophagy. Ovarian cancer has an abnormally high expression of the selective autophagy receptor SQSTM1/p62, as shown by the analysis of the TCGA data. The level of autophagy is negatively regulated by the deubiquitinase PSMD14, as determined by our screening. According to functional studies, knocking down PSMD14 has the opposite effect, whereas increasing PSMD14 expression significantly increases the malignancy of ovarian cancer cells. In addition, in vivo tests demonstrate that ovarian cancer growth, lung, and abdominal metastasis are inhibited by PSMD14 knockdown. PSMD14 inhibits autophagy through the LRPPRC/Beclin1-Bcl-2/SQSTM1 signaling pathway by directly interacting with LRPPRC and inhibiting its ubiquitination. Next, we demonstrate that LRPPRC is positively correlated with PSMD14 expression and that PSMD14 is upregulated in ovarian cancer. We provide insight into the regulatory mechanism of autophagy in ovarian cancer and clarify the role of autophagy in regulating the phenotype of the disease.