Novel Insights in Endometrial Cancers

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Editorial

Breast and endometrial cancers are two of the most frequent malignancies in women. In some cases, these tumours exhibit estrogen-dependent growth, necessitating the use of hormone therapy. Tamoxifen is an effective hormone medication that has traditionally been used for individuals with oestrogen receptor-positive breast cancer. Tamoxifen medication, on the other hand, raises the likelihood of a bad prognosis kind of endometrial cancer. The immunohistochemistry expression of biomarkers, primarily the Estrogen Receptor (ER), Progesterone Receptor (PgR), and Human Epidermal Growth Factor Receptor 2, is used to identify the therapy strategy for breast cancer (HER_2). However, a kind of breast cancer known as Triple-Negative Breast Cancer (TNBC) is more aggressive since it is negative for all of these indicators. As a result, new therapeutic strategies are required. Endometrial cancer is divided into two types based on histology, grade, and gene expression pattern. Endometrial cancer of type I is typically G_1 and G_2 endometrioid cancer, which is thought to be caused by an excess of oestrogen. Papillary serous carcinoma, clear cell
carcinoma, carcino sarcoma, and G_3 cancer are all examples of type II endometrial cancer.

Anti-estrogen therapy is commonly utilized in ER-positive breast cancer; however, its efficacy in type I endometrial cancer is thought to be limited. Breast and endometrial cancers are both classified as hormone-dependent malignancies; nevertheless, the distinctions and similarities of oestrogen signals in each malignancy remain unknown. Furthermore, comparing the hormone-dependent and hormone-independent features of breast and endometrial cancers may help to better understand them. The growing use of genetic analysis has resulted in even more advancements in cancer classification. For example, The Cancer Genome Atlas (TCGA) team divided endometrial cancer into four prognostic categories based on integrated genomic characterization: POLE/ultra-mutated, microsatellite instable/ hyper-mutated, copy number low/$TP5_3$ wild type and copy number high/$TP5_3$ mutant.

The most recent scientific information on therapeutic target molecules, prediction markers of therapy effect, and prognostic variables for endometrial cancer has been compiled. The Levo Nor Gestrel-releasing Intra Uterine System (LNG-IUS) has been shown to slow the progression of atypical endometrial hyperplasia and early endometrioid there is, however, no marker for identifying patients who would benefit from LNG-IUS therapy. A prior study investigated the association between serum or intratumoral Human Epididymis protein 4 (HE_4) and the effect of LNG-IUS. Serum and intratumoral HE_4 levels are elevated in aggressive endometrial malignancies. As a result, serum HE_4 has been identified as a biomarker for predicting the LNG-IUS response. Recently, emphasis has been drawn to the critical function of the DNA Damage Response (DDR) in the chemoresistance mechanism in several forms of cancer.

The main DDR pathways are the cross-talking ataxia telangiectasia mutant and Rad3 related and checkpoint kinase 1 (ATR) and ataxia telangiectasia mutated and Rad3 related and checkpoint kinase 2 (ATM). *In vitro* investigations on endometrial cancer revealed that co-treatment with both ATR and ATM inhibitors increased the lethal effects of chemo-drugs and irradiation. A cluster of microRNAs uniquely expressed in endometrial cancer patients was identified using a novel technique during the search for biomarkers. Extracellular vesicles recovered from patients' peritoneal lavage were used to establish very sensitive and specific indicators of endometrial cancer. Non-coding RNA, which does not code for proteins, was thought to constitute transcriptional noise. However, the regulatory influence of short non-coding RNAs on gene expression, known as RNA interference, has been defined, and the importance of non-coding RNAs such as microRNA and long non-coding RNA has been extensively acknowledged. Two outstanding review studies on the role of non-coding RNA in endometrial cancer have been published, and microRNA and long non-coding RNA are predicted to be therapeutic targets, prognostic markers, and markers for predicting treatment effects or resistance in endometrial cancer. Further bioinformatics analysis has been carried out in order to conduct a full gene expression study. HiChIP chromatin loop analysis was done on an endometrial cell line to discover putative target genes at 16 identified endometrial cancer GWAS risk loci.

These data are expected to be a valuable resource for genetic information on the risk of endometrial cancer and genetic investigations of other endometrial illnesses. Several biomarkers in blood and tissues have been identified for endometrial cancer diagnosis; however none have been converted into therapeutic usage. High-throughput proteomics and advanced genomes are thought to be valuable methods for identifying biomarkers for endometrial cancer detection. These exams should be carried out using non-invasively acquired samples, such as endometrial fluid or peritoneal lavage fluid. The mouse xenograft model is useful for studying tumour development mechanisms and medication effects *in vivo*. The investigation of biomarkers employing an *in vitro* model and database analysis has made significant strides. As technology advances, it will be important to create a bio imaging system that allows for exact inspection.

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