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Study on Molecular Pathophysiology behind Vulvar, Cervical and Cancer in Corpus Uteri

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Introduction

Any cancer that begins in a woman's reproductive organs is referred to as gynecologic cancer. Cancer is always named after the body part where it first appears. Gynecologic malignancies can start anywhere in a woman's pelvis, which is the area beneath her stomach and between her hip bones. Pathology reports offer histopathologic diagnosis; they also include precise information about prognosis and treatment. As a result, pathologists must be well-versed in the staging, classification, and therapy of gynecologic malignancies in order to ensure that their findings convey therapeutically useful information. Similarly, the gynecologic oncologist must be familiar with the terminology used in gynecologic pathology in order to fully comprehend the pathology report [1].

Pathology behind Vulvar Cancer

It begins in the vulva, the outer part of the female genital organs. There are different kinds of vulvar cancer. Single or numerous squamous intraepithelial lesions (VIN) might be macular, papular, or plaque-like. Low-grade SIL (VIN 1) denotes mild dysplasia, while high-grade SIL (VIN 2–3) denotes moderate and severe dysplasia, respectively. The most prevalent type of SIL (squamous intraepithelial lesion) is high-grade SIL (VIN 3), which includes squamous cell carcinoma in situ (CIS). A wide excision is used to treat this condition.

Although the majority of tumors are exophytic, some may be ulcerative. The tumor is made up of invasive nests of malignant squamous epithelium with keratin pearls in the centre. The tumors usually grow slowly and spread to the surrounding skin, vagina, and rectum. They usually spread to the superficial inguinal lymph nodes first, then to the deeper inguinal, femoral, and pelvic lymph nodes [2].

Pathology behind Cervical Cancer

Cervical cancer starts in the cervix, which is the uterus's lower, narrow end. (The womb is another name for the uterus.) CIN (cervical intraepithelial neoplasia) and cervical cancer are caused by persistent HPV infection. A permissive infection is low-grade SIL (CIN1) (i.e. HPV is episomal, freely replicates, and thereby causes cell death). Before a koilocyte can be seen, a large number of virus particles must aggregate in the cytoplasm. Viral DNA integrates into the cell genome in the majority of cases of higher-grade SIL (CIN2-3). HPV 16 proteins produced by the E6 and E7 genes bind and inactivate the tumor suppressor proteins p53 and Rb, respectively, rendering them useless. In many cases of high-grade dysplasia and all invasive malignancies, copies of the complete virus do not accumulate when HPV integrates into host DNA, and koilocytes are absent. HPV types 16, 18, 31, 33, 35, 39, 45, 51, 52, 56, 58, 59, and 68 are commonly found in high-grade CIN cells. HPV types 16 and 18 are detected in 70% of invasive malignancies worldwide, with some regional variation; the other high-risk variants account for the remaining 25% [3].

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SIL CIN is almost always a transformation zone illness of metaplastic squamous epithelium. CIN disrupts the normal maturation process of cervical squamous epithelium, as demonstrated by morphological abnormalities in cellularity, differentiation, polarity, nuclear characteristics, and mitotic activity. Severe dysplasia and CIS are synonymous with high-grade SIL (CIN3).

Pathology behind Cancer in Corpus Uteri:

Endometrial carcinoma: With an age-standardized incidence rate of 8.2 per 100 000, endometrial carcinoma is the sixth most common cancer diagnosed in women worldwide. It is the most prevalent gynecologic cancer and the fourth most frequent cancer in women in developed countries. Endometrial cancer affects three-quarters of postmenopausal women.

Pathology

According to dualistic model of endometrial carcinogenesis, Normal endometrial cells become endometrioid carcinoma, according to this hypothesis, due to replication mistakes, so-called microsatellite instability, and the accumulation of mutations in oncogenes and tumor suppressor genes. Changes in p53 and loss of heterozygosity on numerous chromosomes induce malignant transformation in nonendometrioid carcinomas.

Microsatellite instability (25%–30% of cases), PTEN mutations (30 percent–60 percent), PIK3CA mutations (26%–39%), ARID1A (20 percent), K-RAS mutations (10%–30%), and CTNNB1 (-catenin) mutations with nuclear protein accumulation (25 percent–38 percent) have all been identified in type I endometrioid carcinomas. Most type II nonendometrioid carcinomas, on the other hand, feature p53 mutations, Her-2/neu amplification, and heterozygosity loss on numerous chromosomes. Through tumor development and subsequent p53 mutations, nonendometrioid carcinomas can also arise from endometrioid carcinoma with microsatellite instability [4].

The Cancer Genome Atlas (TCGA) has completed the most thorough genomic investigation of endometrial carcinomas to date. The TCGA has added four new molecular subgroups to the dualistic classification of endometrial carcinoma (types I and II): (1) an ultra-mutated POLE subgroup; (2) a hyper mutated microsatellite unstable subgroup; (3) a copy-number low/microsatellite stable subgroup; and (4) a copy-number high/serous-like subgroup. POLE mutations predict better prognosis, particularly in high-grade cancers, despite the fact that overlapping molecular genetics data make it difficult to distinguish significant prognostic categories. Patients with endometrioid tumors that are serous-like at the molecular level, on the other hand, may benefit from therapy utilized for serous carcinomas. The molecular classification of grade 3 endometrial endometrioid carcinomas demonstrates that they represent a collection of molecular subtypes of endometrial carcinoma rather than a single entity. Prognostic subgroups can be identified using molecular markers, which has therapeutic implications [5].

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