

Polycystic Ovary Syndrome and the Risk of Endometrial Cancer

Guest Editor



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Dear Colleagues,

Endometrial cancer (EC) is one of the most frequent and the fifth most common malignancies among all cancers with an increasing incidence in women. Most cases of EC are hormone-driven, and estrogen (E2) signalling through the estrogen receptor in particular seems to function as an oncogenic signal for EC development. Genes involved in cell cycle regulation, proliferation, and morphogenesis were also discovered to be linked to the early stages of myometrial infiltration in the formation of endometrioid cancer, according to data on global gene expression data. Additional important risk factors for EC in obese patients include higher chronic inflammation, decreased serum adiponectin levels, and insulin resistance.

Polycystic ovary syndrome (PCOS) is the most frequent heterogeneous endocrine disorder, with prevalence rates ranging from 5 to 20 percent. After ruling out other endocrine disorders, the National Institutes of Health (NIH) established the criteria for PCOS in 1990, necessitating the presence of chronic anovulation and clinical and/or biochemical evidence of hyperandrogenism (HA). Women with PCOS present with a three-to five-fold increased risk of EC. This risk is most likely explained by endometrial hormonal, metabolic, and inflammatory abnormalities. In fact, oligo-amenorrhea, unopposed E2, obesity, hyperandrogenism, hyperinsulinemia, inflammation, and genetic susceptibility are all risk factors for EC in women with PCOS. Indeed, approximately 70–80% of endometrial cancers are Type I (EC1) tumours and reported to be over-represented in PCOS. According to in vitro research, endometrial stromal cells from women with PCOS have an inflammatory character that may make them more susceptible to endometrial cancer. Overall, there is a lack of information regarding endometrial cancer in women with PCOS, and larger, more thorough studies are needed to evaluate the underlying mechanisms and risk profiles.

In this special issue, the risk of developing endometrial cancer in PCOS women should be focused on the molecular level in relation to aberrant endometrial milieu in these women.

Key Words: PCOS; Endometrial Hyperplasia; Endometrial Cancer; Unopposed Estrogen Effect; Progesterone Resistance; Endometrial Mesenchymal Stroma/Stem Cells; Obesity; Insulin Resistance; Hyperandrogenism; Inflammation

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