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Original Research Article

Hormone receptors in gynecological cancers: focus on androgen receptor status

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ABSTRACT

Background: Hormonal therapy and chemotherapy remain the primary treatment modalities for advanced and recurrent gynecologic malignancies. The presence or absence of hormone receptors has significant prognostic value in gynecological cancers. Previous studies have shown that high estrogen receptor (ER) and progesterone receptor (PR) levels in endometrial cancer predict favorable survival, while loss of PR expression in ovarian cancers correlates with recurrence. However, the role of androgen receptors (AR) in these cancers is not fully understood.
Methods: This observational study analyzed the immunohistochemical expression of AR, ER alpha (ERα), PR, and p53 in patients diagnosed with gynecologic cancers at a tertiary care center. Hormonal receptor expression was evaluated and correlated with tumor type and histopathological features.
Results: Prominent expression of ARs was observed across all categories of gynecological cancers included in the study. The expression profiles of ER, PR, and p53 varied among tumor subtypes, reflecting their potential prognostic and therapeutic significance.
Conclusions: Androgens appear to play a role in the pathogenesis of gynecological cancers. Hormonal expression profiling may guide future endocrine therapy strategies and could be considered as a potential salvage treatment option similar to that used in ovarian cancers. These findings underscore the importance of detecting hormone receptor expression in all gynecological malignancies.

Keywords: Gynecological cancers, Hormone receptors, Androgen receptor, Estrogen receptor, Progesterone receptor, p53, Endocrine therapy

INTRODUCTION

The incidence of gynecological cancers has been increasing, influenced by genetic predispositions, unhealthy lifestyle choices, and poor dietary habits.[1] Depending on the tumor's site of origin, gynecological cancers include uterine, ovarian, vulvar, vaginal, and the very rare fallopian tube cancer.[2,3] The global cancer observatory (GLOBOCAN) 2022 indicate a rise in new cases and deaths from gynaecological cancers over the next two decades if current morbidity and mortality patterns persist.[4] Gynecological cancers account for 15.25% of all cancer cases in women, and 15.77% of all cancer-related deaths among women. Common treatments for recurrent and advanced gynecological cancers include hormone therapy and chemotherapy. Hormone therapy works by either blocking hormone production or preventing hormones from stimulating the growth and division of cancer cells. The effectiveness of hormone therapy and the presence of hormone receptors are well established in epithelial ovarian cancer. Notably, the expression of estrogen (ER) and PRs is linked to improved survival rates, irrespective for age, site, stage and grade of

the tumor at the time of diagnosis.[5,6] On the other hand, the significance of ARs in predicting the outcome of ovarian cancer is lesser known. However, several studies have indicated that reduced AR expression is associated with an increased risk of extra-pelvic metastases.[7-10] While the positive expression of estrogen (ER) and PRs is well established as a favorable prognostic factor in endometrial cancer, the role of AR is less well defined. Androgens, particularly testosterone and dihydrotestosterone (DHT), along with their receptors, may represent potential therapeutic targets in endometrial cancer.[11]

The aim of this article is present the incidence of hormone receptors in gynaecological cancers and the significance of AR expression in these cancers.

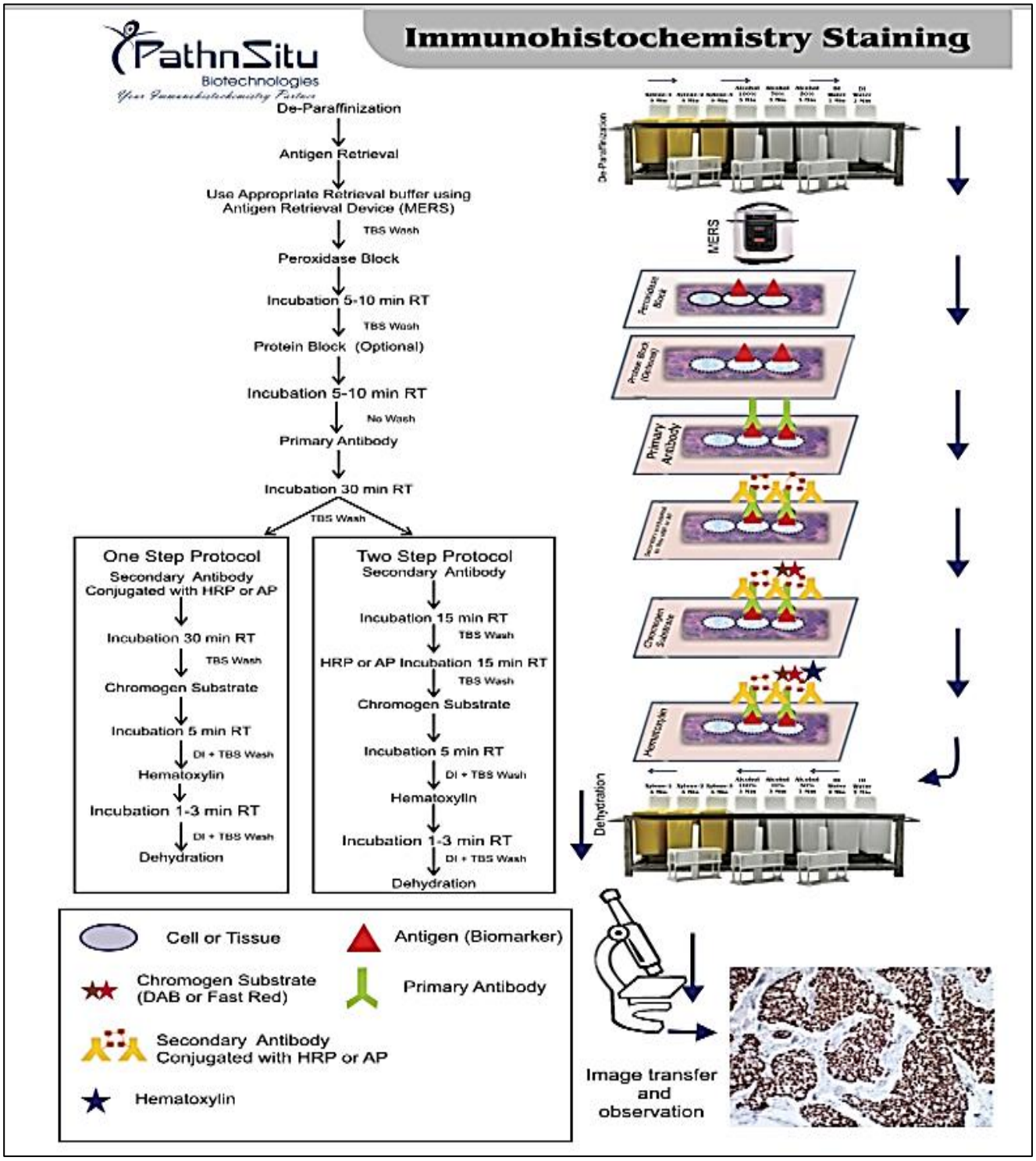

Figure 1: IHC staining method.

METHODS

This study was conducted in the department of gynaecological oncology in a tertiary hospital in Kochi, Kerala, India in year 2016-2018 between October 2016-May 2018 (20 months) after obtaining the approval of the Institutional ethical committee clearance. A total of 109 cases were obtained for the study, out of which 99 had complete follow up details. Five panel immunohistochemistry is performed in each of the 99 cases. Out of the 109 cases 10 cases had missing tissue blocks for which immunohistochemistry could not be performed.

The gynaecological cancer intergroup (GCIG) criteria were followed to evaluate response, progression and recurrent disease as per the consensus. All Recurrences were confirmed by biopsy. The histological diagnoses is based on the WHO criteria.[12] The samples were reviewed and classified as low and high-grade serous carcinoma based on the two-tiered grading system by dedicated experienced gyneconcological pathologist.

Immuno histochemical staining was performed for ER, PR, AR, WT1 and p53 manually. Staining was performed using the (ER, PR, AR-DAKO) and (p53- BIOGENIX) following the manufacturer's protocol.

ER, PR, and AR levels: >10% indicating any degree of positive nuclear staining is regarded as positive. P53 level: intense nuclear staining in more than 60% of tumour cells. Complete absence of staining (null phenotype/nonsense mutation) are taken as positive staining.

The data was analyzed using SPSS for Windows [ver. 26.0, IBM Corp, Armonk, NY]. Categorical data was compared using the Chi-Square test and continuous data was compared using the Welch t-test respectively. Results were presented using graphs and tables. The level of significance was set at $p<0.05$.

RESULTS

It was found that 28.3% of participants were in the age group of 51-60 years, 26.3% of participants were in the age group of 61-70 years, and 20.2% of participants were in the age group of 41-50 years respectively.

The 64.6% of participants had carcinoma of the ovaries, 20.2% of participants had carcinoma of the endometrium, 10.1% of participants had carcinoma of the cervix, about 1% of participants had carcinoma of the vagina, and 4% of participants had uterine sarcoma.

The 57.8% of participants were in the early stage of carcinoma ovaries while 85% of participants with carcinoma endometrium were in the advanced stage. About 80% of participants with carcinoma of the cervix and all participants with carcinoma of the vagina and uterine sarcoma were in advanced stages respectively ($p=0.001$). Family history was associated with 29.7% of participants with carcinoma ovaries, 20% of participants with carcinoma of cervix, 5% of participants with carcinoma of the endometrium, and all participants with carcinoma of the vagina. This distribution is statistically significant ($p=0.02$).

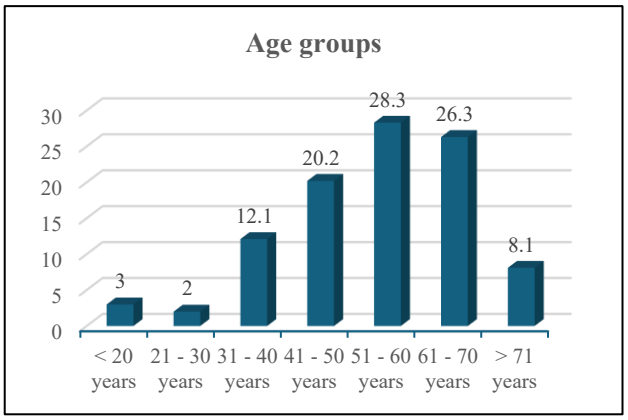

Figure 2: Distribution of participants according to age groups.

It was found that about 89.9% of participants were multiparous and 10.1% were nulliparous.

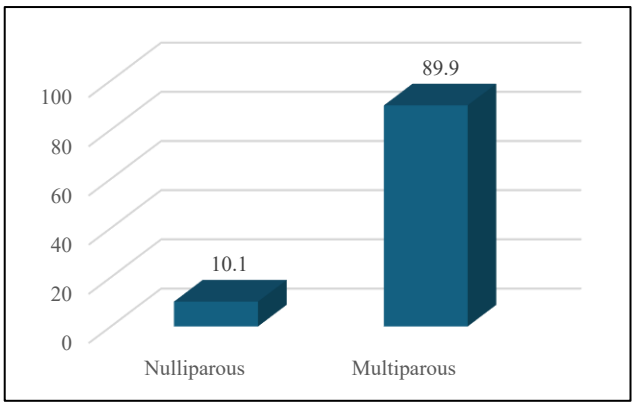

Figure 3: Distribution of participants according to parity.

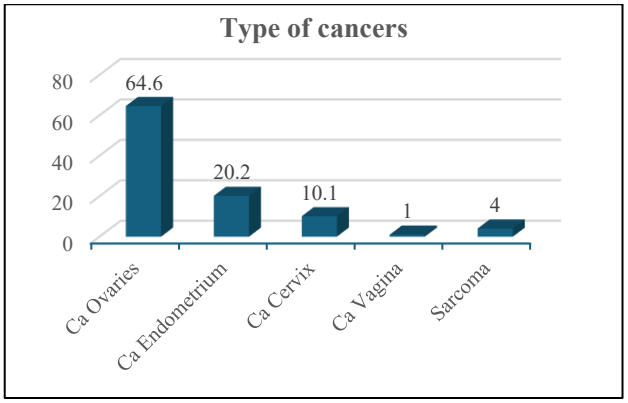

Figure 4: Distribution of participants according to type of cancers.

It was found that the recurrence of carcinoma of ovaries was observed among 18.8% of participants. In addition, the recurrence of carcinoma of the cervix and endometrium was observed among 10% and 5% of participants respectively (p=0.48).

It was found that AR receptors were found to be present among 42.2% of participants with carcinoma of the ovaries, 45% of participants with carcinoma of the endometrium, and all participants with carcinoma of the vagina (p=0.001).

Among 40.6% (n=64) of ovarian cancer 64.1% (n=41) are type II ovarian tumours and among the type II ovarian cancer (n=16) 39% are AR positive.

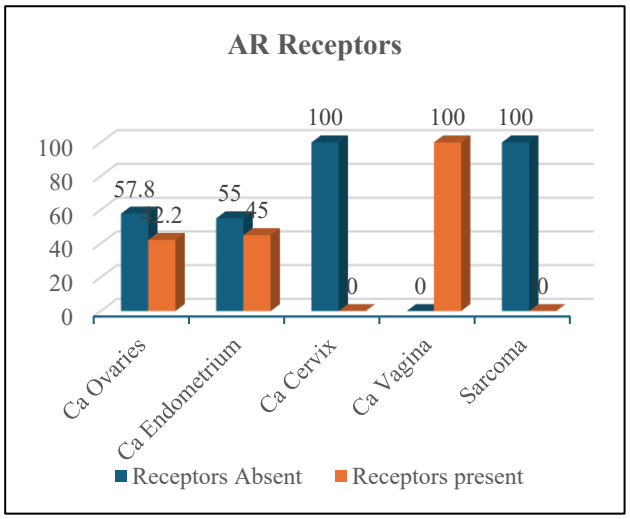

Figure 5: Distribution of participants according to the presence and absence of AR receptors.

ER receptors were found mainly among participants with carcinoma of ovaries and endometrium. ER receptors were found among 85.9% and 95% of participants with carcinoma of ovaries and endometrium respectively (p=0.43).

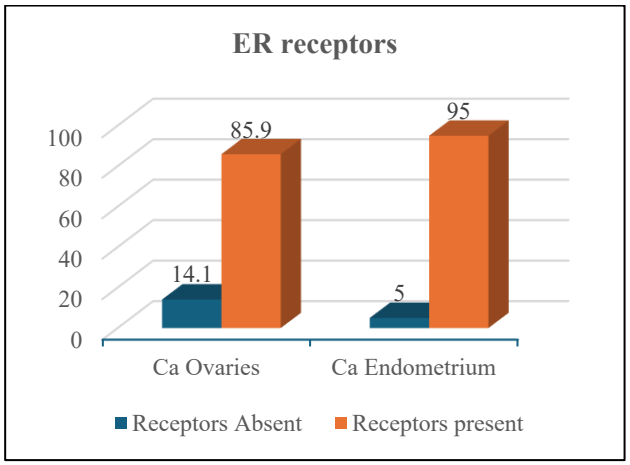

Figure 6: Distribution of participants according to the presence and absence of ER receptors.

PR receptors were found mainly among participants with carcinoma of ovaries and endometrium. PR receptors were found among 56.3% and 85% of participants with carcinoma of ovaries and endometrium respectively. It indicates that more percentage of participants with carcinoma of endometrium had PR receptors when compared to participants with carcinoma of ovaries (p=0.03).

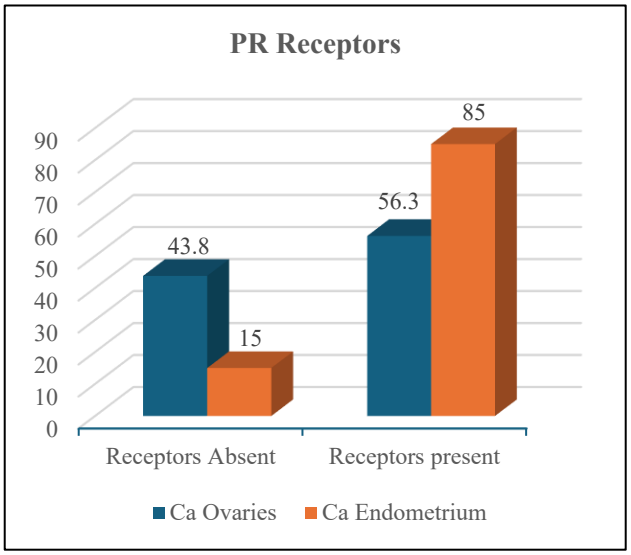

Figure 7: Distribution of participants according to the presence and absence of PR receptors.

P53 receptors were found mainly among participants with carcinoma of ovaries and endometrium. P53 receptors were found among 64.1% and 85% of participants with carcinoma of ovaries and endometrium respectively. It indicates that more percentage of participants with carcinoma of endometrium had P53 receptors when compared to participants with carcinoma of the ovaries (p=0.01).

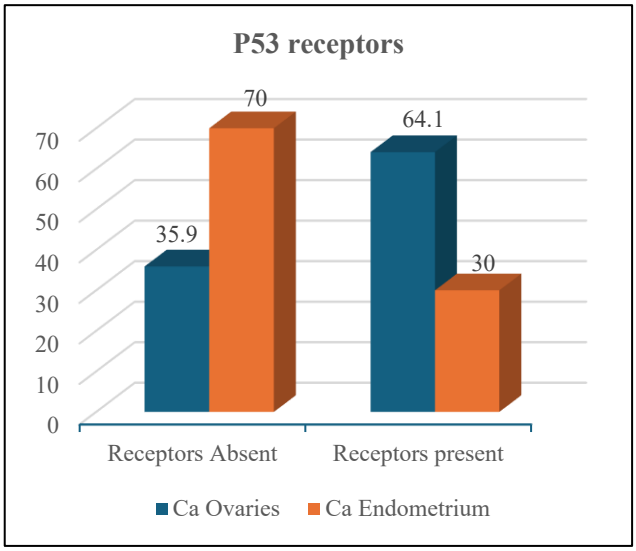

Figure 8: Distribution of participants according to the presence and absence of P53 receptors.

Participants with early-stage carcinoma had a longer recurrence time when compared to participants with advanced carcinoma (p=0.17). It was found that among those who died, about 80% had carcinoma of the ovaries and 20% had carcinoma of the cervix. In addition, among those who were alive, 62.9% had carcinoma of the ovaries, 22.5% had carcinoma of the endometrium, 9% of participants had carcinoma of the cervix, 4.5% had sarcoma, and 1.1% had carcinoma of the vagina (p=0.36).

DISCUSSION

Hormone receptors are broadly expressed across gynecological malignancies. While extensive evidence implicates ARs in the proliferation and progression of various tumor types, their precise role in malignancies beyond prostate cancer remains inadequately characterized.[11] Herein we review the existing evidence on the prognostic and/or treatment predictive values of new and already reputable biomarkers in clinically well-annotated patient cohorts with respect to hormone receptors.

Cervical cancer

An upstream glucocorticoid/progesterone response element has been identified in the regulatory region of the common E7/E6 promoter in human papillomavirus (HPV). Progesterone has been shown to enhance the transformative potential of viral DNA, thereby facilitating oncogenic progression.[13-15]

The most common histological subtype of cervical cancer is squamous cell carcinoma. Papillomavirus-associated lesions tend to worsen with the use of oral progestin-based contraceptives and during pregnancy, periods characterized by elevated progesterone levels. Estrogen enhances the expression of PRs, thereby facilitating HPV activation and promoting the expression of the viral E6 and E7 oncogenes.[16,17] PR poses to be a therapeutic target for cervical cancer, similar to its role in endometrial cancer. While 20-40% of human cervical malignancies show PR positivity, in a mouse model PR was present in all cervical malignancies.[18,19] While selective ER modulators (SERMs) have been demonstrated to suppress tumour growth in cervical cancer caused by HPV in mice models, estrogen and o ERα are essential in the pathogenesis of cervical cancer. Experimental data from HPV transgenic mouse models indicate that estrogens promote cervical tumorigenesis, whereas progesterone exhibits a protective, inhibitory effect.[20]

One study found AR expression in all 30 cases of normal cervical epithelium, all cases of low-grade CIN, 63% (i. e 19 out of 23) of high-grade CIN, and 23% of invasive squamous cell carcinoma. AR expression loss is frequently observed is common during malignant cervical transformation according to studies. However, presence of AR is of clinical importance in a significant subset of gynaecologic cancers.[11]

Uterine cancer

The prognostic significance of estrogen and PRs in endometrial cancer is well established, with higher levels of ER and PR expression correlating with improved overall survival (OS) and progression-free survival (PFS).[21] Historically, endocrine therapy was the standard frontline treatment for endometrial cancer, with therapeutic efficacy largely dependent on ER and PR status.[22] Yang et al identified AR positivity as a strong predictor of endometrial cancer risk. In a study by Ito et al AR were present in 88.6% of endometrial carcinomas, while Brys et al reported AR expression in 8% (1 out of 12) of normal endometrial tissue compared to 16% (4 out of 25) of endometrial cancers. The observed variability in AR expression may be attributed to inconsistencies in methodological approaches.[11] Current evidence suggests that AR loss is associated with reduced disease-specific survival, both in the general patient population and among those with early-stage disease (FIGO stages I/II).[23] Although the role of AR in endometrial cancer remains less clearly defined, androgens-particularly testosterone and DHT may represent promising targets for future therapeutic strategies

Uterine sarcoma

Hormonal receptors have significant prognostic value: particularly in patients treated with hormonal therapy and hormonal receptor expression has shown to correlate with better OS and PFS.[24] In a review of 65 uterine sarcoma cases, no AR was found. However, Moinfar et al found AR positivity in 45% (9/20) of malignant endometrial stromal neoplasms. According to Leitao et al 32% (6/19) of benign uterine leiomyomas and 40% (10/25) of uterine leiomyosarcomas expressed AR, indicating that benign leiomyomas respond to hormone treatment. Although AR was not substantially linked to an increase in OS after adjusting for stage, AR positivity was linked to decreased risk of recurrence.[26]

Vaginal cancer and vulvar carcinomas

Vaginal cancers may arise through both HPV-dependent and HPV-independent pathways. The progression of vaginal cancer involves several histological stages, including vaginal intraepithelial neoplasia (VAIN), carcinoma in situ, microinvasive carcinoma, and invasive cancer, arranged from least to most malignant.[16,27] Although HPV types 16 and 18 play a role in cervical carcinogenesis, whereas TP53 gene mutation is the main cause of vulvar cancer. Conversely, the development of vaginal cancer appears to be associated with both.[28] Tuboendometrial type of atypical cervical ectropion and atypical vaginal adenosis may be precursor lesions to clear cell carcinoma of the cervix and vagina, according to Robboy et al study on estrogen-induced maturation arrest of the Müllerian ducts.[29]

Postmenopausal women using vaginal oestrogen have a risk profile for major cancers and vascular events similar to non-users.[30] Vulvar cancers are infrequently hormone-dependent neoplasms.[31] In addition to prolactinoma, renal, pancreatic, and thyroid cancers, hormone replacement treatment (HRT) has no effect on type II endometrial cancer, uterine carcinosarcoma, adenosarcoma, some ovarian cancers, and squamous cell carcinomas of the cervix, vagina, and vulva.[32] Nonetheless, tissue-selective estrogen complexes incorporating bazedoxifene with conjugated estrogens (BZA/CE) may represent a viable menopausal therapy for postmenopausal women.[33]

Ovarian cancer

Epithelial ovarian cancer (EOC) ranks as the sixth most common malignancy and the seventh leading cause of cancer-related mortality among women globally. It is also recognized as the eighth most lethal malignancy in women worldwide. Despite initial treatment-typically involving staging or cytoreductive surgery followed by platinum-based adjuvant chemotherapy-approximately 50% of patients experience relapse within 16 months. Consequently, there is an urgent need for effective clinicopathological biomarkers to guide prognosis and treatment decisions.[34-36]

In advanced serous ovarian and endometrioid carcinomas, ER and PR expression have been recognized as predictive markers. In a large trial involving 2,933 women with advanced epithelial ovarian cancer, PR expression was strongly associated with improved disease-specific survival in both endometrioid carcinoma (log-rank $p<0.0001$) and high-grade serous carcinoma (log-rank $p=0.0006$).[36]

Jones et al also looked at ovarian tumours with steroid cells and found that 64% (9 out of 24) of the samples had AR positivity. Additionally, 18% (28 in 154) of instances with epithelial ovarian cancer had high AR expression.[37]

Steroid hormones are implicated in the development and progression of ovarian cancer.[38] Clinical parameters such as advanced FIGO stage, post-surgical disease status, and tumor histology significantly influence both progression-free survival (PFS) and OS.[35-37,39]

AR expression, which is known to be associated with favorable outcomes in breast cancer, shows a similar trend in ovarian cancer.[40] Androgen deprivation therapy (ADT) aims to lower circulating androgens-mainly testosterone and dihydrotestosterone (DHT)-which are regulated by adrenocorticotropic hormone (ACTH).[34,37] While adrenal and peripheral androgen synthesis plays a minimal role in healthy males, these sources become more relevant during androgen suppression. Peripheral tissues, including skin, fat, liver, and the urogenital tract, contribute significantly to androgen production.[35,36,40]

Evidence suggests that initiating anti-androgen therapy early in the course of ovarian cancer may be more effective, as AR expression diminishes with chemotherapy exposure.[34] This reduction in AR levels may underlie the limited response to hormonal therapy observed in heavily pretreated patients. Therefore, early intervention with anti-androgen agents could enhance therapeutic outcomes.[41] Although plasma androgen concentrations have not been clearly associated with AR status, physiological androgen levels remain low in both pre- and postmenopausal women.[35]

A pivotal study by Feng et al (Fudan university, 2016) reported discordant hormone receptor status between primary and recurrent ovarian tumors for the first time. Notably, the poorly prognostic PR-/ER+/AR- subgroup increased in frequency in recurrent high-grade serous carcinoma (HGSC). While hormone therapies such as tamoxifen and aromatase inhibitors are used as salvage treatments in recurrent disease, clinical outcomes have generally been unsatisfactory.[34,38] However, ER-positive patients in a phase II trial of letrozole demonstrated improved response rates.[35] These findings emphasize the importance of reassessing hormone receptor status in recurrent disease to tailor endocrine therapies more effectively.[36]

CONCLUSION

The results presented in this study contribute to the incidence and importance of hormone receptors in gynecological cancers; especially the AR. Nevertheless, these results have to be cautiously interpreted as this is a prospective study of short duration with is the limitation, single-center design, possibly introducing some degree of bias. There exists the potential to further this current study by adding a prospective large sample cohort study component to it, by active long follow-up. Along with the prognostic value of presence or absence of hormone receptors, hormone therapy is a promising therapeutic option for certain recurrent gynaecological cancers. AR based combination therapies have to be explored as potential treatment for specific subtype of gynecological cancers. AR antagonists-enzalutamide and bicalutamide have a good tolerability and safety in AR+ ovarian cancer. Additional research; in particular, more multicenter, prospective, well-designed, and randomized clinical trials are warranted to define the role of hormone receptors in treatment of gynaecological cancers.

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Ethical approval: The study was approved by the Institutional Ethics Committee

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