American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2025; 26: e946296
DOI: 10.12659/AJCR.946296

Received: 2024.08.23
Accepted: 2024.11.06
Available online: 2024.11.26
Published: 2025.01.03

Anti-Estrogen Therapy Achieves Complete Remission and Stability in Recurrent Cervical Cancer: A Case Study

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1,2 Mun-Kun Hong
BEF 1 Ching-Hsiang Chiang
BDE 3 Chiu-Hsuan Cheng
ACDEF 1,2,4 Tang-Yuan Chu

1 Department of Obstetrics and Gynecology, Hualien Tzu Chi Hospital, Buddhist Tzu Chi Medical Foundation, Hualien, Taiwan, R.O.C.
2 Institute of Medical Sciences, Tzu Chi University, Hualien, Taiwan, R.O.C.
3 Department of Anatomic Pathology, Hualien Tzu Chi Hospital, Hualien, Taiwan, R.O.C
4 Center for Prevention and Treatment of Gynecological Cancers, Department of Research, Hualien Tzu Chi Hospital, Hualien, Taiwan, R.O.C.

Corresponding Author: Tang-Yuan Chu, e-mail: jeff06038@gmail.com
Financial support: None declared
Conflict of interest: None declared

Patient: Female, 84-year-old
Final Diagnosis: SCC of cervix
Symptoms: Vaginal bleeding
Clinical Procedure: Biopsy
Specialty: Obstetrics and Gynecology • Oncology

Objective: Unusual or unexpected effect of treatment

Background: Studies using transgenic mouse models have demonstrated that estrogen is necessary for the development of cervical cancer, particularly in tissues responsive to estrogen. Estrogen also protects cervical cancer cells from apoptosis, suggesting its role in the survival and persistence of cancer cells.

Case Report: An 84-year-old woman with diabetes mellitus, hypertension, and stage III chronic renal failure was diagnosed with cervical squamous cell carcinoma, FIGO stage IB2. She underwent complete concurrent chemoradiotherapy, but central recurrence was found 9 months later. However, instead of salvage chemotherapy, substitutionary anti-estrogens were given due to her poor medical condition and advanced age. Complete remission was noted after tamoxifen therapy. Since the cervical cancer relapsed again 40 months after tamoxifen use, the anti-estrogen therapy was shifted to letrozole. The SCC-Ag level decreased dramatically after letrozole therapy, and disease stability was achieved until 29 months afterward. After 5 years and 9 months of anti-estrogen use only, the patient died due to noncancer-related pneumonia and heart failure.

Conclusions: This report demonstrates the tumor-stabilizing and therapeutic effect of anti-estrogens in the treatment of squamous cervical carcinoma. Further clinical trials are warranted to evaluate the efficacy of anti-estrogen therapy in cervical cancer patients.

Keywords: Estrogen Antagonists • Estrogen Receptor Modulators • Letrozole • Tamoxifen • Uterine Cervical Neoplasms

Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/946296

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Introduction

Human papilloma virus (HPV) infection is a necessary factor for cervical carcinogenesis. Recently, estrogen and estrogen receptor (ER) were found to be crucial in the development of invasive cervical cancer by HPV [1,2]. Epidemiological studies reported that in non-screened populations, the incidence of cervical cancer in women increases with age and plateaus during the perimenopausal period [3]; therefore, cervical cancer was suspected to be related to sex hormones. In line with this, long-term hormone exposure, which occurs alongside high parity and long-term oral contraceptive use, was found to be an independent risk factor for cervical cancer [3,4] and cervical intraepithelial neoplasia (CIN) [5]. A clinical study evaluated the effects of hormonal therapy (ie, progesterone, estrogen, or both in combination) was administered to 804 cases of cervical intraepithelial neoplasia grade 3 (CIN3/CIS) and 261 cases of invasive cervical cancer (ICC) among peri- and postmenopausal women. Hormone therapy was associated with a significantly lower risk of ICC, but estrogen alone was linked to a higher incidence of CIN3/CIS [6]. In contrast, in a population-based follow-up study, long-term anti-estrogen use was associated with a lower risk of cervical neoplasm in breast cancer patients [5].

Estrogen and ERα expressed in the stroma were found to be essential for carcinogenesis in a transgenic mouse model of HPV16 E6/E7 expression in basal cells [2]. Furthermore, the precancerous and cancer lesions of these mice were efficiently cleared after therapy with an ER antagonist or selective ER modulator [1,7]. An in vitro study found that tamoxifen suppressed the proliferation of 3 cervical carcinoma cell lines. At doses greater than 5 μM, tamoxifen can cause progressive cell death and cytotoxicity [8].

The results from these previous epidemiological and preclinical studies highlight the role of estrogen in the development of cervical cancer, suggesting that anti-estrogens may be effective in treating cervical neoplasia. However, to our knowledge, there are currently no ongoing clinical trials, and the literature reports only 1 small study on the use of tamoxifen for treating non-squamous cervical cancers, which showed modest efficacy [9]. Consequently, the effectiveness of anti-estrogens in treating squamous cell cervical cancer remains uncertain.

In this report, we present a case of recurrent cervical squamous cancer that achieved complete remission and remained stable over 5 years following anti-estrogens use. This case study demonstrates the potential tumoristatic and therapeutic effects of anti-estrogen agents for treating squamous cervical cancer.

Case Report

An 84-year-old woman with an overweight body mass index (BMI; 27.3 kg/m^2), diabetes mellitus, hypertension, and stage III chronic kidney disease had had persistent and aggravated vaginal bleeding for about 1 year before being diagnosed with cervical cancer, FIGO stage IB2. Upon referral to a gynecology oncologist, pelvic and rectal examination revealed an endophytic cervical tumor with easy contact bleeding, and the parametria were freely movable. Transvaginal ultrasound revealed a retroverted uterus, measuring 6.1×4.6 cm with minimal fluid accumulation in the uterine cavity and a 4×3 cm heterogeneous cervical tumor. Cervical biopsy revealed papillary squamous cell carcinoma (**Figure 1**). The HPV DNA test was positive for genotypes 16, 18, and 35. CT scan of the abdomen and pelvis revealed an abnormal soft tissue mass measuring 3.33×4.12 cm in the cervix. Further laboratory tests revealed an elevated SCC antigen (SCC-Ag) impaired renal function (blood urea nitrogen: 28 mg/dL; creatinine: 1.2 mg/dL), and poorly controlled hyperglycemia (blood glucose: 250 mg/dL; HbA1c: 8.0%). Due to her advanced age, bulky cervical carcinoma, and unfavorable medical conditions, concurrent chemoradiotherapy (CCRT)

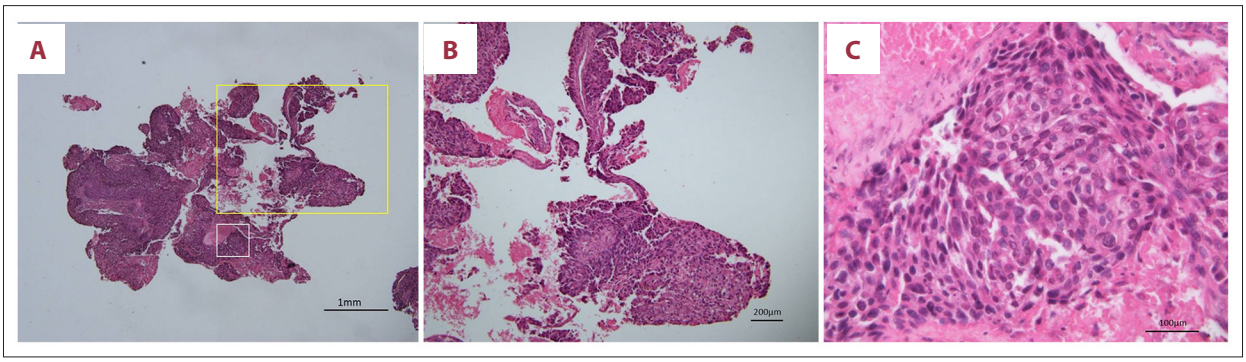

Figure 1. (**A**) Fragments of tumor with papillary structures are seen under microscope (H&E stain, magnification 40×). (**B**) Infiltration irregular nests with tumor necrosis (yellowish inset in **A**, magnification 100×). (**C**) Non-keratinizing squamous cell carcinoma characterized by polygonal to slightly elongated cells with eosinophilic cytoplasm without keratin pearl is presented (white inset in **A**, magnification 400×).

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

Table 1. Timeline of the patient's symptoms, diagnosis, treatment, and follow-up.

Date	Month since first recurrence	Antiestrogen	SCC-Ag	Acessment
Apr. 09	-12		1,2	Vaginal bleeding, diagnosed as SCC by cervical biopsy
May 09	-11		1,3	
Jun. 09	-9		1,4	CCRT
Jul. 09	-8		0,8	
Oct. 09	-5		0,4	SCC-Ag came to nadir
Mar. 10	-1		0,3	23-Mar-10 First PET-CT indicated *first central recurrence of SCC (SUVmax: early: 4.59, delayed: 6.34).
May. 10	0		1,1	Cervical biopsies revealed necrotic debris
Aug. 10	6	Tamoxifen	1,4	
Nov. 10	9		0,9	
Oct. 11	20		1	
Feb. 12	24		1,7	1-Jun-12 2nd PET-CT
May 12	27		1,4	
Dec. 12	34		1,5	
Mar. 13	37		0,9	
Jun. 13	40		3,5	Significant elevation of SCC-Ag indicating *2nd recurence
Sep. 13	43	Change tamoxifen to Letrozole	5,5	
Oct. 13	44		6,9	27-Nov-13 3rd PET-CT
Jul. 14	53		1,6	
Oct. 14	56		1,7	
Dec. 14	58		2,1	
Jun. 15	69		3,1	Died of noncancer-related pneumonia and acute decompensated heart failure

SCC – squamous cervical carcinoma; CCRT – concurrent chemoradiotherapy; PET-CT – positron emission tomography-computed tomography. The exact date was shaded (left only month and year) to prevent identifacation of personal data.

was arranged. The patient received 6 doses of weekly carboplatin (AUC=2.5-3) alongside external beam radiation (5040 cGy) and intracavitary radiation (3000 cGy). The SCC-Ag level reached its nadir at 0.3 ng/ml after CCRT. She was regularly followed up every 3 months thereafter (**Table 1**).

The disease remained stable until 9 months after CCRT. The SCC antigen (SCC-Ag) at cervical cancer diagnosis 12 months ago was 1.2 ng/m (**Figure 2**, point A); it reached a nadir of 0.3 ng/ml after CCRT, but gradually rose to 1.4 ng/ml (**Figure 2**, point B). A grossly fibro-necrotic lesion at the tip of posterior lip of cervix was noted on pelvic examination. Abdominal CT revealed enlargement of the cervix with an abnormal irregular soft tissue mass measuring 2.2×2.5 cm. Fluorodeoxyglucose positron emission CT (FDG-PET CT) showed a glucose hypermetabolic lesion at the cervix (**Figure 3A**) (early and late maximum standardized uptake value [SUVmax] of 4.59 and 6.34), indicating central recurrence (**Figure 4**). Because the patient was elderly, had already received full-dose radiotherapy, and had comorbidities such as poorly controlled diabetes mellitus and impaired renal (creatinine: 1.3 mg/dL, creatinine clearance: 27.4 ml/min) and cardiac function, we opted for anti-estrogen

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

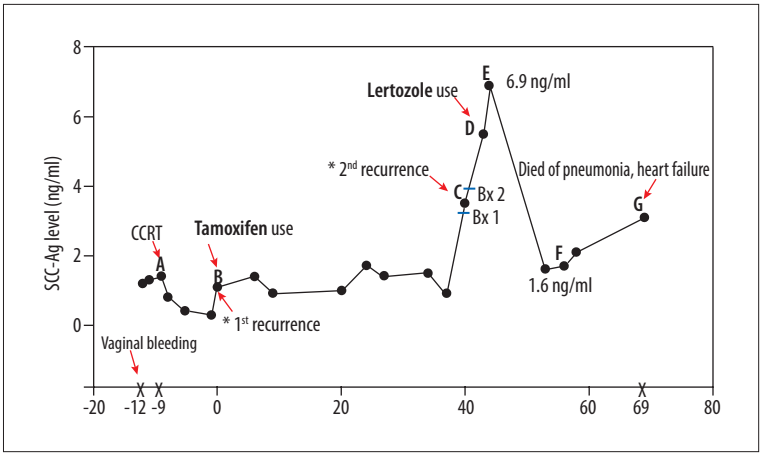

Figure 2. Changes in SCC-Ag levels, and clinical course before and after anti-estrogens use. **A**) Cervical cancer was first diagnosed 9 months before anti-estrogen use, with an SCC-Ag level of 1.2 ng/ml; **B**) After FDG-PET CT revealed central recurrence, treatment with tamoxifen was started. **C**) The SCC-Ag level increased to 3.5 ng/ml after 43 months of tamoxifen use. **D**) Three weeks later, the SCC-Ag level increased to 5.5 ng/ml, and the anti-estrogen therapy was shifted to letrozole. **E**) The SCC-Ag level peaked at 6.9 ng/ml, then dropped gradually to its nadir of 1.6 ng/ml in the following 11 months. **F**) The SCC-Ag level rebounded to 2.1 ng/ml after 15 months of letrozole use. **G**) The patient died 69 months after starting anti-estrogen treatment due to noncancer-related pneumonia and heart failure. CCRT – concurrent chemoradiotherapy; Bx – cervical biopsy.

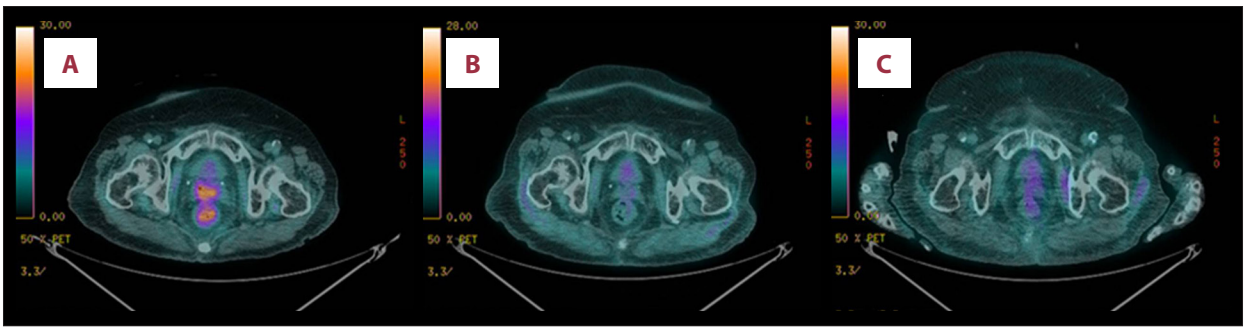

Figure 3. (**A**) FDG-PET/CT revealed recurrence of cervical carcinoma (9 months after primary CCRT). A glucose hypermetabolic lesion is located at the cervix. (**B**) The glucose hypermetabolic lesion completely disappeared 28 months after tamoxifen use. (**C**) No evidence of recurrence of cervical carcinoma on subsequent follow-up FDG-PET CT done 40 months after tamoxifen use.

therapy with tamoxifen (Nolvadex®, AstraZeneca, Cheshire, United Kingdom) at a dose of 10 mg twice daily rather than salvage chemotherapy. In the following month, the cervical lesion significantly shrank in size and persistence of some necrotic discharge and foul smell were noted on pelvic examination. During the next 2 years, the SCC-Ag level declined to 0.9 ng/ml and slightly fluctuated between 0.9 and 1.7 ng/ml (**Figure 2**, points B to C), and grossly, the cervical tumor remained unchanged. Follow-up FDG-PET CT studies done at 28 months after tamoxifen administration revealed complete remission (or complete response according to the RECIST 1.1 criteria) of disease (**Figure 3B**). Notably, at 43 months after tamoxifen use, the SCC-Ag levels significantly rose to 3.5 ng/ml (**Figure 2**, points C) and further increased to 5.5 ng/ml after 3 weeks (**Figure 2**, point D), indicating a second recurrence. Two cervical biopsies were done after the second recurrence, but pathology only reported necrotic debris and no tumor was seen. Owing to the patient's advanced age and unfavorable renal and cardiac function, she and her family refused salvage chemotherapy. Therefore, the anti-estrogen therapy was shifted to the aromatase inhibitor (AI) letrozole (Femara®; Novartis, Basel, Switzerland) at a dose of 2.5 mg once daily. The SCC-Ag levels peaked at 6.9 ng/ml after 4 weeks (**Figure 2**, point E), then gradually dropped to 1.6 ng/ml in the next 11 months. The patient had no evidence of relapse based on pelvic examination (ie, no gross cervical tumor) and SCC-Ag levels. After 24 months of letrozole use, the SCC-Ag level rose to 3.1 ng/ml (**Figure 2**, point F), but no gross lesion was found on pelvic examination and the subsequent follow-up FDG-PET CT (**Figure 3C**). Throughout the treatment course there was no drug-related side effect observed. After her first CCRT, she had received Pap smear test every 4-6 months on

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

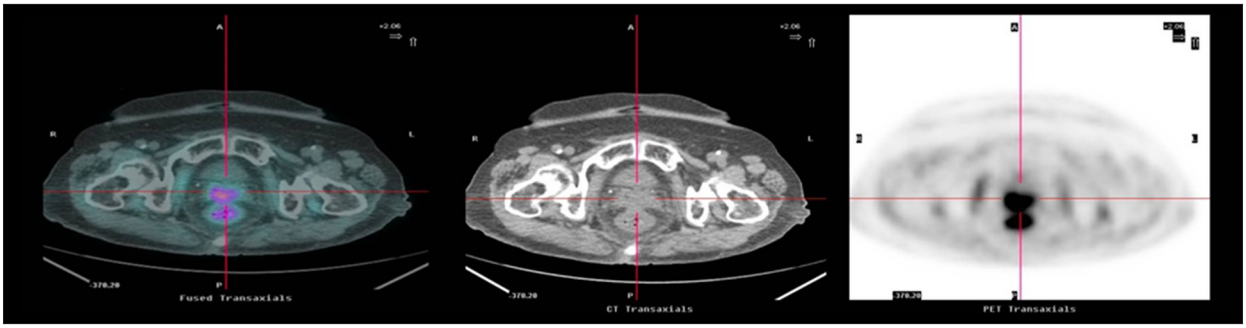

Figure 4. FDG-PET CT showing an early and late maximum standardized uptake value (SUVmax) of 4.59 and 6.34, respectively. These findings indicate central recurrent malignancy.

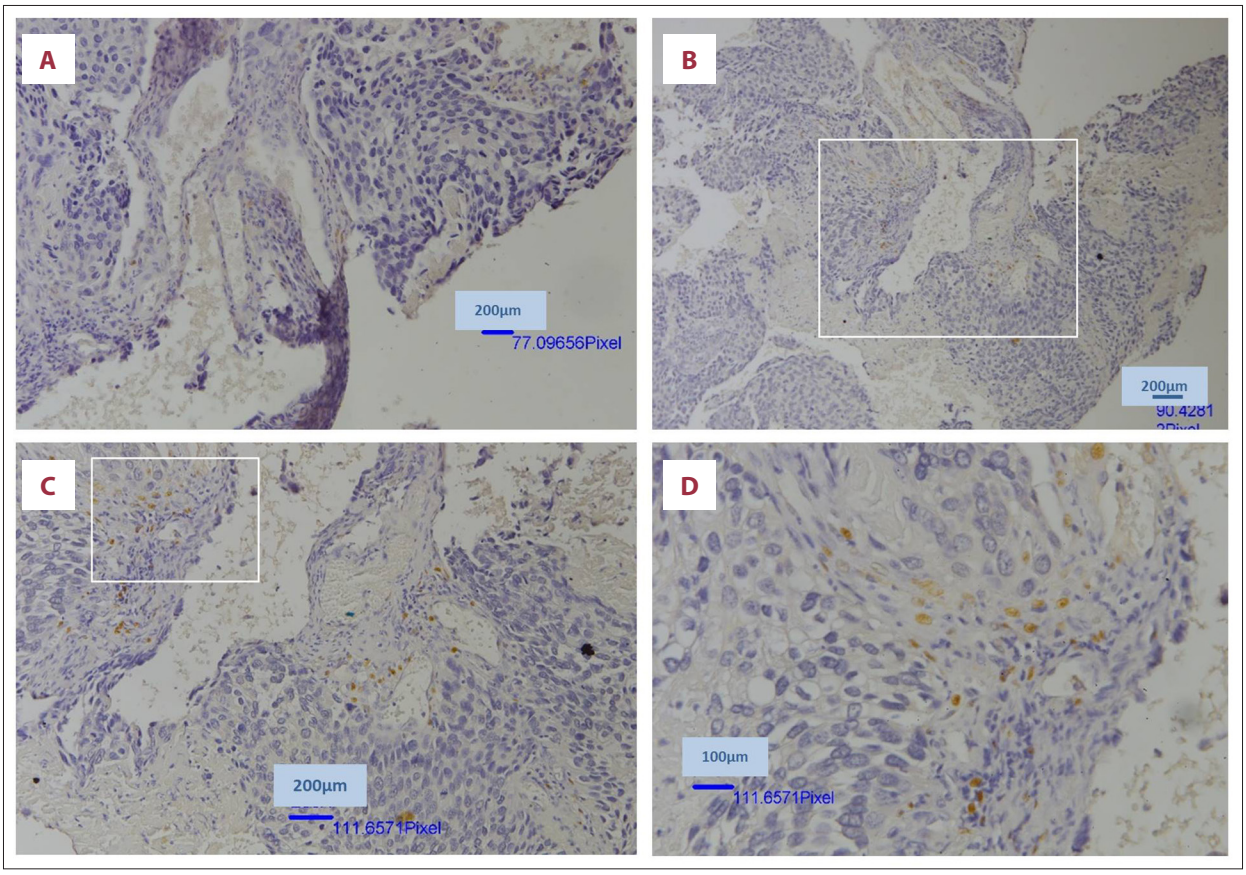

Figure 5. Immunohistochemistry of ERα and PRB in the primary tumor before treatment showed no expression of ERα (**A**) and (**B**) low expression of PRB in the stroma of tumor. 100× magnification. (**C**) Low expression of PRB (white inset in **B**), 200× magnification. (**D**) Low expression of PRB (white inset in **C**), 400× magnification.

follow-up (a total of 11 tests) and no atypical or cancer cells were found, only inflammatory cells. The patient died of non-cancer-related pneumonia and acute decompensated heart failure at 91 years old, which was 5 years and 9 months after initiating treatment with tamoxifen. Regarding sex hormone receptor expression in the tumor obtained before CCRT treatment, the tumor was negative for ERα, and weakly expressed progesterone receptor isoform B(PRB) in the stromal cells but not in the carcinoma cells (**Figure 5**).

Discussion

Chemoradiotherapy or neoadjuvant chemotherapy is indicated for advanced-stage or recurrent cervical cancer [10]. Our patient's advanced age and poor renal and cardiac made her unsuitable for chemotherapy. Instead, she was started on oral anti-estrogens, starting with tamoxifen followed by letrozole. She achieved complete remission and maintained stable disease for 69 months. Notably, a relapse occurred 40 months

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

after initiating tamoxifen treatment, but this was effectively controlled with subsequent letrozole therapy. This case report demonstrates the tumoristatic and/or therapeutic effects of anti-estrogens for treating cervical SCC.

SCC-Ag is a widely used serum tumor marker for cervical SCC. A change in SCC-Ag with an optimal cutoff of 0.95 ng/mL or persistently elevated SCC-Ag levels after treatment indicate recurrent disease [11,12]. In this reported case, the SCC-Ag rose from 0.3 to 1.4 ng/mL at the first recurrence and rose from 0.9 to 6.9 ng/mL at the second recurrence) (**Figure 2**). By contrast, FDG-PET CT is useful tool to detect the recurrence of cervical cancer [2]. The optimal standardized uptake values (SUVmax) cutoff value of 4.0 for posttreatment carcinoma yields a sensitivity of 92% and specificity of 94% for predicting the presence of residual or recurrent cervical tumors [13]. In the present case, FDG-PET CT revealed an early and late SUVmax of 4.59 and 6.34 (**Figure 4**), respectively. The data presented above provide solid evidence of recurrent malignancy for this patient, and they objectively demonstrate the effect of anti-estrogen. According to the RECIST 1.1 criteria for response of solid tumors to treatment, the FDG-PET CT image revealed a complete response (ie, disappearance of the only target lesion at the cervix) (**Figure 3B, 3C**).

Although robust preclinical evidence supporting the essential role of E2/ER in cervical carcinogenesis and the efficacy of anti-estrogen therapy in HPV-induced cervical neoplasia mouse models, there is a notable lack of clinical studies using anti-estrogen to treat cervical cancer or cervical intraepithelial neoplasia (CIN3). The only phase 2 clinical trial, conducted by Bigler, found that administering 10 mg of the anti-estrogen medication tamoxifen twice daily to 34 patients (median age 49 years) with non-squamous cell carcinoma resulted in an objective response rate of 11.1%, including 1 complete response and 2 partial responses [9]. The trial focused on non-squamous carcinoma in a group of younger patients, and therefore is not relevant to our patient, who was 84-year-old old with recurrent cervical squamous cell carcinoma. Different histological types of cervical cancer might respond differently to anti-estrogens, potentially offering new hope for treatment options in cervical cancer. No other clinical studies regarding the use of anti-estrogen in cervical cancer can be found in the literature aside from the Bigler study.

Theoretically, tamoxifen, a selective estrogen receptor modulator (SERM), exerts functions by competing with estrogens for binding to ER. While estrogen receptor (ER) expression is rarely found in the carcinoma component of cervical cancer, it was present in the stroma of 52% of 200 studied cases of cervical squamous cell carcinoma [14]. Despite the lack of ER expression in tumors during the initial diagnosis, the patients favorably responded to tamoxifen when the disease recurred after CCRT. Thus, the ER/PR status of the tumors may have changed after chemotherapy. A clinical study investigated the effect of neoadjuvant chemotherapy on the expression of hormone receptors and Ki67 in 520 cases of female breast cancer. The ER, PR, and Ki67 status changed from negative to positive in 7.7%, 13.6%, and 23.7% of the patients, respectively [15]. In our patient, we are unsure if the ER status became positive, as it was an endophytic and necrotic tumor, and 2 cervical biopsies were done after recurrence, both revealing only necrotic debris and no tumor. Thus, we were unable to ascertain the ER status of the tumor before tamoxifen use. Given that estrogen may exert effects independently of ER and that aromatase inhibitors can effectively mitigate estrogenic effects on tumorigenesis, the role of ER as a guide for hormone therapy warrants further investigation. In the future, when a patient is a poor candidate for salvage chemotherapy and tamoxifen is considered, it is recommended to re-evaluate the ER status of the tumor before tamoxifen use because patients with ER-positive cervical SCC are good candidates for tamoxifen.

Letrozole is an aromatase inhibitor (AI), in which aromatase is a critical enzyme responsible for converting androgen in estrogen biosynthesis. About 35% of cervical carcinomas exhibit aromatase overexpression, and the expression was associated with increased expression of estrogen receptors [15]. Levels of aromatase and its enzyme activity were increased in cervical carcinomas as compared to normal tissue, and were associated with advanced stage [16]. When the aromatase gene is overexpressed in HPV-positive cervical cancer cells, it increases transformation phenotypes, including cellular proliferation, anchorage-independent growth, and ER expression and activity [16]. This may explain why AI seemed to be more effective than tamoxifen in our patient with advanced-stage cervical cancer.

In the presented case, AI worked better than tamoxifen in controlling the disease. This is in line with the use of anti-estrogens in preventing cervical neoplasia and recurrence of breast cancer. Use of AI consistently showed a better performance in lowering the risk of cervical neoplasia compared to tamoxifen use [5]. In preventing the recurrence of breast cancer after primary therapy, AI outperforms SERMs [5]. Likely, non-genomic activities of estrogen, such as binding with G-protein coupled receptor (GPCR), also contributed to the malignancy of cervical cancer.

The expression of PR in cervical cancer also significantly declines with age [17], such as in the present case. Stromal PR has been identified as an independent favorable prognostic marker of SCC [17]. Our patient exhibited favorable outcomes of complete remission and maintained disease stability for years, mainly due to the tumor-stabilizing and therapeutic effects of the anti-estrogens. Accordingly, the expression of PR

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]

could have also positively contributed to the good prognosis. Based on the clinical course and outcomes of the present case, anti-estrogens demonstrated tumor-stabilizing and therapeutic effects in cervical squamous cell carcinomas and should be considered in certain cases.

Conclusions

In this reported case, recurrent cervical cancer was effectively controlled with tamoxifen for 40 months. Subsequently, a rapid escalation of relapse was successfully suppressed using an aromatase inhibitor. This case highlights the tumor-stabilizing and potential therapeutic effects of anti-estrogens in squamous cervical carcinoma. These treatments may be empirically used in late-stage, recurrent cervical cancer patients, especially given the lack of effective alternatives and the minimal adverse effects associated with these drugs. Further clinical studies are warranted to evaluate the indications, efficacy, and safety of the anti-estrogens, as well as the role of estrogen receptor immunohistochemistry as a guide to efficacy.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Brake T, Lambert PF. Estrogen contributes to the onset, persistence, and malignant progression of cervical cancer in a human papillomavirus-transgenic mouse model. Proc Natl Acad Sci USA. 2005;102(7):2490-95
2. Chung SH, Wiedmeyer K, Shai A, et al. Requirement for estrogen receptor alpha in a mouse model for human papillomavirus-associated cervical cancer. Cancer Res. 2008; 68(23):9928-34
3. International Collaboration of Epidemiological Studies of Cervical Cancer. Cervical carcinoma and reproductive factors: collaborative reanalysis of individual data on 16,563 women with cervical carcinoma and 33,542 women without cervical carcinoma from 25 epidemiological studies. Int J Cancer. 2006;119(5):1108-24
4. International Collaboration of Epidemiological Studies of Cervical Cancer, Appleby P, Beral V, et al. Cervical cancer and hormonal contraceptives: collaborative reanalysis of individual data for 16,573 women with cervical cancer and 35,509 women without cervical cancer from 24 epidemiological studies. Lancet. 2007;370(9599):1609-21
5. Hsieh CJ, Hong MK, Chen PC, et al. Antiestrogen use reduces risk of cervical neoplasia in breast cancer patients: A population-based study. Oncotarget. 2017;8(17):29361-69
6. Roura E, Travier N, Waterboer T, et al. The influence of hormonal factors on the risk of developing cervical cancer and pre-cancer: Results from the EPIC cohort. PLoS One. 2016;11(1):e0147029
7. Chung SH, Lambert PF. Prevention and treatment of cervical cancer in mice using estrogen receptor antagonists. Proc Natl Acad Sci USA. 2009;106(46):19467-72
8. Grenman S, Shapira A, Carey TE. In vitro response of cervical cancer cell lines CaSki, HeLa, and ME-180 to the antiestrogen tamoxifen. Gynecol Oncol. 1988;30(2):228-38
9. Bigler LR, Tate Thigpen J, Blessing JA, et al; Gynecologic Oncology Group. Evaluation of tamoxifen in persistent or recurrent nonsquamous cell carcinoma of the cervix: A Gynecologic Oncology Group study. Int J Gynecol Cancer. 2004;14(5):871-74
10. Tao W, Wang W, Zhai J, Guo L. Efficacy analysis of neoadjuvant versus adjuvant cisplatin-paclitaxel regimens for initial treatment of FIGO stages IB3 and IIA2 cervical cancer. Med Sci Monit. 2023;29:e940545
11. Ryu HK, Baek JS, Kang WD, Kim SM. The prognostic value of squamous cell carcinoma antigen for predicting tumor recurrence in cervical squamous cell carcinoma patients. Obstet Gynecol Sci. 2015;58(5):368-76
12. Fu J, Wang W, Wang Y, et al. The role of squamous cell carcinoma antigen (SCC Ag) in outcome prediction after concurrent chemoradiotherapy and treatment decisions for patients with cervical cancer. Radiat Oncol. 2019;14(1):146
13. Choi J, Kim HJ, Jeong YH, et al. The role of (18) F-FDG PET/CT in assessing therapy response in cervix cancer after concurrent chemoradiation therapy. Nucl Med Mol Imaging. 2014;48(2):130-36
14. Kwasniewska A, Postawski K, Gozdzicka-Jozefiak A, et al. Estrogen and progesterone receptor expression in HPV-positive and HPV-negative cervical carcinomas. Oncol Rep. 2011;26(1):153-60
15. Wu YT, Li X, Lu LJ, et al. Effect of neoadjuvant chemotherapy on the expression of hormone receptors and Ki-67 in Chinese breast cancer patients: A retrospective study of 525 patients. J Biomed Res. 2017;32(3):191-97
16. Nair HB, Luthra R, Kirma N, et al. Induction of aromatase expression in cervical carcinomas: Effects of endogenous estrogen on cervical cancer cell proliferation. Cancer Res. 2005;65(23):11164-73
17. Hong MK, Wang JH, Su CC, et al. Expression of estrogen and progesterone receptor in tumor stroma predicts favorable prognosis of cervical squamous cell carcinoma. Int J Gynecol Cancer. 2017;27(6):1247-55

Indexed in: [PMC] [PubMed] [Emerging Sources Citation Index (ESCI)] [Web of Science by Clarivate]