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Case Report

Giant Ovarian Endometrioma and Endometrial Cancer in a Postmenopausal Woman- A Case Report

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Abstract

Endometriosis is a disease of the reproductive years and is very rare after menopause. Endometrial cancer is more often seen in late reproductive years and after menopause. A coexistence of the two conditions in postmenopausal women is extremely rare. We report a case of giant endometrioma and incidentally detected endometrial cancer in a postmenopausal woman and discuss the aetiopathogenesis and management.

Keywords: Endometriosis; Endometrial cancer; Postmenopausal; Giant endometrioma.

Introduction

Endometriosis is believed to be a disease of the reproductive years and is extremely rare after menopause while endometrial cancer is more common in the late reproductive years and after menopause. The two conditions are known to thrive in estrogenic environment, however, a coexistence of the two conditions in the hypoestrogenic postmenopausal state is rare. The presence of these two conditions in postmenopausal women is a diagnostic and management dilemma for the clinician. A case of a giant endometrioma coexisting with an incidentally detected endometrial cancer in a postmenopausal woman is presented and discussed.

Case presentation

An eighty years old para 3 woman with a BMI of 30was referred by her GP to our Gynecology Clinic for vague, moderate and continuous lower abdominal pain of a day's duration. It was not associated with any nausea, vomiting, diarrhea, constipation, frequency or burning micturition. There was no history of postmenopausal bleeding or loss of appetite or weight. Her last cervical screening had been 10 years ago and was normal. There was no family history of breast, genital tract or gastrointestinal cancer.Her general physical examination was essentially normal. Abdominal examination revealed a nontender firm mass in the lower abdomen arising from the pelvis and reaching up to the umbilicus with restricted mobility.

She was investigated for the abdominal mass. She had a haemoglobin of 133 g/L, WBC count of 7.9×10 9 /L, platelets 304×10 9, haematocrit of 0.42 L/L, CA 125 16 U/ml, CA 19-9 of 16 U/ml, CEA of 2.8 mcg/L, alphafetoprotein level of 52.4 kU/L, HCG of 2.4 IU/L, Inhibin B was less than 10 ng/L and HE4 level was 55 pmol/L. Her ROMA score (postmenopausal) was 12.7%. The chest Xray was normal. Ultrasound pelvis suggested a normal uterus with a uniformly thin endometrium with bilateral atrophic ovaries. A large cystic lesion of 18 cm×11 cm×14 cm size with low level internal echoes, a thin single septum and no internal solid mural nodules was noted separate from the ovaries. The origin of this cyst was reportedly unclear on ultrasound. A computerized tomography scan of the abdomen and pelvisreported a large well-marginated parauterine cystic lesion of 110 mm×141 mm×168 mm size with no internal soft tissue nodularity anterior to the uterus and superior to the bladder.

An MRI scan of the abdomen and pelvis was also requested which showed a normal sized uterus with thin (3 mm) uniform endometrium and a homogenous myometrium. A cystic lesion of 14.4 cm×11.4 cm×17.2 cm cystic lesion with minimal thin internal septation, likely representing an ovarian cystadenoma (ORADS 2) was noted anterior to the uterus and superior to the urinary bladder. The mass had a rim of enhancing tissue at the left anterior aspect of the cyst and adjacent left ovarian tissue, suggestive of left ovarian origin while the right ovary was reportedly anormal. The patient was referred to a tertiary GynaecologyOncology Centre that deemed her fit to be managed at the local hospital. She underwent an uneventful total abdominal hysterectomy, bilateral salpingo-oophorectomy and omental biopsy. The specimen histopathology reported endometrioid adenocarcinoma of uterine corpus (3 mm×2 mm size) FIGO grade 1 with limited myometrial invasion (3 mm out of total myometrial depth of 9 mm) and the left cystic ovarian tumor as a haemorrhagic cyst without

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preserved lining. The endometrial tumor, with no lymphovascular invasion, was limited to the uterine corpus and the tubes and opposite ovary were also histologically normal.The peritoneal cytology also returned normal. Immunohistochemistry showed ER 2-3+ and PR 3+ in over 90% tumor cells and p53 wildtype. Additionally, extensive superficial adenomyosis and background endometriosis (along with the left ovarian haemorrhagic cyst)was reported to be consistent with burnt out endometriosis. A molecular analysis was not performed. The patient had an uneventful postoperative course and was referred to the GynaecologicOncology Centre for further management.

Discussion

Endometrial cancer is one of the most common cancers affecting women worldwide and there is a1 in 44 risk of getting the disease by 85 years of age [1]. Endometrioid type (type 1), the most common subtype accounts for 80-90% of all endometrial cancers [2].These tumors are believed to progress from simple to complex endometrial hyperplasia, which then develops into endometrial intraepithelial neoplasia and finally to invasive endometrial carcinoma [3]. Endometriosis affects 10% women in their reproductive years and 2-5% of postmenopausal women on hormone replacement therapy or tamoxifen treatment [4,5]. The recurrence or the de novo appearance of endometriosis in postmenopausal women is well reported [6,7].

Both endometriosis and endometrial cancer share some common possible aetiological pathways. These include hormonal dysregulation and estrogen dependence, chronic inflammation and oxidative stress, molecular aberrant pathways like PI3K/AKT/mTOR and genetic and epigenetic alterations like PTEN, ARID1A and PIK3CA [8]. The exact aetiology of endometriosis in both premenopausal and postmenopausal women remains unclear. Excess estrogen is a common underlying association of this condition. As in most cases of postmenopausal endometriosis, in our case too, it is unclear whether the current endometriotic disease was a continuation of a previously undiagnosed condition or a de novo development. The decline in estradiol concentrations after menopause may not take away the estrogen backing completely as the estradiol is replaced by another estrogen, the estrone. In some women, there may also be an additional source of estrogen in the form of obesity, phytoestrogens or hormone replacement therapy. The crossing of a particular 'estrogen threshold' may activate the remnants of endometriosis in postmenopausal women even though these may remain less active or extensive [9,10].Additionally, endometriomas may have some intrinsic endocrine activity to produce estrogen and aromatase locally [11]. The aromatase is known to convert cholesterol into estrogen under continuous stimulation of prostaglandin E2 [12]. This extra-ovarian local oestrogen may contribute to the growth of endometriosis despite the hypoestrogenic postmenopausal state. This would also mean themeasurement of serum estradiol levels in these women may still be in the postmenopausal range while the endometrioma may continue to grow.

The role of estrogen in endometrial cancer is relatively better studied. Unopposed estrogen-induced proliferation of endometrium is associated with anovulatory states like PCOS and endometrial hyperplasia, including the atypical type. Estrogen signallinghas been suggested to play a mitogenic role in the normal endometrium during the reproductive age group [13]. In the postmenopausal women, the unopposed estrogen theory may be more applicable as the estrone primed endometrium in this subset of women may persist and grow in the absence of sufficient progesterone. While obesity and exogenous estrogen administration are known risk factors for endometrioid cancer of the endometrium, neither of these was present in our case, thus suggesting a possible role of unidentified factors in the complex aetiopathogenesis of this condition.

To the best of our knowledge, only one case of coexisting endometrioma and endometrial cancer has been reported in a perimenopausal woman and our case is the first one in the postmenopausal age group [14].Our case was also different for lack of common risk factors, abnormal uterine bleeding or any suspicious finding of endometrial malignancy at imaging in the backdrop of a giant(over 10 cm size) ovarian endometrioma which is exceptionally rare in postmenopausal women. Matsushima et al reported a huge endometrioma presenting 5 years after menopause [15]. So, should postmenopausal women with past or current endometriosisbe investigated and managed differently than those in the reproductive age group? Given the estrogen-backed aetiological pathway, it may not be unreasonable tosupport a high index of suspicion and early investigation for endometrial malignancy in postmenopausal women with current or past history of estrogen-supported conditions like PCOS, endometriosis and fibroids.

Conclusion

Postmenopausal women with endometriotic ovarian tumours may be at increased risk of endometrial malignancy. A high index of suspicion and inclusion of directed endometrial surveillance in the preoperative workup may help improve detection and guide appropriate management planning in these women.

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