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Case Report

Endometrioma mimicking ovarian malignancy in a post-menopausal woman

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ABSTRACT

Endometriosis is an estrogen dependant disorder of reproductive-age women. It is uncommon after menopause, however, peripheral estrogen production may account for endometriosis in post-menopausal women. We reported a case of a 68 year old post-menopausal woman with an adnexal mass suspected malignant ovarian tumor on imaging and normal serum CA 125 levels. Total hysterectomy with bilateral salpingo-oophorectomy was done. The final histopathology revealed the diagnosis of ovarian endometriosis. Rarely, ovarian endometrioma can mimic ovarian malignancy in a post-menopausal woman.

Keywords: Post-menopausal endometriosis, Ovarian masses, Endometrioma, Ovarian malignancy, Estradiol, CA 125

INTRODUCTION

Endometriosis, a chronic benign inflammatory disease characterized by the presence of endometrial glands and stroma outside the uterine cavity, is an estrogen dependant disorder. The prevalence of endometriosis ranges from 6% to 10% in women of reproductive age group and 2% to 5% in post-menopausal women.[1] Endometriosis should generally resolve with menopause, but in some post-menopausal women, it can be reactivated by the estrogen production from peripheral conversion or due to hormone replacement therapy (HRT) and rarely with normal estradiol levels which have been attributed to the ability of biosynthesizing estrogen via the aromatase activity.[2]

Endometriosis and ovarian malignancy can have similar risk factors like nulliparity, late childbearing and history of infertility, hence either can mimic the other.[3]

We presented a case of ovarian endometriosis in a post-menopausal woman with no previous history of hormonal therapy or endometriosis.

CASE REPORT

A 68 year old, non-obese, P5L5 post-menopausal lady, known case of type II diabetes mellitus and hypertension presented with abdominal distension for the last four months. The patient was well four months ago when she noticed abdominal distension which was insidious in onset, gradual in progression and was associated with weight loss and decreased appetite. There was no history of associated vomiting or abdominal pain or fever. There were no bladder and bowel complaints. General and systemic examination did not reveal any abnormality. On abdominal examination, a 20 week gravid uterus size mass was palpable in the hypogastrium, firm in consistency, smooth surface, regular margins, fixed, immobile and non-tender. The lower margin of the mass could not be reached. On speculum examination cervix was flushed with the vagina and on bimanual examination a mass of 20 weeks gravid uterus size palpable more towards left fornix, going upwards and crossing the midline, firm in consistency, smooth surface and regular margins and non-tender. The uterus was not appreciated separate from the mass. Right

fornix was free and non-tender. Routine investigations and levels of tumor markers were normal. A whole abdominal ultrasound revealed a well-defined cystic lesion of 16.2×15.1 cm with echogenic debris within, atrophic uterus, thin endometrium, liver enlarged (16.9 cm) with grade 1 fatty changes, left kidney atrophic, right kidney normal. MRI of the abdomen confirmed the finding of a complex large abdominopelvic cyst (16.2×17.3×14 cm^3) arising from the left adnexa with hemorrhagic signal intensity and T2 hypointense debris within, likely a complex hemorrhagic ovarian cyst, neoplastic etiology, uterus normal in size and left ovary was not visualized separately. Mild hydronephrosis in right kidney, mildly prominent right ureter seen up to the iliac crossing, compressed by fat displaced by lesion, fat planes between the pelvic lesion and right distal ureter well maintained. Laparotomy was done. Per operatively a left ovarian mass of 20×20 cm was identified which was adherent to the large and small bowel. During dissection, inadvertent rupture of the mass occurred and 2 liters of chocolate-colored fluid got drained. Left-sided salpingo-oophorectomy was done and send for frozen section total hysterectomy with right salpingo-oophorectomy was done. Infracolic omentectomy was done because of a suspicious-looking tumor. Other abdominal, pelvic organs, pelvic and para-aortic lymph nodes were palpated and were found to be within normal limits. The scrape cytology revealed a borderline epithelial ovarian tumor. The final histopathology revealed endometrial glands and stroma showing degenerative atypia and there was evidence of chronic hemorrhage as depicted by hemosiderin-laden foamy macrophages. All these features were consistent with findings of a benign endometriotic cyst. Omentum was normal.

Final histopathology confirmed the diagnosis of ovarian endometrioma. Immunohistochemistry demonstrated estrogen and progesterone receptor positivity.

Figure 1: MRI showing large complex abdominopelvic cyst with hemorrhagic signal intensity and T2 hypointense debri within.

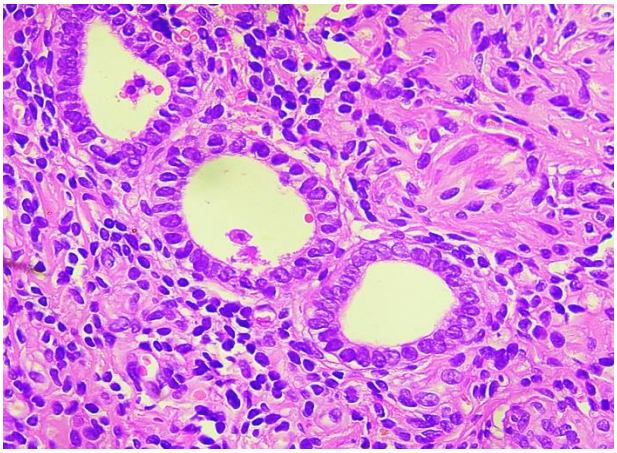

Figure 2: Histopathology section showing endometriotic focus in cyst wall.

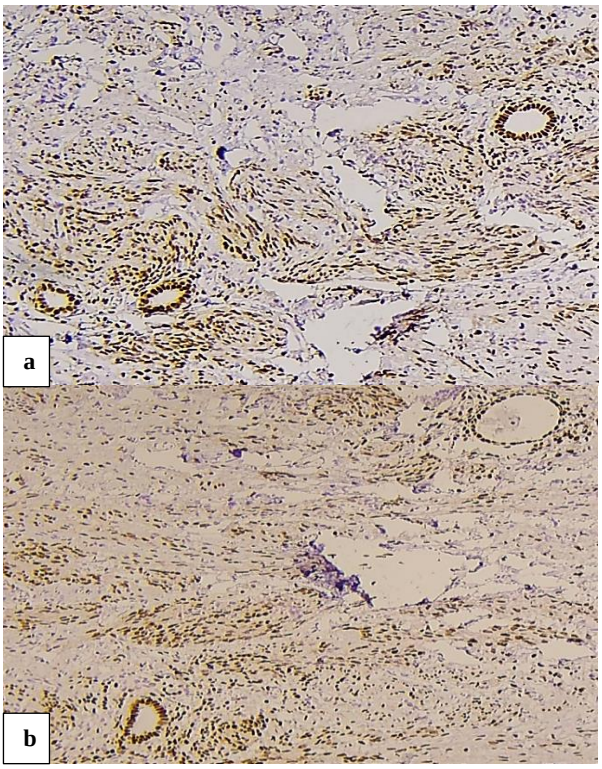

Figure 3 (a and b): Immunohistochemistry demonstrating estrogen and progesterone receptor positivity.

DISCUSSION

Endometriomas form a meagre 4.3% of the ovarian pathologies in the sixth decade of life primarily due to decreased levels of circulating estrogen.[4] Peripheral conversion of steroid precursors to estrogen in tissues especially fat may induce endometriosis reactivation or by receiving HRT.[2] Our case did not receive HRT, although she was non-obese, it did not seem to happen due to endogenous estrogens considering the absence of endometrial hyperplasia and appearance of symptoms several years after menopause. It had been explained in the

various reports that an intrinsic signaling pathway can make endometriotic cells function as an independent unit where ectopic endometrial cells can biosynthesize estrogen from cholesterol in the absence of any external substance, such as adrenal androgens.[5] An alternative explanation given for the progression of disease in postmenopausal women is that PGE2 secreted from macrophages and ectopic endometrial cells stimulates aromatase activity with local estrogen production.[2]

The expression of aromatase in retrograded endometrial cells was about 400 times.[6] This local estrogen production was the principal cause of steering endometriosis in post-menopausal women with normal serum estrogen levels. Also, estrogen plays a chief role in the recruitment of macrophages, the recruited macrophages play a key role in endometriosis production by secretion of vascular endothelial growth factor (VEGF) which may account for extensive vascularization contributing to massive size.[7] This theory seemed to be a reasonable explanation in our case.

The coelomic metaplasia theory has also been put forward for the occurrence of postmenopausal ovarian endometriotic lesions. 8 Interleukins (interleukin (IL)1, IL2, IL6, IL8, IL10) and other inflammatory mediators (tumor necrosis factor alfa, interferon-gamma, monocyte chemotactic protein-1) could play a main role in the endometriosis pathophysiology, triggering celomic metaplasia etiopathogenic mechanism. 9

The rare progressive cases of endometrioma in post-menopausal women commencing a decade after menopause can be explained by genetic- epigenetic theory.[10] The epigenetic mechanisms account for transmitting endometriosis at birth but the development of disease occurs later when additional incidents occur. It can also explain the growth of endometriosis with low circulating levels of estrogen. For each lesion, a specific set of incidents was specific and it can explain the heterogeneity of endometriotic lesions.

CONCLUSION

Endometriosis can occur at any age. Although its occurrence is rare after menopause, the possibility should always be considered. Regardless of their location and patient's age, these may reach up to large sizes. The possibility of malignancy should always be borne in mind. Ovarian endometriomas that are 9 cm or greater in diameter are a strong predictor for the development of ovarian cancer in postmenopausal women of 45 years of age or older.

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