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Stromal luteoma of the ovary: A rare ovarian pathology

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Introduction

Sex-cord ovarian tumours are a rare kind of ovarian neoplasm. Steroid cell tumours that account for 0.1% of all primary ovarian tumours are also a sub-group of sex-cord ovarian tumours. Steroid cell tumours are classified into three groups, according to the origin of the cells that generate the tumour: stromal luteomas; Leydig cell tumours; and steroid cell tumours not otherwise specified (NOS). Stromal luteomas constitute 20 to 25%, Leydig cell tumours 20 to 25% and NOS 50 to 60% of all steroid cell tumours. Steroid cell tumours can be seen in all ages, however the incidence increases in the fifth and sixth decades (Young 2011; Hayes and Scully 1987a).

Symptoms may differ depending on the hormones secreted from the tumour cell. Stromal luteomas are usually present with hyper-oestrogenic symptoms when Leydig cell tumours are mostly present with hyper-androgenism, and the NOS group is often seen with androgenic effects. However in the NOS group, hyper-oestrogenism may rarely be seen, as 25% of NOS tumours may have no hormonal activity (Hayes and Scully 1987a,b).

In this report, we present a woman who came to our clinic with gestagen resistant postmenopausal bleeding, and was diagnosed with stromal luteoma of the ovary from the pathological specimen following total abdominal hysterectomy and bilateral salpingo-oophorectomy.

Case report

A 54-year-old woman, who was in menopause for six years and had abnormal vaginal bleeding for 4 years, was referred to our clinic. She had several probe curettages for increased endometrial thickness, detected by transvaginal ultrasonographic examination (TV-US); since the pathologies were benign, she was followed without drug therapy.

During the follow-ups, the patient's vaginal bleeding continued, and operative hysteroscopy was performed, where an endometrial polyp was excised. The pathological result of the specimen was reported as endometrial hyperplasia without atypia, so norethisterone 3×10 mg treatment was given. After six months, transvaginal ultrasonography (TV-US) examination of the patient showed that the uterus and the ovaries were slightly atrophic, however the endometrial thickness was measured as 11 mm (Figure 1A). Therefore, control curettage was performed and a pathological specimen was reported as 'endometrium with irregular proliferation'. The patient was diagnosed with drug resistant bleeding; with this indication, total abdominal hysterectomy and bilateral salpingo-oophorectomy was performed.

Before the operation, the total blood count and tumour markers were within normal ranges. During the operation, the uterus was seen to be normal in size and shape, and both ovaries were atrophic. Macroscopic examination of the specimen done after the operation, showed a 1.7 cm, dirty yellow and white coloured nodular lesion, with clear boundaries in the right ovary. Tumoral histomorphology seen in the microscopic examination of this region revealed polygonal round cells forming solid islands and irregular clusters in some regions. The cells in this architecture have clear borders in hyalinised fibrocollagen stroma, nuclei localised in the centre and wide eosinophilic granular cytoplasm in some areas (Figure 1B, C). The number of mitoses in the tumoral tissue was 1/10 HPF. In slices, Reinke crystalloids, necrosis, bleeding, high-grade nuclear atypia or pigments were not detected. Immunohistochemical staining with EMA, inhibin, chromogranin and synaptophysin indicated a positive immunoreactivity. All these findings were histopathologically diagnosed as stromal luteoma. Hyperplasia without atypia was observed in the endometrium. Since the final histopathological diagnosis was stromal luteoma, which is a benign lesion, additional treatment was not recommended.

Discussion

Our case, who was referred to our clinic with drug resistant bleeding and had total abdominal hysterectomy and bilateral salpingo-oophorectomy, was diagnosed with stromal luteoma. Postmenopausal bleeding (PMB) should be examined carefully even if the bleeding lasts for a short time or is in small amounts. Endometrial atrophy is the most frequent (60 to 80%) reason for PMB. Endometrial hyperplasia accounts for 5 to 10%, whereas endometrial cancer constitutes 10%. Aetiology of the excessive oestrogen source in endometrial hyperplasia may be obesity, exogenous oestrogen intake or oestrogen secreting ovarian tumours, such as sex-cord stromal tumours (Berek 2007).

Steroid cell tumours account for 0.1% of all primary ovarian tumours, and are classified into three groups, as given above: stromal luteomas, Leydig cell tumours and steroid cell tumours not otherwise specified (NOS). Stromal luteomas constitute 20 to 25% and are

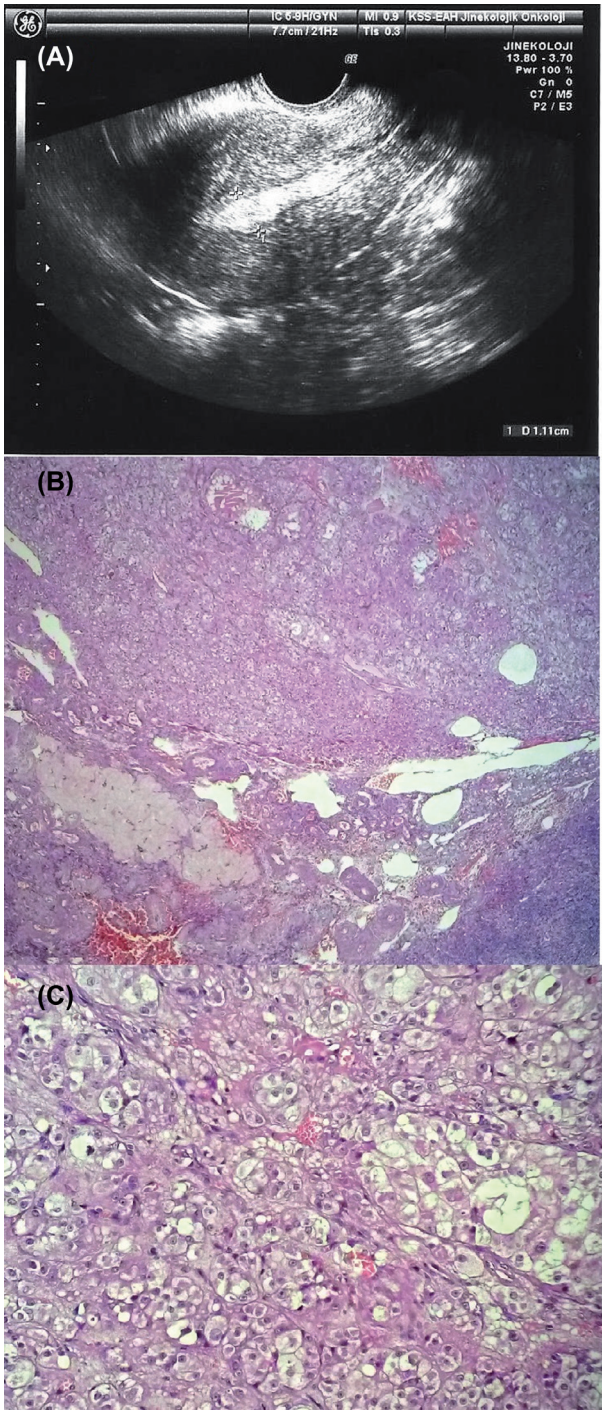

Figure 1. (A) Transvaginal ultrasound indicating 11 mm thickness of endometrium with atrophic ovaries. (B) Tumoral tissue with clear boundaries located inside fibrohyalinised ovarian stroma (H&E, × 40). (C) Tumoral structure formed of polygonal round cells with clear boundaries, nuclei localised in the centre and wide eosinophilic granular cytoplasm. (H&E, × 40).

always benign. They are the tumours which are compromised of small steroid cells in the ovarian stroma, whereas Leydig cell tumours are usually localised in the hilus of the ovary (Sternberg and Roth 1973). About 90% of stromal luteomas show stromal hyperthecosis. Usually, these tumours are < 3 cm and unilateral. They have clear borders, are solid and mostly grey-white or yellow coloured tumours (Hayes and Scully 1987a; Sternberg and Dhurandhar 1977). These properties corresponded with our findings.

Young (2011) showed that stromal luteomas occur mostly (80%) in postmenopausal women, and 60% of the patients present with hyper-oestrogenic abnormal bleeding. Similarly, our case was referred to our clinic with resistant postmenopausal bleeding. Hyper-oestrogenic state was also confirmed with the pathological examination of the endometrium. This may also be seen in some NOS (Hayes and Scully 1987b). Only 12% of the patients with stromal luteoma have androgenic symptoms (Hayes and Scully 1987a).

Young (2011) reported that stromal luteomas and Leydig cell tumours are mostly seen in postmenopausal women, whereas NOS are mostly seen in young premenopausal women (mean age 43). Preoperative TV-US could not detect any lesion; however, a pathological specimen revealed a 1.7 cm sized tumour. Small-sized tumours are mostly seen in stromal luteomas. Mean tumour diameters are 1.3 cm for stromal luteomas, 2.4 cm for Leydig cell tumours and 8.4 cm for NOS (Young 2011).

Stromal luteoma and Leydig cell tumours are not confused pathologically because stromal luteomas are localised in the stroma, whereas Leydig cell tumours localise in the hilus of the ovary (Sternberg and Dhurandhar 1977). Moreover, Leydig cell tumours contain steroid cells that include Reinke crystalloids (Roth and Sternberg 1973). Microscopic examination of the specimen also showed stromal hyperthecosis, which exists in 92% of stromal luteomas, 42% of Leydig cell tumours and 23% of NOS (Young 2011).

Microscopically, stromal luteomas look similar to theca lutein cysts. Theca lutein cysts are always found by a follicle in the stroma, but this is not observed in stromal luteomas. Malignant melanomas can mimic stromal luteomas and can be differentiated with S100 and HMB-45 staining being negative, as in our case (Scully 1964). With clinical and histopathological examination of our case, we diagnosed our patient with stromal luteoma. These tumours are known to be benign tumours, so surgical staging was not recommended. As in epithelial ovarian cancers, treatment of sex-cord tumours is surgery. There is no known chemotherapy agent or radiotherapy for sex-cord tumours, since these tumours are seen very rarely (Haji et al. 2007).

In conclusion, ovaries should be examined carefully with transvaginal ultrasonography to reveal any accompanying ovarian pathology in women with postmenopausal bleeding, and stromal luteomas should be considered as a reason for the postmenopausal bleeding, even though they are rare.

Declaration of interest: The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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