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Sertoli Cell Ovarian Tumor in a 59-Year-Old Postmenopausal Woman: A Case Report and Literature Review

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Abstract

Sertoli-Leydig cell tumors of the ovary are exceedingly rare, whereas Sertoli cell tumors represent an even more exceptional histological subtype. Sertoli cell tumors predominantly occur in young or middle-aged women, while Sertoli-Leydig cell tumors are typically diagnosed in younger patients. In both entities, postmenopausal presentation is uncommon. We report the case of a 59-year-old woman, postmenopausal for eight years, who presented with pelvic pain. Imaging revealed a heterogeneous right adnexal mass adjacent to the uterus. The patient underwent curative surgery consisting of exploratory laparotomy, total hysterectomy, and bilateral salpingo-oophorectomy. The tumor was staged as FIGO stage IC2. Histopathological examination confirmed a Sertoli cell tumor. No clinical evidence of a hormone-secreting syndrome was observed, particularly no postmenopausal bleeding, supporting the non-functional nature of the tumor. The patient received six cycles of adjuvant chemotherapy with carboplatin and paclitaxel, with a favorable postoperative outcome and follow-up.

Keywords

Ovarian Tumor, Sertoli Cell Tumor, Sertoli-Leydig Cell Tumor, Postmenopausal Woman, Adjuvant Chemotherapy

1. Introduction

Sertoli-Leydig cell tumors are very rare neoplasms originating from the sex cord-stromal tumors and account for less than 0.5% of all ovarian tumors [1]. Within this category, Sertoli cell tumors are even rarer, representing approximately 4% of all ovarian sex cord-stromal tumors [2]. They can occur at any age but predomi-

nantly affect young or middle-aged women [3]. Their rarity and nonspecific symptoms make the diagnosis of Sertoli cell ovarian tumors challenging. Definitive diagnosis is confirmed only by histopathological examination after surgical resection [3] [4]. Functionally, Sertoli-Leydig cell tumors most often secrete androgens, which are responsible for signs of virilization [5]. In contrast, Sertoli cell tumors may occasionally secrete estrogens, leading to clinical manifestations such as postmenopausal bleeding [3]. However, some Sertoli cell tumors remain clinically non-functional, as in our case, where the tumor was associated with non-atypical endometrial hyperplasia without clinical signs of hyperestrogenism. We present the case of a 59-year-old woman, postmenopausal for eight years, diagnosed with a right ovarian tumor purely composed of Sertoli cells, without a Leydig cell component, which presented clinically with pelvic pain.

2. Case Presentation

2.1. Patient

A 59-year-old woman, postmenopausal for eight years, G6P6 (six pregnancies, six full-term deliveries), with no history of miscarriage or ectopic pregnancy.

2.2. Personal and Family History

Personal history: No significant medical or surgical history prior to the surgery.
Family history: No similar cases reported in the family.

2.3. Circumstances of Discovery

The patient presented with persistent pelvic pain for eight months. No other hormonal or gastrointestinal symptoms were reported. The ovarian mass was discovered during this evaluation, and the patient underwent surgery in the Gynecology Department of Hassan II University Hospital, Fez. She was subsequently referred to the Medical Oncology Department of the same institution for adjuvant treatment.

2.4. Initial Clinical Examination (Medical Oncology)

- General examination: Patient in good condition (ECOG 0)
- Abdomen: Clean, well-healed laparotomy scar
- Lymph nodes: No palpable lymphadenopathy
- Pelvis: No palpable masses
- Signs of hormone secretion: None observed, including postmenopausal bleeding

2.5. Preoperative Imaging

- Pelvic ultrasound: Normal uterus, thin endometrium, heterogeneous right adnexal mass measuring 6 × 5 cm, arising from the right ovary
- Pelvic MRI (October 20, 2023): Right-sided pelvic soft tissue mass measuring 70 × 65 × 60 mm, consistent with an ovarian tumor (ORADS V)

- MRI review (October 25, 2023): Right adnexal mass adherent to the ipsilateral ovary; ovarian origin probable; gastrointestinal stromal tumor (GIST) not excluded

2.6. Surgical Management

The patient underwent exploratory laparotomy with:

- Right salpingo-oophorectomy
- Total hysterectomy with en bloc left salpingo-oophorectomy
- Infracolic omentectomy
- Biopsies of the right and left paracolic gutters
- Peritoneal cytology

Perioperative findings: Right ovarian mass measuring 80 mm, highly vascularized and partially ruptured, with free tissue fragments in the pelvic cavity; left adnexa, liver, spleen, digestive tract, and paracolic gutters were normal; no peritoneal effusion observed.

2.7. Histopathology and Additional Analyses

- Endometrium and left adnexa: Histological examination of the endometrium revealed a mucosa composed of regular glandular structures, slightly increased in density, without cytonuclear atypia, compatible with simple non-atypical hyperplasia. The cervix, left fallopian tube, and left ovary were histologically normal.
- Right adnexal mass: Tumor proliferation organized in tubules and cords, separated by a thin collagenous stroma. Cells were monomorphic, medium-sized, oval, with fine chromatin nuclei. No Leydig cells or tumor necrosis were observed. Immunohistochemistry showed expression of CK AE1/AE3, calretinin, WT1, and focal CK 8/18, with no expression of CK7, EMA, hormone receptors, chromogranin, or GATA3, consistent with a Sertoli-type sex cord tumor. Molecular analysis revealed no FOXL2 exon 1 mutation, excluding a granulosa cell tumor.
- Omentectomy and paracolic gutter biopsies (right and left): Adipose and fibroadipose tissues were free of tumor proliferation.
- Peritoneal fluid: Cytological examination showed macrophages and lymphocytes on a hemorrhagic background, with regular mesothelial cells; no neoplastic cells identified, consistent with reactive fluid.
- Histopathological conclusion: Simple non-atypical endometrial hyperplasia; right adnexal mass corresponding to a Sertoli-type sex cord tumor; left adnexa, omentectomy, and peritoneal biopsies free of tumor; peritoneal fluid reactive.

2.8. Stage and Classification

FIGO classification: Stage IC2

Rationale: Tumor confined to one ovary, with capsular rupture prior to surgery, no regional lymph node metastases, and no distant metastases.

2.9. Adjuvant Treatment and Follow-Up

The patient received six cycles of adjuvant carboplatin-paclitaxel chemotherapy, initiated on April 7, 2024, and completed on June 26, 2024.

At the latest follow-up, the patient was in good general condition, under regular monitoring, with no evidence of local or metastatic recurrence.

A serum CA-125 tumor marker measurement was performed for the first time on September 25, 2024, three months after the last chemotherapy cycle on June 26, 2024, showing a value of 16.2 U/mL, within the normal range. A second assessment, conducted on May 22, 2025, demonstrated a stable and normal level. No other serum tumor markers were evaluated. This course is consistent with the typically non-secreting nature of pure Sertoli cell tumors.

3. Discussion

Sertoli-Leydig cell tumors are rare ovarian neoplasms, usually unilateral, with bilateral presentation observed in only 1.5% of cases. They are most often confined to the ovary at the time of diagnosis. Histologically, they consist of Sertoli and Leydig cells in variable proportions and are classified according to their degree of differentiation into well-differentiated, intermediate, or undifferentiated forms [6].

In contrast, Sertoli cell tumors are extremely rare and differ from Sertoli-Leydig cell tumors by the complete absence of Leydig cells and immature stroma, which constitutes a major diagnostic criterion [7].

Immunohistochemistry represents a key tool for confirming this distinction: all sex cord tumors express SF-1, while inhibin and calretinin are expressed in the majority of cases, with variability depending on the tumor type. WT1 is expressed in both pure Sertoli cell tumors and Sertoli-Leydig cell tumors. MART-1 (Melan A) is specific to the Leydig cell component and is positive in 94% of Sertoli-Leydig cell tumors, but absent in pure Sertoli cell tumors. Therefore, the immunohistochemical profile of SF-1+, inhibin+, WT1+, and negative MART-1, combined with the morphological absence of Leydig cells, allows confirmation of a pure Sertoli cell tumor and exclusion of the more common Sertoli-Leydig subtype [8].

3.1. Epidemiology and Clinical Features

Sertoli cell tumors are rare ovarian neoplasms that, together with Sertoli-Leydig cell tumors and Leydig cell tumors, account for less than 1% of all ovarian tumors [9]. In the largest series published by Oliva *et al.*, including 54 cases, patients' ages ranged from 2 to 76 years (mean 30 years). All tumors were unilateral, and the majority were diagnosed at an early stage (44 stage I cases: 42 IA and 2 IC) [3].

3.2. Hormonal Profile and Clinical Manifestations

Sertoli-Leydig cell tumors frequently secrete androgens, mainly testosterone, responsible for virilization, while estrogenic secretion is rare [10]. In contrast, Sertoli cell tumors are most often non-functional. When hormonal activity is present,

it is mainly estrogenic, potentially causing menstrual irregularities, postmenopausal bleeding, or endometrial hyperplasia. Progesterone or testosterone secretion is exceptional and may lead to endometrial decidualization or signs of amenorrhea or virilization, respectively [7].

In our case, the patient exhibited no clinical signs of hormonal secretion, confirming the non-functional nature of the tumor. However, histological examination revealed non-atypical endometrial hyperplasia, suggesting the presence of subclinical estrogenic secretion. Although clinically silent, this phenomenon may carry prognostic implications, particularly regarding endometrial surveillance. This observation highlights that the absence of overt hormonal symptoms does not entirely exclude a degree of hormonal activity within the tumor.

3.3. Macroscopic Morphology and Immunohistochemistry

Macroscopically, these tumors usually appear as lobulated, solid masses, yellowish to brown in color. Immunohistochemically, EMA, inhibin, and chromogranin form the most useful triad of markers to exclude two common mimickers of Sertoli cell tumors: endometrioid carcinoma (inhibin−; EMA+; chromogranin−) and carcinoid tumor (inhibin−; EMA+; chromogranin+) [3]. Although CD99 and calretinin are often expressed in these tumors, they are less specific and less useful in differential diagnosis.

In our case, from a differential diagnostic standpoint, a gastrointestinal stromal tumor (GIST) was initially considered based on the preoperative imaging findings. However, histopathological and immunohistochemical analyses allowed this hypothesis to be ruled out. The absence of CD117 (c-KIT) and DOG1 expression, hallmark markers of GISTs [11], together with the positive staining for calretinin and WT1, strongly supported the diagnosis of a sex cord-stromal tumor of the Sertoli cell type.

3.4. Therapeutic Management and Prognosis

For sex cord-stromal tumors, FIGO stage IC2 after complete surgery justifies platinum-based adjuvant chemotherapy due to the increased risk of recurrence associated with capsular rupture.

According to NCCN guidelines, the preferred regimen for these patients is six cycles of carboplatin-paclitaxel. The BEP regimen (bleomycin, etoposide, cisplatin) is an alternative, particularly in younger patients or when carboplatin-paclitaxel is contraindicated. The EP regimen (etoposide-cisplatin) may be used if bleomycin is contraindicated [12].

ESMO recommendations support this approach, emphasizing that adjuvant chemotherapy is indicated for high-risk stage IC tumors following complete surgery [13].

In our case, the postmenopausal patient with a stage IC2 Sertoli cell tumor received six cycles of carboplatin-paclitaxel according to these guidelines, with good tolerance and favorable outcome.

Overall prognosis for sex cord-stromal tumors confined to the ovary remains favorable, but prolonged follow-up is recommended due to the rare but possible occurrence of late recurrences. Given this risk, long-term follow-up is recommended. According to ESMO, the median time to recurrence is 4 - 6 years, with cases reported up to 37 years after diagnosis. Follow-up visits should include a clinical examination and, if indicated, tumor marker assessment every 6 months from the third year onward, with this schedule maintained indefinitely. The NCCN recommends, for early-stage low-risk patients, follow-up visits every 6 - 12 months, and for high-risk stages, every 4 - 6 months during the first two years, with imaging performed only if symptomatic or in case of abnormal laboratory findings [12] [13].

This case illustrates a Sertoli cell tumor occurring in a postmenopausal setting, an exceptionally rare event. Despite the presence of endometrial hyperplasia, the patient exhibited no clinical signs of hormonal secretion, confirming the clinically non-functional nature of the tumor. Recently, Kumar *et al.* (2025) [14] reported a similar case of a pure Sertoli cell tumor incidentally discovered in an atrophic ovary of a 70-year-old postmenopausal woman, underscoring the rarity and clinical relevance of such observations. These cases highlight the importance of a multidisciplinary diagnostic approach and management aligned with international guidelines, while contributing valuable insights to the limited literature on this uncommon entity.

4. Conclusion

Sertoli cell tumors remain exceptionally rare entities, particularly in the postmenopausal setting. This case illustrates that their presentation can be clinically silent, despite detectable biological effects such as endometrial hyperplasia, which renders diagnosis challenging. Documenting such cases contributes to a better understanding of their clinical behavior, guides therapeutic management, and underscores the importance of prolonged follow-up to prevent recurrence.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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