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Case Report

The inner alchemy: a steroid cell transformation of ovary

Harsha O. Rajani[1]*, Deepa V. Mungi[1], Bhakti S. Patil[1], Ashutosh L. Jha[2]

[1]Department of Obstetrics and Gynaecology, Khan Bahadur Bhabha Hospital, Kurla, Mumbai, Maharashtra, India
[2]Department of Radiology, Deben Mahato Government Medical College and Hospital, Purulia, West Bengal, India

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***Correspondence:**
Dr. Harsha O. Rajani,
E-mail: harsharajani5@gmail.com

ABSTRACT

Steroid cell tumors (SCTs) are an uncommon subset of ovarian sex cord-stromal tumors, accounting for approximately 0.1% of all ovarian neoplasms. They are categorized into three subtypes based on cellular origin: Leydig cell tumors, stromal luteomas, and SCTs not otherwise specified (SCT-NOS). SCT-NOS is characterized by an uncertain lineage and a higher likelihood of hormonal activity. These tumors are frequently functional, most commonly secreting testosterone, and may present with clinical features of hyperandrogenism, including virilization, hirsutism, and menstrual disturbances such as amenorrhea. Given their rarity and variable presentation, SCT-NOS tumors pose a diagnostic challenge and require careful clinical, biochemical, radiological, and histopathological correlation for accurate diagnosis and management. This case highlights a 20-year-old female who presented with virilization, sudden voice change, hirsutism, acne, and abnormal uterine bleeding. Ultrasound showed a solid nodule in the right ovary, and salpingo-oophorectomy was performed. Histology confirmed a SCT-NOS of the right ovary. Postoperatively, the patient made a remarkable recovery of menstrual symptoms, demonstrating the importance of prompt diagnosis and surgical management in such cases. During follow-up, she has remained asymptomatic, with no evidence of tumor recurrence on clinical or imaging evaluation.

Keywords: Hyperandrogenism, Virilization, Steroid cell tumor, Ovarian tumor

INTRODUCTION

Steroid cell tumors (SCTs) are a rare group of ovarian sex cord-stromal tumors composed entirely of cells with morphological features suggestive of steroid hormone secretion, accounting for less than 0.1% of all ovarian tumors. They are typically unilateral and benign and are classified into three subtypes based on cellular origin: stromal luteoma, which arises from the ovarian stroma; Leydig cell tumor, which originates from Leydig cells in the ovarian hilum; and steroid cell tumor, NOS, in which the cellular lineage is uncertain. However, the exact etiology of these tumors remains undetermined.[1]

Among ovarian SCTs, the NOS subtype accounts for approximately 56% of all cases.[2] This subtype is most commonly observed in women of reproductive age, particularly during the third and fourth decades of life. These tumors are often functional and may secrete testosterone, resulting in clinical features such as virilization, hyperandrogenism, and amenorrhea.[3] Approximately 56% of patients present with hirsutism. SCTs can produce a wide range of steroid hormones, and elevated estradiol levels have been reported in 6-23% of cases.[4] Additionally, Cushing's syndrome has been described in 6% to 10% of patients.[5] Notably, 25% to 45% of these tumors exhibit malignant behavior.[6] This report describes a case of a 20-year-old woman presenting with virilization due to a right ovarian SCT-NOS, along with a review of key clinical, diagnostic, and management considerations.

CASE REPORT

A 20-year-old nulligravida woman, married since three months, came to the OPD with complaints of irregular menses and pain in abdomen since 6 months, hoarseness

of voice and hirsutism since three months and is anxious about the sudden onset of change in her voice. Previously, her menses were regular, occurring every 28-30 days with normal duration and moderate flow. Over the past 6 months, her cycles have become irregular, occurring every 2-3 months, with decreased flow (soaking 1 pad per day). She denied any history of dysmenorrhea. Pain in the abdomen was intermittent and colicky in nature, not related to menstruation, on and off requiring anti-spasmodic since 6 months. Later, she noticed hoarseness of voice, coarse hair growth on chin, chest, upper lip, hyper pigmentation on neck and armpits-acanthosis nigricans, weight gain of about 3 kg in 2 months. She had no history of contraceptive use, any drug use, stressors or significant personal or family medical history.

Physical examination revealed a deepening voice, hirsutism (Modified ferryman-Gallwey score-9) breast examination tanner stage 5 and no mass detected, on per abdominal examination-no mass felt, whereas on per vaginal examination a firm 5×5 cm mobile mass in the posterior fornix with normal uterine size and no cervical motion tenderness and bilateral fornices free.[7] On local examination, vulval appearance was unremarkable. On general and systemic examination, no significant abnormality was detected; vital signs were stable.

Investigations

Laboratory analysis revealed normal underlying investigations (Complete blood count, liver and renal function test, serum electrolytes, random blood sugar, international normalized ratio, thyroid function test), with elevated serum free testosterone (6.38 ng/dl, normal 0.06-1.08 ng/dl), total testosterone (15 ng/dl, normal 8-60 ng/dl), and inhibin B (210 pg/ml, normal 45-200 pg/ml). Although tumor markers (CA-125, CEA, CA19-9, LDH, AFP and beta-hCG), FSH, LH, prolactin and thyroid test were all within the normal range. Normal DHEAS levels (75 ug/dl; normal 65-380 ug/dl). Ultrasound showed a 55×51 mm well-defined solid mass of right adnexal origin with high vascularity on doppler and right ovary not seen separately, to rule out ovarian dysgerminoma versus ovarian Sertoli Leydig cell tumor (ovarian androblastoma) rest findings of uterus and left ovary were normal. MRI confirmed a well-defined focal lesion is noted in the pelvis on the right ovary measuring approximately 5.7×4.9×5.2 cm. It is isotense on T1-weighted images and heterogeneous signal intensity on T2-weighted images with intermediate signal intensity areas within. There was no evidence of metastasis, ascites, or enlarged lymph nodes. The left ovary, uterus, cervix, and vagina were unremarkable. ECG and chest X-ray done and no abnormality detected (Figure 1).

From an endocrinological perspective, the clinical presentation of rapid-onset virilization with markedly elevated serum testosterone is highly suggestive of an androgen-secreting ovarian neoplasm. Further evaluation, including serum DHEA-S estimation and MRI of the abdomen and pelvis, is recommended to delineate the source of androgen excess and to confirm the ovarian origin of the tumor.

Treatment and follow-up

The patient underwent a right salphingo-oophorectomy with omental biopsy and peritoneal washing. No free fluid or peritoneal collection was noted. Peritoneal washing was performed with normal saline, and the sample was sent for cytology. The omentum appeared grossly normal, with no evidence of metastatic deposits; an omental biopsy was obtained for histopathological examination. A solid right ovarian mass measuring 6×4×2 cm was identified, with the fallopian tube stretched and adhered to the mass. The uterus and left ovary appeared grossly normal. Right-sided salphingo-oophorectomy done and specimen was sent for histopathological evaluation (Figure 2).

Figure 1: Imaging findings on ultrasonography and Doppler examination.

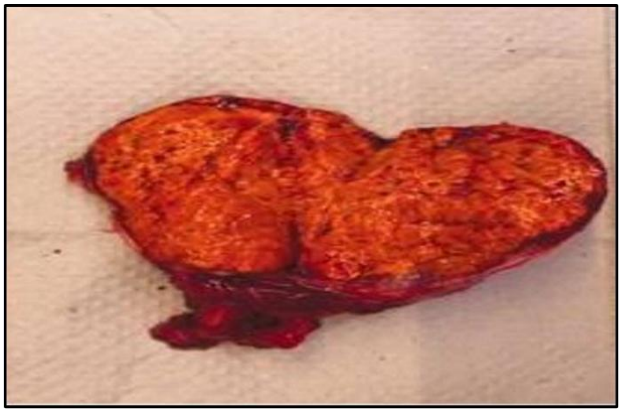

Figure 2: Gross specimen showing the right ovary with attached fallopian tube.

Histopathology

A gross specimen was received with the ovary measuring 65×50×30 mm. External appearance gray white with shiny smooth surface, no capsular breach. Cut section of the ovary showed a well circumscribed, solid, yellow nodule

measuring 40×40 mm with focal areas of pinpoint hemorrhage noted (Figure 3).

Microscopy suggests a well circumscribed neoplasm composed of polygonal to round tumor cells with distinct borders, central nucleoli and moderate to abundant cytoplasm arranged predominantly in a diffuse pattern and focally in a nest, separated by a fibrous septum. No evidence of necrosis, hemorrhage, or marked atypia was observed. Impression-ovarian mass-steroid cell tumor. Immunohistochemistry-stained positive for inhibin-B suggesting SCT-NOS.

Omental biopsy was unremarkable, and peritoneal washings were negative for malignant cells.The diagnosis was clinically supported by the partial resolution of virilizing features and restoration of a regular menstrual cycle following surgical excision of the lesion. During follow-up, the patient has remained asymptomatic, with no clinical or radiological signs suggestive of tumor recurrence.

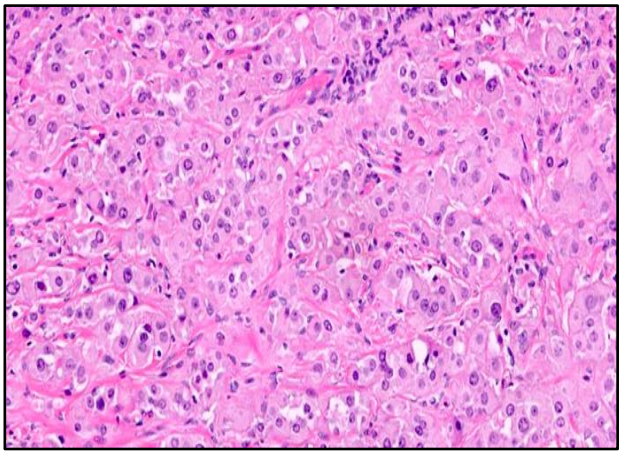

Figure 3: Histopathological appearance.

DISCUSSION

The most important factor to determine in SCTs of the ovary is whether the tumor has malignant features or not.

Surgery is the most important and hallmark treatment for a benign steroid cell tumor, and complete excision of the tumor could provide the regression of symptoms with partial improvement in virilizing effects. The diagnosis of SCTs, NOS, should be made on the basis of the clinical virilizing signs, the microscopic and macroscopic pictures, as well as immune reactivity to some immune-histochemical markers. Therapy should be individualized based on tumor histology, surgical staging, and the desire for fertility preservation.[8] Although SCTs are usually benign, they possess a potential for malignant transformation. In young patients who are keen on fertility preservation, unilateral salpingo-oophorectomy (USO) is the preferred choice of treatment.[9] SCTs in young women without evidence of malignancy on histopathology have excellent surgical outcomes and prognosis. In the present case, USO was favored over unilateral oophorectomy as a fertility-sparing surgical approach in certain conditions such as borderline ovarian tumors (BOTs), as it significantly reduces the risk of recurrence without compromising the chances of successful pregnancy.[10]

SCTs differ from polycystic ovary syndrome and non-classical congenital adrenal hyperplasia (NCCAH) primarily by their rapid onset of severe virilization, markedly elevated testosterone, and presence of a distinct ovarian mass on imaging.[11] In contrast, PCOS and NCCAH follow a gradual course, with milder biochemical abnormalities and no discrete tumor. Elevated 17-hydroxyprogesterone and DHEA-S levels point toward NCCAH, while normal 17-hydroxyprogesterone with very high testosterone favors a steroid cell tumor. Surgical excision leading to symptom resolution confirms the neoplastic nature of SCTs.

Table 1: Comparison of clinical and biochemical features of SCT, PCOS, and NCCAH.

Features	SCT	PCOS	Non-classical congenital adrenal hyperplasia
Onset	Rapid (weeks-months)	Gradual (years)	Gradual
Severity of symptoms	Severe virilization	Mild-moderate hirsutism	Mild-moderate hyperandrogenism
Virilization (voice deepening, clitoromegaly)	Common, rapid, marked	Rare	Uncommon, mild
Testosterone levels	Markedly elevated	Mildly elevated	Mild-moderately elevated
17-hydroxyprogesterone (17-OHP)	Normal	Normal	Elevated (↑ after ACTH stimulation)
DHEA-S levels	Normal or mildly ↑	Normal or mildly ↑	Elevated
Imaging findings	Solid ovarian mass (often vascular)	Polycystic ovaries (multiple follicles)	Normal or PCOS-like ovaries
Nature of disorder	Neoplastic	Functional endocrine	Functional (enzymatic defect)
Response to treatment	Surgical removal → resolution	Medical management	Medical management

CONCLUSION

SCTs NOS are rare ovarian tumors which diagnostic challenge. In a case of rapid onset of hirsutism, virilization, and solid ovarian lesions on imaging, it is extremely important to maintain a high index of suspicion. One can establish a final diagnosis according to the clinical manifestations and laboratory values in addition to imaging studies and operative findings of the ovarian mass. SCTs carry a potential risk of malignancy; therefore, thorough pathological evaluation is necessary to exclude malignant features. Immunohistochemical analysis further aids in establishing an accurate diagnosis.

This case exemplifies rapid diagnosis and successful outcome in reproductive-age women like this 20-year-old, with unilateral salphingo-oophorectomy, highlighting surgery as the cornerstone of treatment balances oncologic safety, cures benign cases (>95%) and averts infertility with childbearing potential; literature confirms successful pregnancies post-resection. SCTs in young women without evidence of malignancy on histopathology have excellent surgical outcomes and prognosis.

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