Neuroendocrinology Letters Volume 46 No. 5 2025
ISSN: 0172-780X; ISSN-L: 0172-780X; Electronic/Online ISSN: 2354-4716
Web of Knowledge / Web of Science: Neuroendocrinol Lett
Pub Med / Medline: Neuro Endocrinol Lett

ORIGINAL ARTICLE

Clinical Analysis of 14 Cases with Androgen-Secreting Ovarian Sex Cord-Stromal Tumors: Diagnosis, Treatment, and Literature Review

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Submitted: 2025-06-17 Accepted: 2025-11-20 Published online: 2025-11-25

Key words: **Ovarian Sex Cord-Stromal Tumors; hyperandrogenism;clinicopathological; differential diagnosis; congenital adrenal hyperplasia**

Neuroendocrinol Lett 2025; **46**(5):259–264 **PMID:** 41337645 46052503 © 2025 Neuroendocrinology Letters • **www.nel.edu**

Abstract

OBJECTIVE: To analyze the clinical data of 14 patients with androgen-secreting ovarian sex cord-stromal tumors (OSCSTs), a rare cause of female hyperandrogenism (HA), aiming to enhance differential diagnosis and reduce misdiagnosis of these diseases.

METHODS: Fourteen female patients with pathologically confirmed OSCSTs were retrospectively enrolled in this study.General clinical data were retrospectively collected from medical records, encompassingclinical manifestations, diagnostic and therapeutic interventions. A systematic literature review about the management of OSCSTs were performed.

RESULTS: Among 14 female patients with OSCSTs, age ranged from 12 to 69 years (mean ± SD: 34.4 ± 20.1 years), with symptom duration prior to diagnosis spanning 1-6 years (2.3 ± 1.4 years). Adolescent and reproductive-aged patients (n = 10) universally exhibited oligomenorrhea/amenorrhea with virilization, while postmenopausal patients (n = 4) presented with virilization alone. Preoperative testosterone levels were markedly elevated in all these patients (range 3.90-119.6 nmol/L; normal: 0.2–2.6). Imaging evaluation revealed ovarian masses on ultrasound in 78.6% (11/14) of cases. After complete tumors resection, testosterone reduced significantly to 0-6.51 nmol/L within 48 hours with complete resolution of hyperandrogenism symptoms. Adjuvant chemotherapy was administered in two patients with moderately/poorly differentiated Sertoli-Leydig cell tumors (Ki67 >30%). At one-month follow-up, testosterone normalized (0.5-2.6 nmol/L) in all patients with concomitant resolution of menstrual irregularities and improvement in virilizing symptoms.

CONCLUSION: OSCSTs are rare but clinically significant causes of hyperandrogenism, characterized by symptoms of HA such as oligomenorrhea, acne, hirsutism, and clitoromegaly. It has always been misdiagnosed due to overlapping features with PCOS and adrenal disorders. Early identification and complete surgical resection are critical for biochemical remission and symptom resolution.

INTRODUCTION

Ovarian sex cord-stromal tumors (OSCSTs) represent a rare and histologically heterogeneous group of ovarian neoplasms, comprising sex cord-like cells, stromal components, and variably differentiated luteinized cells, either alone or in combination (Ordulu *et al.* 2023). A subset of OSCSTs exhibits autonomous hypersecretion of androgens, resulting in virilization symptoms such as oligomenorrhea, acne, hirsutism, and clitoromegaly. The clinical and biochemical features of tumor-induced hyperandrogenism (HA) often overlap with those of other HA etiologies in females, including congenital adrenal hyperplasia (CAH), polycystic ovary syndrome (PCOS), and Cushing's syndrome (CS), posing significant diagnostic challenges and contributing to frequent misdiagnosis or delayed detection. In this study, we summarized the diagnostic and therapeutic outcomes of 14 female patients with androgen-secreting OSCSTs, aiming to enhance differential diagnosis and reduce misdiagnosis of these diseases.

SUBJECTS AND METHODS

Subjects

Fourteen female patients with pathologically confirmed OSCSTs were retrospectively enrolled in this study. All patients presented with elevated testosterone (T) levels on sex hormone testing and were hospitalized at the First Medical Center,Chinese PLA General Hospital between 2009 and 2024. Other causes of hyperandrogenism—including PCOS, CAH, and CS were systematically excluded prior to surgical intervention. The study protocol was approved by the Ethics Committee of the Chinese PLA General Hospital, and written informed consent was obtained from all participants.

Clinical data and methods

Clinical data collected from medical records included: (1) patient demographics and disease timeline (symptom onset to diagnosis); (2) clinical manifestations (menstrual status, virilization signs); (3) diagnostic testing (endocrine panels, imaging, pathology); (4) therapeutic interventions and surgical approaches; and (5) postoperative outcomes and immunohistochemical marker expression.

Hormonal evaluations included serum and urine measurements of T, dehydroepiandrosterone (DHEA), 17α-hydroxyprogesterone (17α-OHP), luteinizing hormone (LH), follicle-stimulating hormone (FSH), adrenocorticotropic hormone (ACTH), cortisol (F), urinary free cortisol (UFC), progesterone (P), and estradiol (E2). For patients with suspected CAH, a medium-dose dexamethasone suppression test was conducted. Baseline fasting blood samples were obtained at 8:00 AM to measure 17α-OHP, DHEA, ACTH, and T, followed by oral dexamethasone administration (0.75 mg every 6 hours for 5 consecutive days). Post-suppression measurements were repeated on the fifth day, with inhibition percentages of testosterone (T) and 17α-hydroxyprogesterone (17α-OHP) calculated to assess adrenal suppression. Imaging examinations consisted of pelvic transvaginal/abdominal ultrasound and contrast-enhanced computed tomography (CT) or magnetic resonance (MR).

Statistical analysis

All statistical analyses were performed with SPSS 22.0 software (IBM Corp., Armonk, NY, USA). Continuous variables were assessed for normality using the Shapiro-Wilk test. Data that followed a normal or approximately normal distribution were expressed as mean ± standard deviation (SD), while non-normally distributed data were reported as median (interquartile range, IQR. categorical variables were presented as frequencies and percentages.

RESULTS

Clinical Characteristics

The study cohort included 14 female patients with histologically confirmed ovarian steroid cell tumors. Patients ranged in age from 12 to 69 years at diagnosis (mean 34.4±20.1 years), comprising 6 adolescents (12-18 years, 42.9%), 4 reproductive women (19-45 years, 28.6%), and 4 postmenopausal women (>45 years, 28.6%). The mean duration from symptom onset to diagnosis was 2.3±1.4 years (range 1-6 years). Patients were hospitalized in endocrinology department (n = 7) or gynecology (n = 7) department. All adolescent and reproductive patients presented with menstrual disturbances (oligomenorrhea or secondary amenorrhea) accompanied by virilization signs, while postmenopausal patients exhibited virilization alone. The most prevalent manifestations were hirsutism (85.7%, 12/14), followed by voice changes (57.1%, 8/14) and clitoromegaly (57.1%, 8/14). Less common features included acne (28.6%, 4/14) and androgenic alopecia (21.4%, 3/14). Several diagnostic challenges were noted in this cohort. One adolescent patient had been misdiagnosed with congenital adrenal hyperplasia and treated with dexamethasone, while a reproductive-age patient was administered with Diane-35 for presumed PCOS. Another patient underwent artificial cycle treatment for menstrual irregularities before the correct diagnosis was established. Two other cases had previously incomplete hyperandrogenism evaluations at referring institutions (Table 1).

Laboratory Tests

Biochemical evaluation revealed significantly elevated T levels across the cohort, ranging from 3.90 to 119.6 nmol/L (normal female range: 0.2-2.6 nmol/L) (Table 1). To exclude CS, 8 patients underwent

detailed hypothalamic-pituitary-adrenal axis evaluation including diurnal cortisol rhythm assessment and urinary free cortisol measurements, all of which yielded normal results. Adrenal androgen profiling was performed in 8 cases, measuring DHEA and 17α-OHP levels. While most values fell within normal limits, three patients (Cases 1, 11, and 12) demonstrated mild elevations in these markers. These individuals subsequently underwent medium-dose dexamethasone suppression testing (0.75 mg every 6 hours for 5 days), which showed appropriate suppression of adrenocorticotropic hormone (ACTH) to <2.2 pmol/L but failed to suppress testosterone levels, excluding the underlying etiology of CAH (Table 2).

Imaging Findings

All patients underwent comprehensive imaging evaluation for ovarian lesion characterization. Gynecological color Doppler ultrasound identified ovarian masses in 12 patients (85.7%), including 5 transabdominal, 4 transvaginal, and 3 transrectal examinations. The ultrasound features demonstrated solid masses in 7 cases, mixed cystic-solid morphology in 3 cases, multilocular cystic lesion in 1 case, and diffuse ovarian enlargement without discrete mass formation in 1 case. Five patients underwent pelvic MRI for further characterization. Case 11 displayed a left ovarian lesion with predominant stromal components, showing restricted diffusion (high signal on DWI) and marked contrast enhancement. Case 12 had multiple round left adnexal lesions

Tab. 1. Clinicopathological features and prognosis of 14 cases of ovarian sex cord-stromal tumors

Case	Age (yrs)	Course (yrs)	Chief complaint		Initial consultation	T (nmol/L)	Imaging findings	Histopathology			Therapy	
			MAS	AME				Side	Diameter (cm)	Pathology	surgery	chemotheray
1	57	2	+	-	Endocrinology	119.6	+	L	4.3	SCT	TAH-BSO	-
2	39	5	-	+	Gynaecology	10.0	+	L	8.0	SLCT(poorly differentiated)	TAH-BSO	BEP
3	42	3	+	+	Endocrinology	25.39	-	R	0.7	LCT	USO	-
4	18	2	+	+	Endocrinology	15.24	+	R	5.5	SLCT (poorly differentiated)	USO	-
5	65	3	+	-	Gynaecology	11.74	-	R	1.5	LCT	TAH-BSO	-
6	13	1	+	+	Gynaecology	44.07	+	R	7.0	SCT-NOS	USO	-
7	31	1	+	-	Gynaecology	3.9	+	L	3.6	SLCT(moderately differentiated)	USO	-
8	18	1	+	-	Gynaecology	18.7	+	L	6.0	SCT-NOS	USO	-
9	58	5	+	-	Endocrinology	16.27	+	L	2.5	SL	BSO	-
10	69	3	+	-	Endocrinology	17.49	+	R	4.0	SL	TAH-BSO	-
11	31	6	+	-	Endocrinology	7.05	+	L	1.0	SL	USO	-
12	13	1	+	+	Endocrinology	14.51	+	L	4.0	SLCT(intermediate to high differentiation)	CYS	BEP
13	12	1	+	+	Gynaecology	16.1	+	R	9.0	JGCT	USO	-
14	15	2	+	+	Gynaecology	11.3	+	R	4.5	SLCT(moderately differentiated)	CYS	-

Abbreviations:MAS: masculinization;AME:amenorrhea;T:testosterone;L:left;R:right;SCT:sertoli cell tumor;LCT:leydig cell tumor;SLCT:sertoli-leydig cell tumor;SCT-NOS: steroid cell tumor,not otherwise specifie;SL:stromal luteoma;JGCT:juvenile granulosa cell tumor;TAH-BSO:total abdominal hysterectomy with bilateral salpingo-oophorectomy;USO:unilateral salpingo-oophorectomy;BSO:bilateral salpingo-oophorectomy;CYS:cystectomy;BEP:cisplatin+Bleomycin+incristine;etoposide+cisplatin+pingyangmycin.

Tab. 2. Medium-dose dexamethasone suppression test

Case	Time	$ACTH_{8AM}$(pmol/L)	F_{8AM}(nmol/L)	DHEA(ug/dl)	17α-OHP(ng/ml)	T(nmol/L)
Case1	pre-dose	3.9	447.5	100	2.3	119.6
	post-dose day 5	<1.1	<25.7	41.7	0.8	114.85
Case11	pre-dose	5.59	419	128	5.5	7.05
	post-dose day 5	<1.1	<25.7	27.5	2.48	6.33
Case12	pre-dose	5.79	305.63	36.1	1.873	10.98
	post-dose day 5	<1.1	<25.7	<15.0	3.12	9.71

Abbreviations:ACTH:adrenocorticotropic hormone;F:cortisol;DHEA:dehydroepiandrosterone;17α-OHP:17α-hydroxyprogesterone;T:testosterone.Model 2: Adjusted for age and sex

with slightly prolonged T1 and T2 signals, mild DWI hyperintensity, no signal loss on opposed-phase imaging, and absent contrast enhancement. Case 13 demonstrated a right adnexal cystic-solid mass with heterogeneous solid components (isointense to slightly T1-hyperintense) showing peripheral arterial-phase enhancement with progressive heterogeneous filling, while non-enhancing cystic components and an arcuate T2-hypointense solid portion were noted. Notably, two patients (14.3%) had no detectable ovarian lesions on comprehensive imaging with the diagnosis of OSCSTs established only through surgical exploration and subsequent pathological examination.

Treatment strategies

All 14 patients underwent definitive surgical management for OSCSTs. The excised tumors demonstrated considerable size variability, ranging from 0.7 cm to 9.0 cm in maximum diameter. Fertility-sparing procedures were performed in 10 patients (71.4%), consisting of unilateral oophorectomy (n = 7) or tumor enucleation (n = 3). The remaining 4 patients (28.6%), all postmenopausal, underwent total hysterectomy and bilateral salpingo-oophorectomy.

All 14 patients had unilateral ovarian masses with no evidence of distant metastasis. The tumors measured 0.7-9.0 cm in diameter (mean 3.98 ± 2.58 cm) and exhibited diverse morphological features including solid masses in 10 cases (71.4%), mixed cystic-solid in 3 cases (21.4%), and purely cystic lesion in 1 case (7.1%). Histopathological examination identified several tumor subtypes including Sertoli-Leydig cell tumors in 5 cases (35.7%), stromal luteomas in 3 cases (21.4%), Leydig cell tumors in 2 cases (14.3%), and nonspecific steroid cell tumors in 2 cases (14.3%). Rare subtypes included one juvenile granulosa cell tumor (7.1%) and one Sertoli cell tumor (7.1%). Immunohistochemical analysis demonstrated consistent expression of Inhibin-α (13 cases, 92.9%), with variable positivity for other markers: CK, Vimentin, and CD56 expressed in 7 cases (50.0%), CD99 in 6 cases (42.9%), and Calretinin in 4 cases (28.6%). The proliferation index, assessed by Ki67 staining, ranged from 15% to 75% in 5 cases (35.7%), indicating variable proliferative activity among the tumors (Table 1).

Follow-Up

Postoperative T levels (day 2: 0-6.51 nmol/L) normalized in all patients, accompanied by complete resolution of virilization symptoms within one month. Normal menstruation resumed within 3 months in all 6 reproductive-age patients who underwent fertility-sparing surgery.Two patients with aggressive Sertoli-Leydig cell tumors (Ki67>30%) received adjuvant chemotherapy (Case 2: cisplatin+bleomycin+vincristine and Case 12: etoposide +cisplatin +pingyangmycin). The 4 postmenopausal patients who underwent bilateral oophorectomy achieved full remission without additional treatment (Table 1).

DISCUSSION

HA in females results from excessive production of adrenal or ovarian androgens including T, DHEA, dehydroepiandrosterone sulfate (DHEA-S), dihydrotestosterone (DHT) and androstenedione (AND). While adrenal-derived androgens dominate DHEA-S production (>90%), ovarian sources account for most circulating T, making it a key biomarker for ovarian hyperandrogenism (Elhassan *et al.* 2018).

HA affects 6-12% of women, representing a prevalent endocrinometabolic disorder with significant health implications (Kushnir *et al.* 2015). Clinical manifestations characteristically include virilization such as hirsutism, clitoromegaly, acne and disappearance of female sexual characteristics such as oligomenorrhea and amenorrhea (Cussen *et al.* 2022). In our cohort,the mean age at diagnosis was 34.40 ± 20.14 years, with adolescents and reproductive women constituting 71.5% (10/14) of the cohort. The mean disease duration prior to diagnosis was 2.3 ± 1.4 years. Significantly elevated T levels (3.90–119.6 nmol/L) correlated with virilization symptoms (hirsutism: 85.7%; clitoromegaly: 57.1%),indicating OSCSTs as the underlying etiology.

The differential diagnosis of HA requires discrimination of adrenal and ovarian pathologies. CAH accounts for 10% of the causes of HA in females, mainly including 21-hydroxylase deficiency (21-OHD) and 11β-hydroxylase deficiency (11β-OHD). These are autosomal recessive diseases caused by gene mutations

encoding enzymes related to cortisol biosynthesis. Patients always have reduced cortisol synthesis and excessive adrenal androgen production, with clinical manifestations including classical and non-classical types (Auer *et al.* 2023). 21-OHD accounts for 90-95% of CAH cases, with non-classical 21-OHD being more common (Al-Rayess *et al.* 2025). Patients with atypical CAH may be asymptomatic or have mild symptoms, such as precocious puberty, rapid height growth, and advanced bone age. Adolescents and young women with atypical CAH exhibit features of androgen excess at puberty, such as hirsutism, oligomenorrhea or amenorrhea, and acne. The medium-dose dexamethasone suppression test can distinguish CAH from other causes of hyperandrogenemia such as adrenal and ovarian tumors. The principle of this test is that dexamethasone suppresses ACTH secretion in CAH patients, thereby reducing the secretion of 17α-OHP, DHEA, and T (Al-Rayess *et al.* 2025). In this study, three patients had significantly elevated baseline 17α-OHP and T levels, among whom one was misdiagnosed with CAH. They were treated with dexamethasone without any effects. Further medium-dose dexamethasone suppression test showed no significant decrease in T, excluding CAH.

CS is a clinical syndrome caused by excessive cortisol secretion from the adrenal cortex for various reasons. CS with HA is mainly caused by adrenal adenomas or carcinomas. Adrenal cortical carcinomas can autonomously secrete large amounts of active androgens and androgen precursor substances, often with DHEA-S >700 μg/dL (Nowotny *et al.* 2024). In addition to clinical manifestations of HA, it is often accompanied by symptoms and signs of CS such as plethoric appearance, proximal muscle weakness, hypertension, and hypokalemia. Diagnosis can be assisted by measuring cortisol rhythm, high and low-dose dexamethasone suppression tests, and adrenal imaging localization (Nowotny *et al.* 2022). Patients exhibiting neither clinical manifestations of CS nor abnormalities in cortisol rhythm or 24-hour UFC were excluded from the diagnosis of CS.

PCOS is the most common cause of HA in females, accounting for 89% and 30% of the causes of hyperandrogenemia in premenopausal and postmenopausal females, respectively. It is characterized by increased androgens, persistent anovulation, and polycystic ovarian change. The clinical manifestations include menstrual disorders, accompanied by hirsutism, acne, androgenetic alopecia, often with metabolic syndrome manifestations such as obesity, insulin resistance, and glucose-lipid metabolism disorders (Gungor *et al.* 2024). T levels usually do not elevate significantly, and virilization progresses slowly. In our study, one patient initially misdiagnosed with PCOS (T, 3.9 nmol/L) showed no metabolic syndrome features and got no effect with oral contraceptive therapy, underscoring the necessity of tumor exclusion in atypical presentations. Consequently, PCOS can only be established after rigorous exclusion of other underlying etiologies of HA.

Androgen-secreting ovarian tumors are a rare cause of HA in females. OSCSTs are the main type, accounting for 5%-8% of primary ovarian tumors, with an incidence of 0.09-0.10 per 100,000 per year (Al *et al.* 2021). OSCSTs with androgen-secreting function account for 0.2% of the causes of HA in females (Harzif *et al.* 2025). They are classified into pure sex cord tumors, pure stromal tumors, and mixed tumors according to cell origin.They include various pathological subtypes, mainly unilateral and benign lesions, with varying degrees of malignant potential (Hanley & Mosunjac, 2019). The pathogenesis of OSCSTs has not been clarified, and multiple studies have shown that their occurrence is often related to gene mutations. More than 90% of adult ovarian granulosa cell tumors (AGCTs) contain FOXL2 (c.402C>G, p.C134W) mutations. The C134W mutation leads to new chromatin remodeling activity of FOXL2, changing the binding sites of glucocorticoid receptors, thereby driving gene expression related to tumor growth (Welte *et al.* 2019). Gene abnormalities related to OSCSTs also include activating changes in AKT1 and GNAS genes detected in 60% and 30% of juvenile ovarian granulosa cell tumors (JGCTs), respectively (Barakizou *et al.* 2022). Such kinds of ovarian tumors can autonomously secrete androgens, with T levels often significantly elevated (usually >5.2 nmol/L) (Rojewska *et al.* 2022).

In this report a total of 14 patients were diagnosed with OSCSTs after excluding CAH, CS, and PCOS through clinical manifestations, biochemical and endocrine hormone tests. Ultrasound is the first choice for further tumor localization. When the tumors are smaller, it is usually difficult to be detected with ultrasound. So further imaging examinations such as ovarian CT, MRI, PET/CT are needed. If this disease is highly suspected but no imaging evidence is confirmed, ovarian venous blood sampling (SOAVS) (Tong & Tai, 2020) or even surgical exploration can confirmed performed for diagnosis. In this study, 85.7% of the patients (12/14) had clear ovarian masses confirmed by gynecological ultrasound. One patient showed only unilateral ovarian enlargement, and five underwent pelvic MR, with three confirming ovarian masses. Two patients had no imaging evidence and were finally diagnosed by surgical exploration, with postoperative pathology confirming both as LCT.

Surgical resection achieved rapid biochemical normalization (T, 0–6.51 nmol/L post-op day 2) and symptom resolution. Two patients with aggressive histology (Ki67 >30%) were administered with platinum-based chemotherapy, reflecting current management paradigms. These outcomes further confirm surgery as definitive therapy, with adjuvant treatment reserved for malignant features.

CONCLUSION

OSCSTs are rare but clinically significant causes of hyperandrogenism that are frequently misdiagnosed due to overlapping features with PCOS and adrenal disorders. Early identification and complete surgical resection are critical for biochemical remission and symptom resolution.

CONFLICT OF INTEREST

The authors declare that they have no conflict of interest associated with this manuscript.

FUNDING

This work was supported by the Fund Program: Key Medical Department of Joint Logistic Support Force (51511222A).

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