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**Case Report**

# Virilizing Leydig cell tumor of the ovary: from presentation to treatment - a case report

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## ABSTRACT

Leydig cell ovarian tumor is a rare sex cord-gonadal stromal tumor which constitutes less than 0.2% of ovarian tumors. As they produce testosterone, virilization is the most common presenting feature. This case report discusses a multiparous, 55 years old, postmenopausal lady who presented with complaints of excessive scalp hair loss and increased facial hair growth since two years. On examination she had androgenic alopecia, increased facial and midline body hair and clitoromegaly. Blood investigations done showed elevated S. testosterone. CT abdomen done showed no pathological lesions in the adrenal glands. MRI done in view of suspicion of Androgen secreting ovarian tumour showed mildly enlarged left ovary measuring 2×1.8 cm with altered signal intensity. She underwent total laparoscopic hysterectomy with bilateral salpingo-oophorectomy. Post operatively, histopathological examination was reported as Benign Leydig cell tumour of left ovary.

**Keywords:** Virilization, Leydig cell tumor, Androgen-secreting ovarian tumor, Hirsutism, Sex cord-stromal tumor

## INTRODUCTION

Leydig cell ovarian tumors are rare sex cord-stromal neoplasms, comprising less than 0.2% of all ovarian tumors.[1] These tumors arise from the Leydig cells, which are responsible for androgen production, primarily testosterone. Due to their androgen-secreting nature, Leydig cell tumors are often associated with virilization, presenting with clinical features such as hirsutism, androgenic alopecia, and clitoromegaly.[2] The rarity of these tumors, coupled with their nonspecific imaging characteristics, makes preoperative diagnosis challenging. A multidisciplinary approach involving gynecologists and endocrinologists is essential for accurate diagnosis and management.

## CASE REPORT

A 55-year-old multiparous woman, postmenopausal for 11 years, presented with a two-year history of excessive scalp hair loss and increased facial hair growth. She had no history of symptoms such as voice deepening, increased muscle mass, or other signs of significant virilization.

Her obstetric history included two term deliveries, and her past medical history was notable for type 2 diabetes mellitus, which was well-controlled with oral hypoglycemic agents. She had no significant surgical history or notable family history of malignancies.

On physical examination, the patient had a body mass index (BMI) of 31.7 kg/m$^2$. Clinical findings included: Androgenic alopecia, a Ferriman-Gallwey score of 14/36, indicating moderate to severe hirsutism, clitoromegaly measuring 1.5 cm and acanthosis nigricans.

Pelvic examination revealed an atrophic, mobile uterus with free bilateral fornices and no adnexal tenderness or palpable masses.

***Investigations***

*Laboratory findings*

*Serum testosterone:* 8 nmol/L (normal range: 0.5-2.4 nmol/L), indicating hyperandrogenism.

*DHEAS:* 300 mcg/dL (upper limit: 700 mcg/dL), within normal range.

*Cortisol:* 11 mcg/dL (normal range: 6-23 mcg/dL), indicating no adrenal insufficiency.

***Imaging studies***

*CT abdomen:* No significant findings in the adrenal glands. The adrenal glands appeared normal without any masses or adenomas.

*Ultrasound (TVS) pelvis:* Size of left ovary -2.7×2.5×2.5 cm (volume - 8.78 cc), size of right ovary -2.3×1.5×2.9 cm (volume -5.2 cc), demonstrated two hyperechoic areas in the left ovary, with the largest measuring 1.6×1.5 cm. Minimal vascularity was detected within the lesions.

*MRI pelvis:* Revealed a mildly enlarged left ovary (2.0×1.8 cm) with altered signal intensity, heterogeneously hyperintense on T2-weighted images and hypointense on T1-weighted images.

Adjacent fat planes were intact, and there was no evidence of pelvic lymphadenopathy. The right ovary appeared normal.

***Treatment***

Given the high clinical suspicion of an androgen-secreting ovarian tumor, the patient underwent total laparoscopic hysterectomy with bilateral salpingo-oophorectomy. During the procedure, the left ovary was noted to be enlarged, with no cystic surface lesions. The right ovary appeared normal. The uterus showed subserosal as well as the intramural fibroids without the other significant pathology.

***Pathology***

Histopathological examination confirmed the presence of a benign Leydig cell tumor in the left ovary. The tumor was characterized by uniform, polygonal cells with abundant eosinophilic cytoplasm. Reinke's crystalloids, which are dense, needle-like structures found in approximately 30-40% of cases, were observed within the tumor cells.

Immunohistochemistry revealed positivity for inhibin, calretinin, and CD56, supporting the diagnosis of Leydig cell tumor. There was no capsular involvement, and the surrounding ovarian tissue was unaffected.


**Figure 1 (A and B): Androgenic alopecia i. e., male pattern baldness.**

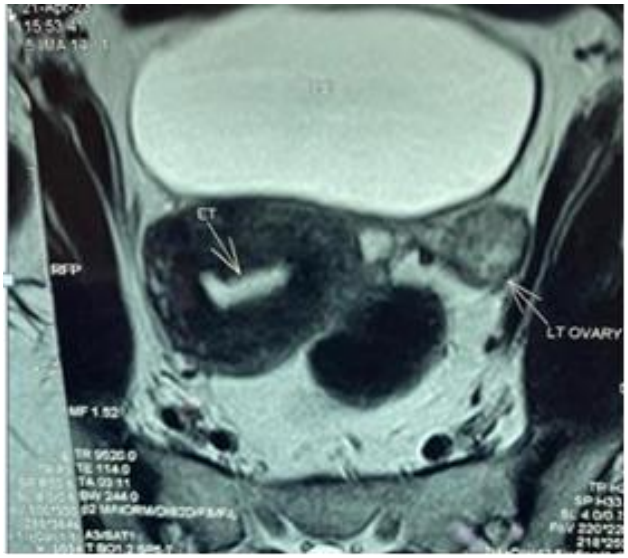


**Figure 2: MRI pelvis showing shows altered signal intensity of left ovary.**

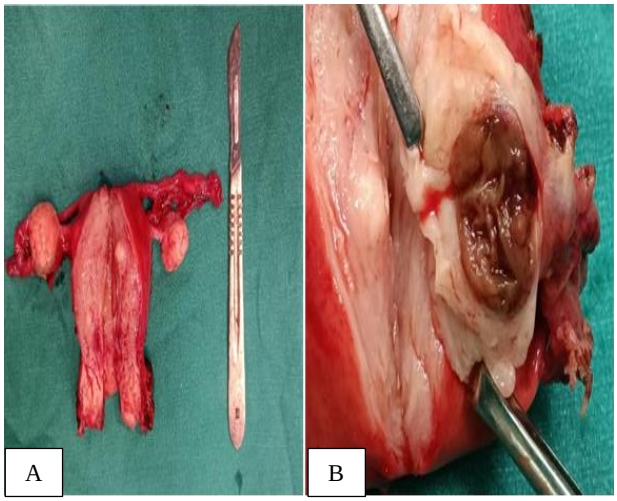


**Figure 3 (A and B): Hysterectomy specimen showing bulky left ovary and cut section showing lesion in the left ovary.**

## DISCUSSION

Leydig cell tumors are the most common non-germ cell neoplasms of the ovary, typically occurring between the third and fourth decades of life. They are often associated with excess androgen production, leading to virilization in 70-85% of patients.[3] Symptoms of virilization include hirsutism, clitoromegaly, deepening of the voice, and receding hairlines. Diagnosis is often challenging due to the small size of these tumors and the nonspecific nature of imaging findings.[4] Imaging modalities such as transvaginal ultrasound (TVS), MRI, and CT scans are instrumental in the initial evaluation. TVS can provide valuable information regarding the size, location, and vascularity of the tumor, while MRI offers detailed anatomical characterization and helps differentiate between malignant and benign lesions.[5] In this case, the MRI findings of heterogeneously hyperintense and hypointense areas were suggestive of a benign lesion, correlating with the histopathological findings.

Histological examination is definitive for diagnosis. Leydig cell tumors are distinguished by the presence of lipid-laden cells and Reinke's crystalloids, which are diagnostic features.[6] Immunohistochemistry can further confirm the diagnosis by identifying markers such as inhibin, calretinin, and CD56.

A high index of suspicion, combined with clinical, biochemical, and imaging findings, is necessary for an accurate diagnosis.[7] The treatment of choice for Leydig cell tumors is surgical excision. In reproductive-aged women, unilateral salpingo-oophorectomy is often sufficient, while older patients or those with larger tumors may require total hysterectomy with bilateral salpingo-oophorectomy. The prognosis for benign Leydig cell tumors is excellent, with a low recurrence rate and a 5-year survival rate exceeding 90%.[3,5]

## CONCLUSION

Leydig cell ovarian tumors, though rare, should be included in the differential diagnosis of postmenopausal women presenting with virilization. Early recognition through a multidisciplinary approach involving gynecologists and endocrinologists is essential for optimal management. Surgical excision remains the cornerstone of treatment and offers an excellent prognosis for benign cases.

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