Open Access
Volume 7 Issue 1
Case Report Article

International Journal of Women's Health care and Gynecology
ISSN: 2694-4081

PUBTEXTO

A Case of Idiopathic Post-Menopausal Hirsutism Mimicking an Androgen-Producing Right Ovarian Mass

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Article Info

Article History:
Received: 05 July, 2025
Accepted: 08 July, 2025
Published: 11 July, 2025

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DOI:
https://doi.org/10.36266/IJWHG/171

Abstract

A 62-year-old female presented to the endocrinology outpatient department for facial hirsutism (Ferriman-Gallwey score 5/36) of 5 years duration. She had no other virilizing features. In the background, she had Type 2 diabetes of 8 years duration, reasonably controlled (HbA1C 7.4%) on twice daily premixed insulin with Metformin 1000 mg/day. Her workup revealed elevated serum testosterone (0.80 ng/mL) with normal levels of other hormones. USG pelvis showed a large (16x12 cm) mixed echogenic right ovarian cyst with a solid component. With suspicion of an androgen-producing right ovarian mass, she underwent surgery for removal of the mass. Postoperative histopathology showed nests of transitional epithelium with squamous metaplasia, consistent with a Brenner tumour. In subsequent follow-up, 3 months after surgery, her hirsutism was the same, with serum testosterone in a similar elevated range. Even though Brenner tumour is rarely reported to produce increased androgen, in this case, it appears coincidental. Mild hirsutism, absence of virilizing features, mildly elevated serum testosterone, and absence of improvement after surgery indicate this case as idiopathic post-menopausal hirsutism rather than an androgen-producing ovarian tumour.

Keywords: Brenner tumour; Squamous metaplasia; Testosterone

Introduction

Hirsutism is a condition in which there is unwanted development of hair in androgen-dependent areas in a female. Hyperandrogenism in postmenopausal women may arise from either ovarian or adrenal sources and can pose a challenging diagnostic dilemma. Polycystic ovarian syndrome (PCOS) is the most common cause of hyperandrogenism in the postmenopausal period, with a prevalence between 8 and 13% (18). However, other causes such as ovarian hyperthecosis, androgen-producing adrenal tumour, androgen-producing ovarian tumour, non-classic congenital adrenal hyperplasia, and Cushing's syndrome should be ruled out if the patient is having signs of severe hyperandrogenism such as frontal baldness, deepening of voice, breast atrophy, clitoromegaly [10]. Testosterone levels above 5 nmol/L are associated with virilization and require prompt investigation to rule out an androgen-producing tumour initially [10]. Brenner tumour is a benign tumour of the ovary, though rarely, it can be associated with excess androgen production [12-14].

Case Report

A 62-year-old postmenopausal woman was referred to the endocrinology outpatient clinic with complaints of unwanted hair growth over the upper lip and chin for the past 4-5 years. The hairs were initially thin but gradually became coarse and thicker over time, requiring removal by shaving every 4-5 days. She reported no other signs of hyperandrogenism, such as acne, frontal balding, voice deepening, breast atrophy, or clitoromegaly. The hirsutism was primarily a cosmetic concern. Following advice from a relative regarding the possibility of hormonal imbalance, she sought endocrinological consultation. There was no history of recent weight gain, reddish-purple striae, or easy bruising. She was multiparous with four children and had attained menopause at the age of 51. Her medical history included type 2 diabetes mellitus for 20 years, managed with biphasic insulin (20 units before breakfast, 12 units before dinner), metformin 1000 mg twice daily, and sitagliptin 100 mg once daily. She was also on telmisartan 40 mg daily for hypertension, which was well controlled. On examination,

she weighed 77 kg and had a BMI of 38 kg/m² with a blood pressure of 128/76 mmHg. The general examination revealed signs of insulin resistance, including acanthosis nigricans (grade 3) and skin tags. She had male pattern terminal hair growth over the upper lip (FGS 2) and chin (FGS 3). Systemic examination was otherwise unremarkable. We advised her to have her serum testosterone level checked, which came out to be 0.8 ng/mL (normal: 0.029-0.4) with a normal other hormonal profile (Table 1). Ultrasound of the abdomen was advised to rule out any ovarian or adrenal etiology which showed hepatomegaly with a grade 1 fatty liver and a large, well-defined cyst with internal echoes, noted in the right ovary, measuring 19.9× 16.8 cm, with a normal appearance of the left ovary (Figure 1). Given the size of the mass, malignancy was suspected. Tumour marker profile revealed a significantly elevated CA 19-9 level of 813 U/mL (normal: 0-37 U/mL), a mildly elevated CEA of 3.63 ng/mL (normal: <3 ng/mL), and a normal CA-125 level of 20 U/mL (reference: 0-35 U/mL). Elevated levels of CA 19-9 can be observed in ovarian tumours. She underwent a whole-body FDG PET-CT scan to rule out any metastasis and to determine the extent of the disease, which showed a large hypodense abdominopelvic cystic lesion in the right ovary of size 18 cm ×19.4×15 cm with an FDG avid mural solid soft tissue component in the postero-inferior aspect of the lesion on the left side, measuring 5cm ×5.9 cm (SUV max 5.8) (Figure 2). She underwent ovarian cystectomy with infracolic omentectomy. The surgery was uneventful. Her histopathology report showed nests and lobules of transitional epithelial cells admixed with fibrous stroma (Figure 3). The cells contained oval to elongated nuclei with nuclear grooves, accompanied by clear cytoplasm, without nuclear atypia or mitotic features consistent with a Brenner Tumour (Figure 4). Despite surgery, her hyperandrogenic symptoms persisted. She was subsequently started on spironolactone and referred to a dermatologist for the laser therapy.

Table 1: *Normal hormonal and biochemical profile.*

Parameter	Patient's Value	Reference Interval (postmenopausal)
FSH	42.3	30-150
LH	18	15-65
Total Testosterone	0.8	0.029-0.4 ng/ml
DHEAS	85	10-130 µg/dl
CA 19-9	813	0–37 U/mL
CEA	3.63 ng/ml	<3 ng/ml
CA-125	20	0-35 U/mL
Fasting plasma glucose	104	80-130 mg/dl
Post-breakfast glucose	178	< 180 mg/dl
HbA1c	7.4	< 7.0%

Discussion

Hirsutism in postmenopausal women is uncommon. In the postmenopausal state, ovarian and adrenal androgen production typically declines, and the development of hirsutism with elevated testosterone levels raises strong suspicion for an androgen-secreting neoplasm or ovarian hyperthecosis [1,2].

The differential diagnosis of hyperandrogenism in postmenopausal women includes androgen-secreting ovarian tumours (such as Sertoli-Leydig cell tumours and steroid cell tumours), adrenal tumours, and ovarian stromal hyperthecosis [2,3]. Rarely, exogenous androgen exposure or severe insulin resistance may contribute, although these are less common in this age group [4].

Hirsutism caused by ovarian tumours accounts for approximately 1% of all cases of hirsutism [5]. Androgen excess due to ovarian tumours may cause deepening of the voice, clitoromegaly, facial hair growth, decreased breast size, and amenorrhea. In our case, the patient initially presented with hirsutism without features of virilization. The progressive course of the hirsutism with elevated serum testosterone level and no prior history of menstrual irregularities strongly indicated the possibility of an adrenal or ovarian source of androgen production rather than PCOS.

Considering the patient's normal cortisol rhythm, normal DHEAS levels, and absence of cushingoid features, Cushing's syndrome and androgen-producing adrenal tumours were ruled out. The normal menstrual history prior to menopause and absence of hyperandrogenism during reproductive years argued against a delayed diagnosis of congenital adrenal hyperplasia or polycystic ovary syndrome.

Certain medications (e.g., anabolic steroids, transfer of topical testosterone gels, dexamethasone, phenytoin, valproate, minoxidil, cyclosporine) have been linked with hirsutism [6]. However, our patient did not have a history of medication use apart from standard antidiabetic and antihypertensive therapy.

In the present case, detection of an ovarian mass on imaging, together with hyperandrogenism, led to the decision for surgical intervention, given the possibility of an androgen-secreting tumour. Malignancy and metastasis were considered less likely, given the FDG PET-CT findings and normal biochemical tumour markers, except for elevated CA 19-9 levels. CA 19-9 has been cited as a more specific marker than CA 125 in advanced ovarian malignancy although elevated levels may also occur in benign pancreatobiliary, hepatic, gynaecologic diseases, and diabetes mellitus [7,8].

In small studies of women with hyperthecosis or ovarian tumours, GnRH suppression testing resulted in gonadotropin suppression as well as a ≥ 50% reduction in testosterone levels in both groups [9]. Selective ovarian or adrenal venous catheterization and hormonal sampling may be a method for diagnosing and localizing tumours. However, the procedure is invasive, and successful catheterization could be challenging [10]. In our case, GnRH testing and ovarian vein sampling were not performed initially due to the presence of an identifiable ovarian mass.

Histopathology revealed features consistent with a Brenner tumour, which constitutes about 2% of all ovarian epithelial neoplasms, with a mean age of diagnosis around 50 years. The majority are benign (99%) and hormonally inactive [11]. When hormonally active, Brenner tumours are typically associated with oestrogen rather than androgen production [12]. Very few reports describe Brenner tumours associated with androgen excess, were stromal elements, rather than the epithelial component, may be the source [13]. Synchronous occurrence of rare tumours, such as Leydig cell tumours and Brenner tumours, has also been described in virilised postmenopausal women [14].

In our case, postoperative persistence of hyperandrogenism indicated that the tumour was not the source, or that another androgen-producing focus (such as ovarian hyperthecosis or an occult tumour) was present. Persistent hyperandrogenism after surgery underscores the complexity of localizing androgen excess in postmenopausal women. Bilateral ovarian hyperthecosis, which can be microscopic or macroscopic, may coexist with other ovarian lesions and may be missed unless both ovaries are removed and carefully examined histologically [15]. Adrenal sources were considered less likely given the absence of radiological abnormalities and the predominant elevation of testosterone over DHEAS.

Medical management remains an important strategy in such cases. Antiandrogen therapy, including spironolactone, cyproterone acetate, or finasteride, can reduce hair growth and other androgenic effects [16]. Cosmetic treatments, such as laser or electrolysis, may provide additional benefits. GnRH analogues could be considered if residual ovarian androgen production is suspected and surgery is not an option [17].

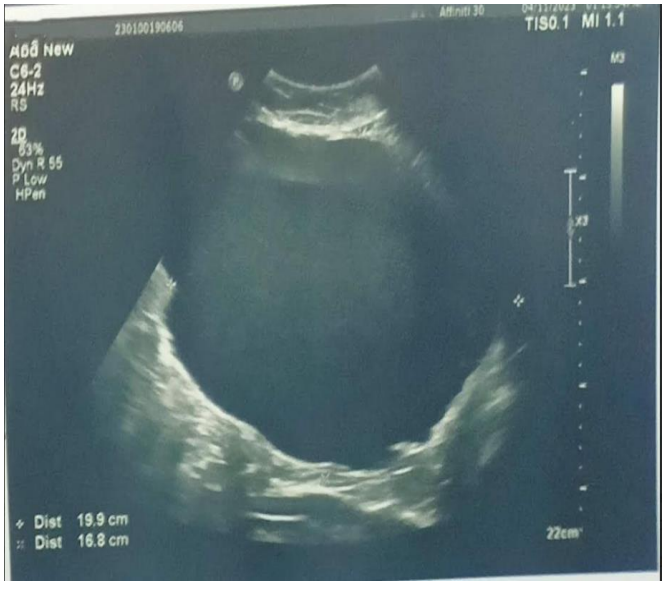

Figure1: *Transabdominal pelvic ultrasound showing a large, well-defined anechoic cystic lesion measuring approximately 19.9 × 16.8 cm, occupying the pelvis.*

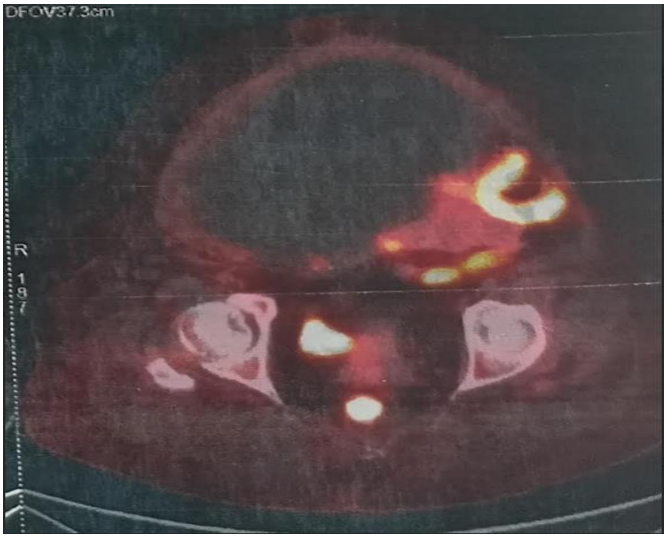

Figure 2: *Axial PET-CT fusion image showing a large hypodense abdominopelvic cystic lesion, arising from the right ovary, measuring approximately 18 × 19.4 × 15 cm. A metabolically active FDG-avid mural solid soft tissue nodule is seen along the posteroinferior aspect of the lesion on the left side, measuring 5 × 5.9 cm with a maximum standardized uptake value (SUV max) of 5.8*

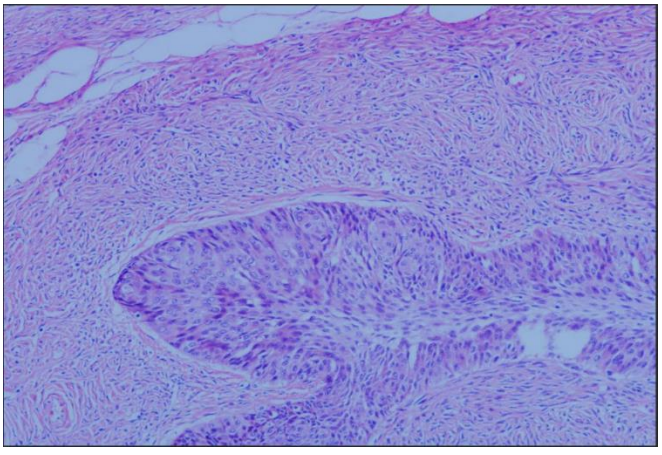

Figure 3: *100x low-power microscopic images showing nests and lobules of transitional epithelial cells interspersed with fibromatous stroma.*

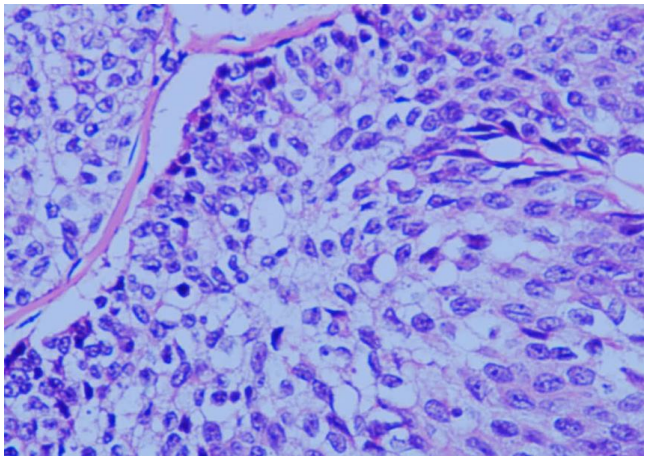

Figure 4: *400x high-power image displaying cells with oval to elongated nuclei that have nuclear grooves, accompanied by clear to eosinophilic*

cytoplasm. No nuclear atypia is observed, and there is no evidence of stromal invasion. This is suggestive of a benign Brenner tumour.

Conclusion

This case highlights the diagnostic and therapeutic challenges of postmenopausal hyperandrogenism. It illustrates the importance of comprehensive preoperative evaluation and the role of medical therapy when surgery does not resolve hyperandrogenism. A multidisciplinary approach involving endocrinology, gynaecology, dermatology, and pathology is essential for optimal management.

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