A Malignant Androgen-Secreting Ovarian Tumour Misdiagnosed as Polycystic Ovary Syndrome: A Case Report

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Abstract

Background: Hyperandrogenism in women most commonly arises from polycystic ovary syndrome (PCOS). However, rare androgen-secreting ovarian tumours may present with overlapping symptoms, posing a diagnostic challenge and contributing to delayed diagnosis.

Methods: A 35-year-old woman presented with a 5-month history of irregular menses, initially managed as PCOS without improvement. Evaluation included history, examination, hormone assays, pelvic ultrasound, MRI and histopathology.

Results: Examination revealed virilization including frontal balding, severe hirsutism, voice deepening, and clitoromegaly. Laboratory results showed significantly elevated total testosterone of 257 ng/dL (ref 15–70 ng/dL) and free testosterone of 3.2 ng/dL (ref 0.1–1.9 ng/dL), with elevated androstenedione of 3.7 ng/mL (ref 0.3-3.3 ng/mL) but normal DHEAS, FSH, LH and 17-OHP. Pelvic ultrasound/MRI identified a 2×4 cm right ovarian mass. Histopathological examination confirmed a malignant androgen-secreting ovarian steroid cell tumour, not otherwise specified (NOS).

Conclusion: Progressive virilization and significantly elevated testosterone in presumed PCOS should raise suspicion for alternative aetiologies including androgen-secreting ovarian tumours which are rare but are clinically significant due to their potential malignancy.

Case Background

Hyperandrogenism can result from a variety of medical conditions. Polycystic ovary syndrome (PCOS) is the most common cause, accounting for approximately 75% of cases (1). Patients with PCOS typically present with hirsutism, irregular menstruation, and hair loss reflecting chronic hormonal imbalance, among other symptoms (2).

Although PCOS is common, the presence of rapidly progressive symptoms of virilization should prompt evaluation for alternative aetiologies like androgen-secreting tumours (3). Androgen-producing ovarian and adrenal tumours are rare, representing only 0.2% of cases of hyperandrogenism (1). Androgen-secreting ovarian tumours are mostly benign representing two-thirds of cases but there are rare cases of malignant tumours, highlighting the rarity of this case presentation (4,5).

Steroid cell tumours can be further divided into three subtypes based on their cell of origin: stromal luteoma which arises from ovarian stroma, Leydig cell tumour which arises from ovarian hilar Leydig cells, and steroid cell tumours not otherwise specified (NOS) where their definitive histological lineage cannot be determined (5).

Case Details

A 35-year-old female presented to the endocrinology clinic with irregular menstruation for the past 5 months. The patient was initially diagnosed with Polycystic Ovary Syndrome (PCOS) by multiple family medicine physicians. The patient was also given treatment for PCOS but no significant improvement was noted.

On history taking and clinical examination, the patient was found to be stable and afebrile with normal vitals. The patient reported changing and deepening of her voice for the past 3 months. There was noted frontal balding with hair loss in other parts of the scalp and generalised hirsutism (body hair growth with a male pattern) which the patient said progressively worsened in the last 2 months (estimated Ferriman-Gallwey score of 24) (1). The patient also reported social embarrassment and distress as a result of her progressive hirsutism and how it negatively affects not only her self-esteem but also quality of life. Moreover, on genital examination the patient was found to have clitoromegaly.

The patient was requested to get laboratory tests. Laboratory investigations revealed total testosterone of 257 ng/dL (ref 15-70 ng/dL), free testosterone of 3.2 ng/dL (ref 0.1–1.9 ng/dL), androstenedione of 3.7 ng/mL (ref 0.3-3.3 ng/mL) and normal levels of dehydroepiandrosterone sulfate (DHEA-S), serum cortisol, adrenocorticotropic hormone (ACTH), follicle stimulating hormone (FSH), luteinizing hormone (LH), 17-hydroxyprogesterone (17-OHP).

Given the severity of virilization and the markedly elevated testosterone, an androgen-secreting tumour was suspected. Normal DHEA-S, ACTH, cortisol, FSH, LH, and 17-OHP helped exclude adrenal and other endocrine causes.

Pelvic ultrasound then revealed a 2 × 4 cm right ovarian mass, which was further characterized on MRI, with no other pelvic pathology identified. A laparoscopic biopsy retrieved tissue for histopathology, which showed steroid cell tumour NOS: lipid-laden polygonal cells with >2 mitoses/10 high-power fields, necrosis, grade 2 nuclear atypia, and haemorrhage, diagnostic of malignancy. Clinical findings, and diagnostic assessment supported the diagnosis of a malignant androgen-secreting ovarian steroid cell tumour- not otherwise specified (NOS), classified as FIGO stage IA. The patient then underwent laparoscopic right salpingo-oophorectomy and was referred to the oncology department for surveillance. At 1-month follow-up, serum testosterone normalized, with no adjuvant chemotherapy required. Moreover, hirsutism and voice changes are expected to improve over 6-12 months.

Consent was obtained from the patient for the publication of this case report and accompanying clinical details.

Discussion

Hyperandrogenism in women is most commonly caused by PCOS, which accounts for the majority of cases. PCOS typically presents with chronic anovulation, mild to moderate hyperandrogenism, hirsutism, and irregular menstruation, making it a frequent initial diagnosis in women with androgen excess (2,6). The repeated diagnosis and treatment of PCOS was made because the patient presented with menstrual irregularities and generalized hirsutism, and the small right ovarian mass may have been mistaken for a PCOS-related cyst due to its small size and location. The initial diagnosis of PCOS likely reflected anchoring bias toward a far more common cause of hyperandrogenism, despite the presence of progressive virilization and markedly elevated testosterone levels. However, androgen-secreting tumours should always be considered in the differential diagnosis when symptoms are progressive or atypical, as delayed recognition can lead to worsening virilization and tumour progression, particularly in malignant cases (3).

Several red flags in this case were inconsistent with PCOS.

These include virilization such as clitoromegaly, frontal balding, deepening of voice, and rapidly progressive hirsutism which are uncommon in PCOS but are strongly suggestive of pathological androgen excess. In addition, clinical recommendations suggest that total testosterone levels exceeding 150-200 ng/dL should raise suspicion for an androgen-secreting tumour rather than PCOS (3,6). Moreover, normal levels of FSH and LH are less consistent with PCOS, which is often associated with elevated LH due to altered gonadotropin-releasing hormone (GnRH) pulsatility (3,6). Recognition of these features is critical as they help distinguish tumour-related hyperandrogenism from more common conditions.

Laboratory and imaging investigations are essential to localise whether androgen excess originates in the ovaries or in the adrenal gland since androgens are secreted by both (3). In this case the patient had significantly elevated free testosterone and total testosterone with elevated androstenedione, a biochemical pattern strongly suggestive of an androgen-secreting tumour (6). Moreover, her normal adrenal markers including DHEA-S, serum cortisol, ACTH, and 17-OHP confirm that hyperandrogenism is of an ovarian origin. Normal cortisol and ACTH also help rule out Cushing syndrome, an important differential diagnosis for hirsutism and irregular menstruation (5). In addition, pelvic imaging, including both the ultrasound and MRI, revealed a right ovarian mass and further ensured the patient's adrenal glands were normal. Overall, the biochemical, radiological, and histopathological findings were consistent with a malignant androgen-secreting ovarian steroid cell tumour - NOS. These represent characteristic findings of androgen-secreting ovarian tumours, as described in published clinical reviews, including testosterone levels >200 ng/dL, rapidly progressive virilization, unilateral solid ovarian mass, elevated androstenedione and normal adrenal markers (DHEAS, 17-OHP) (3,5).

Considering the psychosocial aspect of not only androgen-secreting tumours but hyperandrogenism in general, many of the female patients' symptoms can significantly affect their mental health (7).

In this case the patient reported severe embarrassment from progressive changes in her appearance and voice, causing social discomfort that led her to avoid social and family gatherings.

She also experienced workplace anxiety when her colleagues started noticing her deepening voice. Thus the prolonged period of misdiagnosis contributed not only to delayed tumour recognition but also to psychological distress with social withdrawal and reduced quality of life, underscoring the importance of holistic patient management. Long-term exposure to these changes can negatively affect the patient's mental health by exacerbating things like depression, lowered self-esteem and anxiety (7). Following surgery and testosterone normalisation, hirsutism regression and psychological counselling are expected to improve her quality of life and help restore her self-confidence.

Conclusion

This case underscores the importance of how misdiagnosis and delayed recognition of androgen-secreting tumours can lead to prolonged virilization, tumour progression and psychological distress. Therefore physicians should maintain a high index of suspicion when a patient shows minimal improvements with PCOS treatment and presents with rapidly progressive atypical symptoms. Moreover, comprehensive hormonal, imaging and histopathological assessment are crucial to differentiate PCOS from rare but clinically significant ovarian tumours. This would result in early surgical intervention and optimised patient outcomes as it can prevent mismanagement and improve hormonal and oncological prognosis (5).

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