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(CASE REPORT)

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Ovarian stromal hyperplasia: An uncommon cause of postmenopausal hyperandrogenism, case report and a brief review of literature

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Abstract

Ovarian stromal hyperplasia (OSH) is a non-neoplastic proliferation of ovarian stromal cells associated with hyperandrogenism. This condition mainly affects perimenopausal and postmenopausal women, making diagnosis challenging due to similar clinical symptoms. We present the case of a 61-year-old postmenopausal woman with progressive virilization, including hirsutism, voice deepening, and clitoromegaly. Biochemical evaluation revealed elevated serum testosterone with normal adrenal androgens, suggesting an ovarian origin. Subsequent cross-sectional imaging demonstrated bilateral ovarian enlargement without a distinctive mass, raising suspicion for a diffuse stromal process rather than a focal neoplasm. A gonadotropin-releasing hormone agonist trial was used as part of the diagnostic workup; ultimately, the patient underwent bilateral salpingo-oophorectomy. Histopathological examination demonstrated diffuse and nodular proliferation of luteinized stromal cells without cytologic atypia, consistent with ovarian stromal hyperplasia. Postoperatively, serum androgen levels were normalized, and the patient reported clinical improvement. This case emphasizes consideration of ovarian stromal hyperplasia in postmenopausal women who present with hyperandrogenism, particularly when laboratory findings suggest an ovarian source despite inconclusive imaging.

Keywords: Ovarian stromal hyperplasia; Immunohistochemical; Estrogen; Progesterone; Testosterone; Luteinizing hormone; Follicle-stimulating hormone (FSH).

1. Introduction

Ovarian stromal hyperplasia (OSH) is a rare non-neoplastic disorder characterized by diffuse proliferation of ovarian stromal cells, predominantly affecting postmenopausal women. [1] It represents an uncommon cause of hyperandrogenism, which may manifest as varying degrees of virilization. Although benign, the clinical presentation can mimic androgen-secreting ovarian or adrenal tumors necessitating prompt and through evaluation. [2]

Postmenopausal hyperandrogenism is a clinical condition that occurs in women due to the overproduction of androgens from the ovaries or adrenal glands. [3] Virilization in form of clitoromegaly, deepening of the voice, or severe hirsutism, raises concern for neoplastic etiologies and requires immediate evaluation. Nonetheless, distinguishing neoplastic from

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non-neoplastic causes remains challenging, particularly when biochemical overlap and imaging fail to reveal an identifiable lesion. [4]

Ovarian stromal hyperplasia and its related conditions (i.e., ovarian hyperthecosis) are underrecognized causes of postmenopausal hyperandrogenism, with few cases reported in the literature. [5] OSH and hyperthecosis are related, non-neoplastic disorders that produce androgens and primarily affect postmenopausal women. OSH involves diffuse or nodular overgrowth of the ovarian stroma, whereas hyperthecosis represents a more severe form, marked by stromal proliferation along with the presence of luteinized stromal cells. Although the two conditions are not identical, they exist along a shared clinical spectrum. [6] One area where this results in diagnostic uncertainty and variability in treatment options is determining not only the role of hormone-suppression tests but also the timing of surgical intervention.

We present this case to highlight the importance of considering ovarian stromal hyperplasia when investigating severe postmenopausal hyperandrogenism, the challenges in diagnosis, and the need for a multidisciplinary approach in determining appropriate management.

2. Case Presentation

2.1. Clinical Presentation and Initial Investigations

A 61-year-old postmenopausal woman, who had undergone menopause at age 52, presented to her gynecologist with an 18-month history of progressively worsening virilization. She initially noted frontotemporal hair thinning, followed by the gradual development of coarse facial hair on the upper lip and chin, along with increased terminal hair growth on her forearms and abdomen. Approximately six months prior to presentation, her husband observed a subtle deepening of her voice. The patient also reported clitoral enlargement, which she had attributed to normal aging and had been reluctant to disclose due to embarrassment. Her medical evaluation had previously been limited to a primary care visit for fatigue and mild weight gain, during which thyroid function testing was normal, and her symptoms were attributed to aging. She sought further evaluation after her daughter expressed concern that these changes were not consistent with normal aging. She denied any use of exogenous androgens, anabolic steroids, or over the counter or herbal supplements.

On physical examination, her vital signs were within normal limits, and her body mass index was 34 kg/m^2. She demonstrated mild temporal hair recession, coarse, dark terminal hair on the face, an elevated Modified Ferriman-Gallwey–adapted (mFG) score for a postmenopausal woman, and clitoromegaly measuring approximately 2.5 cm. Mild seborrhea was also noted. There was no evidence of acanthosis nigricans. Abdominal examination was unremarkable, with no palpable masses or tenderness. Her past medical history was significant for well-controlled type 2 diabetes mellitus managed with metformin and managed hypertension with an ACE inhibitor. There was no personal or family history of ovarian, adrenal, or endocrine malignancies.

Laboratory evaluation revealed a markedly elevated total testosterone level of 285 ng/dL, with significantly elevated free testosterone. Dehydroepiandrosterone sulfate (DHEA-S) levels were within normal limits, arguing against an adrenal source of androgen excess. Androstenedione was mildly elevated. Gonadotropin levels (LH and FSH) were suppressed, an unusual but supportive finding suggesting gonadotropin-dependent ovarian androgen production. Estradiol was in the low-normal range. Additional endocrine evaluation, including 17-hydroxyprogesterone, cortisol with adrenocorticotropic hormone stimulation testing, and human chorionic gonadotropin, was all within normal limits, effectively excluding congenital adrenal hyperplasia, Cushing syndrome, and hCG-mediated processes.

2.2. Imaging Findings and Differential Diagnosis

Transvaginal pelvic ultrasound demonstrated bilaterally enlarged ovaries with diffusely increased echogenicity relative to expected postmenopausal morphology, without evidence of discrete masses or follicles. This subtle finding raised suspicion of a diffuse stromal process. Subsequent magnetic resonance imaging (MRI) of the pelvis with contrast revealed bilateral ovaries with homogeneous low T2 signal intensity and minimal enhancement, features more consistent with stromal proliferation than with focal neoplasm. No adnexal masses were identified. Computed tomography (CT) of the abdomen and pelvis demonstrated normal adrenal glands without enlargement or focal lesions and no retroperitoneal lymphadenopathy. Considering these findings, an adrenal source was considered unlikely.

The differential diagnosis included ovarian stromal hyperplasia or hyperthecosis, which can present with diffuse bilateral ovarian involvement and hyperandrogenism in postmenopausal women. However, the possibility of an

androgen-secreting ovarian neoplasm, such as a Leydig cell tumor or other sex cord-stromal tumor, could not be fully excluded, particularly given the markedly elevated testosterone levels. Other considerations included an occult adrenal tumor, late-onset congenital adrenal hyperplasia, Cushing syndrome, and exogenous androgen exposure. However, the latter diagnoses were rendered less likely based on laboratory and clinical findings.

2.3. Multidisciplinary Tumor Board Discussion & Management Plan

The case was presented at a multidisciplinary tumor board including specialists in gynecologic oncology, endocrinology, radiology, and pathology. The discussion focused on determining whether the process was neoplastic or non-neoplastic, the necessity of surgical intervention, and the optimal management strategy. The absence of a discrete lesion on imaging and the presence of bilateral ovarian involvement favored non-neoplastic stromal etiology. Nonetheless, the board acknowledged that small androgen-secreting tumors, particularly Leydig or steroid cell tumors, may evade detection on imaging and cannot be excluded solely on biochemical grounds.

Endocrinology proposed a diagnostic and therapeutic trial of a gonadotropin-releasing hormone agonist to suppress ovarian androgen production. Suppression of testosterone levels following treatment would support an LH-dependent ovarian source. The tumor board agreed with this approach as a useful interim step prior to definitive management. Given the patient's postmenopausal status, comorbidities, and high likelihood of bilateral disease, the consensus recommendation was to proceed with laparoscopic bilateral salpingo-oophorectomy following completion of the GnRH agonist trial, with intraoperative frozen section evaluation if any gross abnormalities were identified. Preoperative cardiology clearance was advised, considering her diabetes and hypertension.

2.4. Pathology, Immunohistochemistry (IHC) & Special Studies

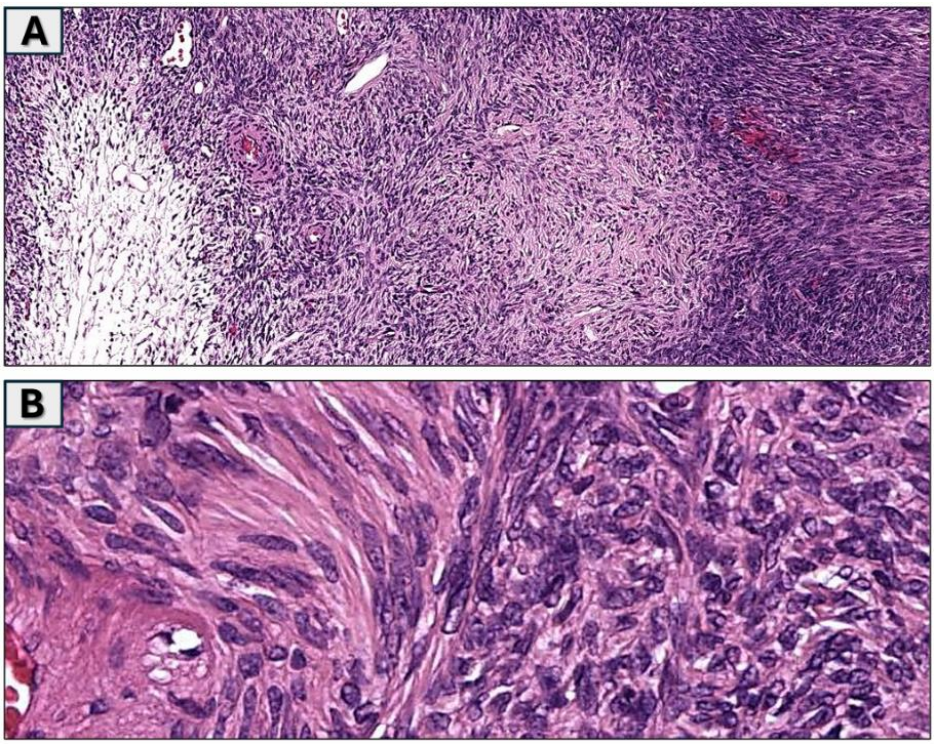
At surgery, both ovaries were noted to be mildly enlarged, measuring 4.1 × 2.8 cm on the right and 3.6 × 2.5 cm on the left. The external surfaces were smooth. On sectioning, the ovarian parenchyma was homogeneous, firm, and pale yellow-white, without discrete nodules, hemorrhage, or necrosis. The intraoperative frozen section consultation demonstrated stromal proliferation without evidence of malignancy, and the specimens were submitted for comprehensive histopathological evaluation.

Permanent pathology sections revealed diffuse and nodular proliferation of plump, lipid-laden luteinized stromal cells within a hypercellular ovarian stroma. The features included proliferation of ovarian stromal cells in a storiform pattern with scant intervening stroma and no atypia; scattered cystic follicles were also seen. In addition, focal nodules of vacuolated luteinized cells were present within the ovarian stroma. The histomorphology was consistent with ovarian stromal hyperplasia. (Figure 1 A, B) No evidence of a sex cord-stromal tumor or other neoplastic process was identified. Immunohistochemical (IHC) analysis demonstrated positivity for inhibin-α, calretinin, and steroidogenic factor-1, confirming steroidogenic stromal differentiation. The Ki-67 proliferation index was low (<3%), supporting a non-neoplastic process. CD99 and epithelial membrane antigen were negative, arguing against Sertoli-Leydig cell tumor and epithelial malignancy, respectively. Estrogen (ER) and progesterone (PR) receptors showed focal positivity, a finding commonly observed in ovarian stromal cells. Molecular studies were not pursued, as the histological and IHC features were diagnostic.

Postoperatively, biochemical evaluation at six weeks demonstrated normalization of total testosterone levels to below 20 ng/dL, with a return of LH and FSH to the expected postmenopausal range. Clinically, the patient reported cessation of new terminal hair growth within eight weeks and gradual regression of clitoromegaly over the subsequent six months.

2.5. Follow-Up and Outcome

At 12 months following bilateral salpingo-oophorectomy, the patient remained clinically stable with persistently normal testosterone levels. Features of virilization had partially regressed, with no further progression noted. There was no evidence of recurrence, and her metabolic comorbidities remained well controlled.



1A: Intermediate power view showing proliferating stromal cells with no atypia and scattered cystic follicles. To the left, there is a focal nodule of vacuolated luteinized cells within the ovarian stroma (H&E, stain X40); 1B: High power view showing stromal spindle cells appearing as plump, fibroblastic-type cells arranged in dense, intersecting bundles, whorls, and a storiform pattern with no atypia or necrosis (H & E Stain, X60)

Figure 1 Microscopic findings of Ovarian Stromal Hyperplasia

3. Discussion

3.1. Background (History, epidemiology, and risk factors)

OSH and ovarian hyperthecosis commonly occur in postmenopausal women with an average age of 55 years (age range: 51-64 years). [2] These conditions tend to affect both ovaries and occur in less than 1% of women of reproductive age. [6,7] The etiology of OSH and hyperthecosis conditions is not well known; however, these conditions have been associated with elevated levels of gonadotropins in postmenopausal women, hyperinsulinemia, insulin resistance, and obesity. Due to these strong associations, women with ovarian stromal hyperplasia are at an increased risk for metabolic conditions such as type 2 diabetes and hypertension. [8] There is also an increased risk of developing endometrial hyperplasia, polyps, and endometrial adenocarcinoma secondary to hyperestrogenism caused by aromatization of excess testosterone to estrogen. [9]

New onset and rapidly progressive virilization in women warrant further diagnostic workup to assess for underlying causes such as an androgen-producing ovarian tumor, congenital adrenal hyperplasia, Cushing's Syndrome, and thyroid dysfunction. This patient's laboratory results were significant for suppressed FSH and LH levels, indicating a gonadotropin-dependent process. Suppressed gonadotropin levels could be secondary to a neoplastic or non-neoplastic ovarian process, warranting further imaging, including a pelvic ultrasound and MRI, which may show bilaterally enlarged ovaries without hypervascularization. [2] Bilateral salpingo-oophorectomy is the standard treatment for ovarian stromal hyperplasia and confirms the diagnosis by immunohistochemistry. [10]

3.2. Pathogenesis, Pathophysiology

The pathogenesis begins with the permanent depletion of ovarian follicles. As developing follicles, the primary source of endogenous estrogen and inhibin, are lost, the natural negative feedback on the pituitary gland is eliminated. This results in persistently elevated levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). [1,11]

Within this high-gonadotropin environment, the ovarian stroma assumes a central role. Unlike follicular tissue, stromal cells retain responsiveness to LH. Chronic stimulation induces stromal hyperplasia, leading to symmetric ovarian enlargement and a characteristic firm, tan appearance on gross examination. [11,12]

As hyperplasia progresses, the stroma undergoes a functional transformation. Under continued LH influence, often exacerbated by concurrent insulin resistance, stromal cells may luteinize, accumulating lipid droplets and acquiring a metabolically primed state. At this juncture, the ovary effectively repurposes itself leading to sustained androgen production shifting away from cyclic hormonal production toward sustained synthesis of androgens, particularly androstenedione and testosterone. [2,3]

Clinical symptoms arise from how the body processes this late-life androgen excess. The pathophysiology follows two distinct pathways: First: *The Androgenic Pathway:* Elevated circulating testosterone directly stimulates hair follicles and vocal cord tissues. Affected women may experience abrupt onset of scalp hair thinning or growth of coarse facial hair (hirsutism), signs of virilization atypical in the postmenopausal period. [2,3] Second: *The Estrogenic Pathway:* More insidious is the peripheral conversion of androgens. Within adipose tissue, aromatase converts androstenedione to estrone. Because the postmenopausal woman no longer produces progesterone to counterbalance estrogen, the endometrium is subjected to unopposed stimulation. This creates a clinically dangerous sequence: endometrial hyperplasia may develop, which in severe cases progresses to malignant transformation into endometrial adenocarcinoma. [2]

3.3. Comparative Analysis of Our Case with Existing Literature. (Clinical, radiology, pathology, Lab, diagnosis, management, and outcome)

3.3.1. Clinical presentation, investigations and diagnosis

OSH, often discussed alongside ovarian hyperthecosis within the spectrum of non-neoplastic ovarian stromal causes of postmenopausal hyperandrogenism, remains an uncommon but important diagnostic consideration. [2,3] Most reported patients are postmenopausal women with obesity, insulin resistance, or type 2 diabetes who develop slowly progressive androgenic symptoms, usually hirsutism and scalp hair loss, with more advanced cases showing voice deepening or clitoromegaly. [4,7,12] Our patient fits much of that profile, given her postmenopausal status, obesity, diabetes, and 18-month history of virilization. What made the presentation more concerning was the degree of androgenism and the presence of clear virilization, such as clitoromegaly and voice change, which often raise concern for an occult androgen-secreting neoplasm rather than a non-neoplastic stromal disorder. [3,4]

That marked difference between a diffuse benign stromal process, and an occult tumor shaped the diagnostic evaluation. As in prior reports, markedly elevated testosterone with normal DHEA-S favored an ovarian rather than adrenal source of androgen excess. [4,7,12,14] Imaging also pointed toward ovarian stromal disease with bilaterally enlarged ovaries and no discrete androgen-producing neoplasm. [4] At the same time, imaging cannot reliably exclude small Leydig cell or steroid cell tumors, and this remains a central diagnostic challenge. [3,4] The suppressed LH and FSH added further complexity because recent pooled data suggest that lower gonadotropin levels may be seen more often in tumorous ovarian hyperandrogenism. For that reason, the differential diagnosis remained appropriately broad and included OSH, androgen-secreting sex cord-stromal tumor, occult adrenal neoplasm, and other endocrine causes of hyperandrogenism. [3,4]

Definitive diagnosis and clarification came from pathology. Reported cases of OSH and related stromal lesions describe diffuse stromal proliferation with luteinized stromal cell nodules, usually without significant atypia, as a characteristic morphologic pattern. [2,12,14] The findings in our case closely aligned with that description, showing diffuse and nodular proliferation of stromal cells within a hypercellular stroma, without a discrete neoplastic lesion. The immunophenotype further supported a stromal process: inhibin-α, calretinin, and steroidogenic factor-1 were positive, the Ki-67 index was very low, and epithelial markers were not expressed, arguing against epithelial malignancy and against a more proliferative sex cord-stromal neoplasm. [12,14, 15] As emphasized in the pathology literature, IHC is most useful for confirming lineage and excluding mimics, whereas final interpretation still depends on the overall morphologic pattern. [14,15]

3.3.2. Management and outcome

Management in the present case was consistent with current published practice. In postmenopausal women with suspected ovarian-source hyperandrogenism, bilateral salpingo-oophorectomy is generally considered the definitive diagnostic and therapeutic approach, particularly when bilateral disease is suspected. Fertility preservation is not relevant. [2,3,4,7,13,16] The interim GnRH agonist trial was a reasonable step because it supported ovarian dependence on androgen production and helped guide management. However, prior reports caution that biochemical suppression with GnRH analog therapy does not reliably distinguish OSH or hyperthecosis from an androgen-producing ovarian tumor, since both may respond to gonadotropin suppression. [4,12] The approach in our case, therefore, aligned with the literature while also illustrating why multidisciplinary consultation is valuable: surgery was pursued not because

the diagnosis was already certain, but because uncertainty persisted despite a thoughtful endocrine and radiologic workup.

The postoperative course was consistent with published experience. Testosterone normalized within weeks, no new virilizing features developed, and existing manifestations improved gradually over time. This pattern mirrors prior reports, in which biochemical correction is often rapid, whereas hirsutism, alopecia, and clitoromegaly regress more slowly and may not be fully resolved. [3,7,12,13] Recent literature suggests that recurrence after surgery is uncommon, although many published cases have relatively short follow-up. [3] In that context, the absence of recurrence at 12 months provides meaningful follow-up and strengthens the clinical relevance of this case.

Overall, this case adds to the limited literature by showing that OSH can present with marked virilization and testosterone levels high enough to mimic an androgen-secreting tumor, even when no discrete ovarian mass is identified

4. What Have We Learned from This Case?

This case highlights the importance of reporting ovarian stromal hyperplasia as an uncommon but clinically significant cause of postmenopausal hyperandrogenism. Its value lies in reminding clinicians that diffuse ovarian stromal pathology should remain in the differential diagnosis even when the initial biochemical profile and clinical presentation raise concern for an androgen-secreting neoplasm. Recognition of this possibility may help avoid premature diagnostic closure, refine preoperative evaluation, and support more accurate counseling and management.

Equally, this case underscores the importance of a multidisciplinary approach involving gynecology, endocrinology, pathology, radiology, and, when needed, oncology. Such collaboration is essential for integrating biochemical findings, imaging results, clinical features, and histopathology in diagnostically challenging presentations. By sharing this case, we hope to strengthen awareness of ovarian stromal hyperplasia within the medical community and to support more thoughtful diagnostic and therapeutic decision-making in postmenopausal women presenting with hyperandrogenism. [2,3,4]

Abbreviations

- Ovarian stromal hyperplasia (OSH);
- Immunohistochemical (IHC);
- Estrogen (ER);
- Progesterone (PR);
- Luteinizing hormone (LH) and
- follicle-stimulating hormone (FSH).

5. Conclusion

This case is worth reporting because it emphasizes that ovarian stromal hyperplasia, although uncommon, should remain an important diagnostic consideration in postmenopausal hyperandrogenism, even when the initial biochemical and clinical impression suggests neoplasia. The case also highlights the value of multidisciplinary consultation in achieving accurate diagnosis and guiding appropriate management. Increased awareness of this entity may help clinicians avoid misclassification, broaden the differential diagnosis, and improve care for similar patients.

Compliance with ethical standards

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Disclosure of conflict of interest

All authors make the following declarations:

- Payment/services information: All authors have declared that they received no financial support from any organization for the submitted work.
- Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might be interested in the submitted work.

Statement of ethical approval

Ethical review and approval were not required for this study involving human participants. The paper has been sufficiently anonymized to maintain the patient's confidentiality.

Data access statement

All relevant data are included in the paper.

Author contributions

All authors contributed equally to producing this manuscript.

Statement of informed consent

This study was conducted as a retrospective review of archival pathology material collected during routine clinical care. All data were fully de-identified prior to analysis. No patient contact or intervention was involved, and informed consent was not required in accordance with institutional policies.

References

[1] Mark S, Shanik MH, Romao IJ. 5048 A Rare Cause of Hyperandrogenism in Postmenopausal Women: Ovarian Stromal Hyperplasia. Journal of the Endocrine Society. 2024 Oct;8(Supplement_1):bvae163-1542.

[2] Araque TL, Ortiz IR, González JE, García AJ, Hidalgo IN, Gamarra VA, Soler CP, Raga FG. Ovarian stromal hyperplasia: a rare cause of postmenopausal hyperandrogenism. Journal of Menopausal Medicine. 2020 Apr 6;26(1):39.

[3] Forte A, D'Elia L, De Luca C, Fiore A, Barbato A, Abate V, Vergatti A, Verde N, De Filippo G, Venetucci P, De Angelis MC. Postmenopausal ovarian hyperandrogenism of surgically treated patients: a case report and scoping review with individual patient's data analysis. Frontiers in endocrinology. 2025 Aug 1;16:1495930.

[4] Hirschberg AL. Approach to investigation of hyperandrogenism in a postmenopausal woman. The Journal of Clinical Endocrinology & Metabolism. 2023 May 1;108(5):1243-53.

[5] Roque JJ, Alves IB, Fernandes AM, Bugalho MJ. A benign cause of hyperandrogenism in a postmenopausal woman. Endocrinology, diabetes & metabolism case reports. 2021 Feb 17;2021(1).

[6] Beksac S, Selçuk İ, Boyraz G, Güner G, Turgal M, Usubutun A. Two patients with marginal symptoms showing hyperthecosis at the edge of malignancy: presentation of two cases. Journal of the Turkish German Gynecological Association. 2013 Jul 10;14(3):182.

[7] Metzker LS, Ferreira LA, Borges JC, Guzzo MF, Ferreira RN, Silva LL, Cavedo RM, Filho AC. Postmenopausal hyperandrogenism due to ovarian hyperthecosis. Case Reports in Obstetrics and Gynecology. 2023;2023(1):2783464.

[8] Zaman A, Rothman MS. Postmenopausal hyperandrogenism: evaluation and treatment strategies. Endocrinology and metabolism clinics of North America. 2021 Jan 11;50(1):97.

[9] Zhang C, Sung CJ, Quddus MR, Simon RA, Jazaerly T, Lawrence WD. Association of ovarian hyperthecosis with endometrial polyp, endometrial hyperplasia, and endometrioid adenocarcinoma in postmenopausal women: a clinicopathological study of 238 cases. Human pathology. 2017 Jan 1;59:120-4.

[10] Bužinskienė D, Marčiukaitytė R, Šidlovska E, Rudaitis V. Ovarian Leydig cell tumor and ovarian hyperthecosis in a postmenopausal woman: a case report and literature review. Medicina. 2023 Jun 6;59(6):1097.

[11] Haroon S, Adnan HA. Stromal hyperplasia and hyperthecosis. PathologyOutlines.com website. https://www.pathologyoutlines.com/topic/ovarynontumorhyperthecosis.html. Accessed April 14th, 2026.

[12] Yousaf S, Nizar R, John L, Simpson A. A case of ovarian hyperthecosis in a postmenopausal woman. JCEM Case Reports. 2023 Nov;1(6):luad148.

[13] Knott JA, Morris J. Ovarian Hyperthecosis Presenting With Postmenopausal Virilization and New-Onset Type 2 Diabetes. JCEM Case Reports. 2025 Jul;3(7):luaf118.

[14] Devins KM, Young RH, Oliva E. Sex Cord–Stromal Tumors of the Ovary: An Update and Review. Part I—Pure Ovarian Stromal Tumors. Advances in Anatomic Pathology. 2024 Jul 1;31(4):215-30.

[15] Gupta S, Ahuja S, Kalwaniya DS. Immunohistochemistry markers in ovarian and fallopian tube neoplasms: a comprehensive review. Indian Journal of Surgical Oncology. 2024 Sep;15(Suppl 3):465-80.

[16] Tulandi T, Yu OH, Boucher LM, Fu L, Arseneau J. Virilization caused by ovarian nodular hyperthecosis in a postmenopausal woman. CMAJ. 2022 Dec 12;194(48):E1647-9.