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Case Report

Androgen-secreting borderline mucinous ovarian tumor in pregnancy: multidisciplinary management of gestational hyperandrogenism

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ABSTRACT

Most adnexal masses diagnosed during pregnancy are benign, but a subset is malignant, borderline, or hormonally active. Androgen-secreting tumors are particularly rare and carry an added risk of fetal virilization. We report the case of a 38-year-old pregnant woman incidentally diagnosed with a complex adnexal mass at 13 weeks of gestation, which showed progressive growth and was associated with elevated androgen levels suggestive of a functional tumor. A multidisciplinary team initiated conservative management with spironolactone to reduce the risk of fetal virilization, and surgery was performed at 28 weeks due to tumor growth progression. Histopathology confirmed a borderline mucinous tumor with Leydig cell hyperplasia. The patient had an uneventful recovery, with normalization of testosterone levels and a term delivery of a healthy newborn with no signs of virilization. This case, compared with the only three previously reported cases of this tumor in pregnancy, illustrates that an individualized, multidisciplinary approach to gestational hyperandrogenism caused by adnexal masses, combining conservative management with timely surgical intervention when indicated, can achieve favorable maternal and fetal outcomes.

Keywords: Androgen-secreting ovarian tumor, Borderline mucinous ovarian tumor, Pregnancy, Gestational hyperandrogenism, Adnexal mass

INTRODUCTION

The diagnosis of adnexal masses during pregnancy is a growing challenge. Most of these masses are incidental benign findings that often resolve spontaneously postpartum.^{1,2} However, a small percentage represent malignant or borderline neoplasms and may be functional, introducing more complex diagnosis, management, and prognosis for both mother and fetus.³

Borderline mucinous tumors are rare, accounting for approximately 10-20% of all diagnosed borderline ovarian tumors.³ Managing these tumors during pregnancy is

challenging not only due to their malignant potential but also due to the need for maternal and fetal risk balance. Androgen-secreting tumors, such as Sertoli-Leydig cell tumors, though uncommon, can further complicate management have given the risk of fetal virilization and maternal hyperandrogenism and obstetric complications such as premature birth and obstructed labor.^{2,4}

The authors present a clinical case of a pregnant woman with a rare androgen-secreting ovarian tumor, compare it with similar cases reported in the literature, and discuss therapeutic approaches and challenges in the multidisciplinary management of adnexal neoplasms during pregnancy.

CASE REPORT

This case report was approved by the Ethics Committee of Unidade Local de Saúde de São João (approval issued on March 28, 2025), and written informed consent was obtained from the patient for publication of clinical data and images.

A 38-year-old pregnant woman, without significant medical history or risk factors, BMI 23.3 kg/m², G2P1, with a spontaneous pregnancy of a female fetus. Cervical cancer screening conducted in 2022 was normal. Family history included a father with renal cancer diagnosed at age 50 and a paternal aunt with breast cancer diagnosed at age 60.

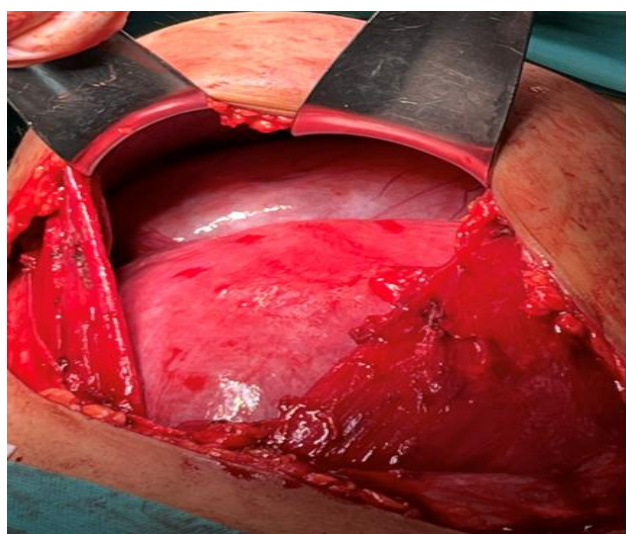


Figure 1: Pfannenstiel incision. Gravid uterus below, with the adnexal mass positioned superiorly.



Figure 2: Gravid uterus on the right and adnexal mass on the left.

At 13 weeks of gestation, during a routine first-trimester obstetric ultrasound, a complex unilateral adnexal mass

measuring 68×53×74 mm was incidentally detected. MRI at 15 weeks showed a unilateral adnexal mass measuring 100×54×89 mm, complex, multiseptated, predominantly cystic with internal hemorrhagic areas and suggestive signs of ovarian torsion (edematous ovary with multiple follicles). The contralateral ovary appeared normal. No iliac or para-aortic lymphadenopathy.

The adnexal tumor demonstrated progressive growth throughout the pregnancy, reaching 145mm by 20 weeks. Initial tumor markers (LDH, CA 15.3, CEA, CA 19.9) were negative, but the patient had progressively elevated total testosterone (1039 ng/dl, N<56.94), alpha-fetoprotein (57.76 ng/ml, N<8.04), and inhibin A (128.5 pg/mL, N<92), suggesting an androgen-secreting ovarian tumor, though without signs and symptoms of maternal virilization.

At 20 weeks, the patient was referred to our center, where multidisciplinary management, by a team comprising gynecologic oncologists, endocrinologists and obstetricians, began. At this time, an ultrasound showed a complex multilocular abdominal-pelvic mass measuring 145×95×132 mm. The mass was predominantly cystic with >10 locules, showing an anechoic/low echogenicity content and ground-glass appearance in one locule, regular internal contour and no solid components. The Ovarian-Adnexal Reporting and Data System (O-RADS) risk stratification classified as O-RADS 4, with an estimated malignancy risk between 10-50%.⁵

Given the mass's indeterminate etiology, significant androgen elevation, and potential risk of fetal virilization, a multidisciplinary discussion considered the following:

Etiology of the ovarian mass: the unilateral adnexal mass with gestational hyperandrogenism suggested a Sertoli-Leydig cell tumor, which can be benign or malignant depending on the differentiation grade, but with an increased risk of malignancy during pregnancy.⁶ In the absence of maternal symptoms, no extra-ovarian spread, and a stable tumor morphology, delaying surgery until postpartum was initially considered.

Risk of fetal virilization: given the elevated intrauterine androgen exposure of the female fetus, the risk of virilization was taken into account. This risk is most critical during the development of external genitalia in the first trimester. The timing of hyperandrogenism onset was unclear. A 21-week ultrasound showed no signs of fetal virilization (the mother had no signs of virilization as well).

Need for maternal intervention: the patient was informed about the experimental use of hormonal blockers like spironolactone to mitigate fetal androgen exposure. After discussion, spironolactone (100 mg/day) was initiated as a preventive measure.

At 28 weeks, the patient decided to undergo surgery. A laparotomic right adnexectomy (Pfannenstiel incision) under perioperative tocolysis was performed (Figure 1 and 2). Intraoperative rupture of the tumor occurred. No pelvic-abdominal lesions were identified. Macroscopy revealed a 180×173×60 mm right ovarian tumor with cystic and solid components, and histology confirmed a borderline mucinous tumor (FIGO IC1) with capsule-confined involvement, Leydig cell hyperplasia, and intratumoral stromal luteinization. Peritoneal washings were negative for malignancy.

Postoperative recovery was uneventful with normalization of testosterone levels. At 39 weeks, the patient had a spontaneous vaginal delivery, resulting in a healthy female newborn weighing 2920g, Apgar 9/10/10, with no signs of virilization.

DISCUSSION

The diagnosis of adnexal masses in pregnancy is rising in incidence.^{1,2} Most are benign incidental findings, mainly functional cysts, teratomas, and cystadenomas, with spontaneous resolution in the postpartum period, though a minority are malignant or borderline.¹⁻³ Borderline mucinous tumors are rare, representing 10-20% of borderline ovarian neoplasms, and compared with the serous variant are nearly always unilateral with less frequent peritoneal spread.³ In pregnant women presenting with virilization, the risk of malignancy is estimated at 50%.⁶

Ovarian tumors that secrete androgens are uncommon, and are most frequently associated with Sertoli-Leydig cell tumors, which carry a wide range of virilization risk.⁷ In non-pregnant women these tumors are usually benign; however, the proportion of malignant variants appears to increase during pregnancy.^{4,6} Nonetheless, any ovarian tumor containing cells with testicular differentiation can potentially secrete androgens.^{4,7}

There are various causes of hyperandrogenism during pregnancy, but the primary differential diagnosis should include, in the absence of an abdominal-pelvic mass, exogenous androgen administration or placental aromatase deficiency; and in the presence of a mass, adrenal tumors, Krukenberg tumors, and primary ovarian tumors.

Serum androgen levels alone do not reliably differentiate between benign and malignant ovarian tumors. Elevated serum inhibin A may indicate a Sertoli-Leydig cell tumor, while markers such as CA 19.9 and CA 125 may be elevated in epithelial tumors, although CA-125 can also rise physiologically in the first trimester of pregnancy, which limits its specificity in this setting.⁸

Most ovarian neoplasms detected during pregnancy are at an early stage and tend to have favorable outcomes compared with those diagnosed outside of pregnancy,

though management of an androgen-secreting, progressively growing tumor remains challenging.²

In this case, the initial decision for expectant management was influenced by the tumor's stability, the absence of maternal symptoms or fetal virilization, and the maternal and fetal risks associated with early surgical intervention. This conservative approach, combined with spironolactone treatment, aimed to mitigate fetal androgen exposure, although the use of hormonal blockers during pregnancy lacks robust evidence. Spironolactone, an antiandrogen and potassium-sparing diuretic, is commonly used for hyperandrogenic disorders such as hirsutism. A systematic review found that most of the animal data suggesting feminization of male offspring used doses well above typical human exposure, and did not identify feminization in the human cases reported.⁹ Case reports describe spironolactone use throughout pregnancy without feminization of male infants, including an accidental exposure in mid-gestation and a case of Bartter's syndrome treated with 400 mg/day across three pregnancies, all resulting in unaffected infants.^{10,11}

As the pregnancy progressed, tumor growth increased the suspicion of malignancy and the risk of maternal complications such as preterm labor, ovarian torsion, and obstructed labor.^{1,2} Torsion risk rises with mass size, while a growth rate greater than 0.35 cm/week has been associated with a substantially higher risk of malignancy.¹² Given the risk of mass rupture and peritoneal spillage with a laparoscopic approach, surgery via laparotomy was chosen.

In general, surgical intervention during pregnancy is reserved for acute abdomen, severe clinical manifestations, malignancy suspicion, or anticipated dystocia, and is best deferred to the second trimester when feasible; surgery later in pregnancy can be technically more challenging and carries a higher risk of adverse obstetric outcomes.² Minimally invasive laparoscopic surgery offers a better outcome profile in terms of hospital stay, blood loss, and operative complications, but current guidelines recommend laparotomy when malignancy is suspected, in order to avoid rupture and spillage of tumor cells.^{13,14}

In the present case, several ultrasonographic features classically associated with malignancy were present, including a multilocular, irregular mass greater than 10 cm, thickened septa, papillary projections, prominent blood flow, and the absence of a clearly benign morphology.¹⁵

The histopathological analysis revealed a borderline mucinous tumor with focal Leydig cell hyperplasia and stromal luteinization, accounting for the significant androgen production observed. Such findings are rare, as androgen-secreting ovarian tumors are uncommon overall.⁷ Although borderline mucinous tumors are generally considered to have low malignant potential, the

presence of Leydig cells and significant hormonal production complicates clinical management.³

Favorable maternal-fetal outcomes have generally been reported for adnexal neoplasms diagnosed during pregnancy, particularly when managed through a multidisciplinary approach.² In this case, despite the heightened risk of fetal virilization from Leydig cell hyperplasia and androgen production, initial conservative management followed by timely surgical intervention led to positive outcomes for both mother and newborn, with no signs of virilization in either.

To our knowledge, only three prior case reports describe androgen-secreting borderline mucinous tumors in pregnancy. Casanova et al reported a pregnant woman with a multilocular solid adnexal mass detected in the first trimester, managed with an open unilateral adnexectomy at 14 weeks, with histology confirming a borderline mucinous tumor.¹⁶ Fanara et al described a woman with an adnexal tumor detected at 23 weeks, presenting with acne and hirsutism, managed with laparotomic oophorectomy at 31 weeks.¹⁷ Pather et al reported a case with an androgen-secreting ovarian mass and ascites, removed by caesarean section at 28 weeks.¹⁸

CONCLUSION

This case underscores the importance of a multidisciplinary, individualized approach to the management of adnexal neoplasms in pregnancy, in which a thorough risk-benefit assessment of expectant versus early surgical management is essential to optimize maternal and fetal outcomes. By detailing the diagnostic work-up, the use of spironolactone as a bridging strategy, and the timing of surgery in a rare androgen-secreting borderline mucinous tumor, this report adds to the very limited existing literature on this specific tumor subtype in pregnancy and reinforces that individualized, evidence-based, multidisciplinary decision-making can achieve favorable outcomes for both mother and fetus.

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