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Case Report

The silent synechiae: female genital tuberculosis masquerading as resistant polycystic ovarian syndrome-induced amenorrhoea

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ABSTRACT

Female genital tuberculosis (FGTB) is a significant cause of menstrual disturbances and infertility in tuberculosis-endemic regions. However, its diagnosis is frequently delayed due to its paucibacillary nature and nonspecific presentation. We report the case of a 28-year-old woman with a known history of polycystic ovarian syndrome (PCOS). She presented with secondary amenorrhoea of six months' duration following the discontinuation of oral contraceptive pills (OCPs). While amenorrhoea is common in PCOS due to anovulation, this patient failed to exhibit withdrawal bleeding after a therapeutic challenge with progesterone and a subsequent course of OCPs. Pelvic ultrasonography provided false reassurance, showing polycystic ovarian morphology with normal endometrial thickness. A diagnostic hysteroscopy was performed to rule out outflow tract obstruction. It revealed filmy fundal synechiae and pale, unhealthy endometrium. Histopathology confirmed caseating epithelioid-cell granulomas with Langhans giant cells, and TB PCR detected *Mycobacterium tuberculosis* DNA. The patient was treated with a standard six-month anti-tubercular therapy (ATT) regimen under the national tuberculosis elimination programme (NTEP), resulting in the resumption of regular menstrual cycles. This case underscores the necessity of investigating structural causes, such as FGTB, in women with PCOS who present with atypical amenorrhoea refractory to hormonal withdrawal.

Keywords: Genital tuberculosis, Polycystic ovarian disease, Amenorrhoea

INTRODUCTION

Female genital tuberculosis (FGTB) remains an under-recognized yet clinically significant form of extrapulmonary tuberculosis, particularly in developing nations where the disease is endemic.

It is a silent masquerader, often presenting with vague symptoms such as infertility, pelvic pain, and menstrual irregularities ranging from oligomenorrhoea to complete amenorrhoea.

Historical cohorts suggest that genital TB accounts for nearly 14.7% of amenorrhoea cases, with contemporary literature reporting menstrual abnormalities in 25-60% of affected patients.¹

A major diagnostic dilemma arises when FGTB coexists with reproductive endocrine disorders such as PCOS. PCOS is characterized by hyperandrogenism and anovulation, leading to irregular cycles.

Typically, the endometrium in PCOS patients remains responsive to hormones; thus, they exhibit withdrawal bleeding following a progesterone challenge.²

When a patient with PCOS fails to bleed despite adequate hormonal priming, it signals a "endometrial factor" failure rather than a hormonal one.

This report illustrates a case where the diagnosis of FGTB was unmasked only after the patient failed standard hormonal management for PCOS, highlighting the critical role of hysteroscopy and molecular diagnostics.

CASE REPORT

Patient profile and history

A 28-year-old nulligravid woman presented to the gynecology outpatient department with a chief complaint of secondary amenorrhoea persisting for over six months.

Medical history

The patient had a documented history of PCOS. She had previously maintained regular menstrual cycles while on OCPs for cycle regulation.

Presenting complaint

Upon discontinuing the OCPs, she expected a return to her baseline cycle or mild irregularity. Instead, she developed complete cessation of menses.

Clinical evaluation

Initial management followed the standard protocol for secondary amenorrhoea in PCOS:

Progesterone challenge

The patient was administered a standard course of progesterone to induce a withdrawal bleed. This failed to produce menstruation.

Combined hormonal challenge

Suspecting hypoestrogenism or insufficient priming, a repeat course of OCPs was prescribed. This also failed to induce withdrawal bleeding.

Investigations

Laboratory analysis

Hormonal assays (FSH, LH, Prolactin, TSH) and virology screening were unremarkable.

Ultrasonography (USG)

A transvaginal ultrasound revealed bilateral polycystic ovarian morphology, consistent with her known diagnosis. Crucially, endometrial thickness (ET) reported as normal.

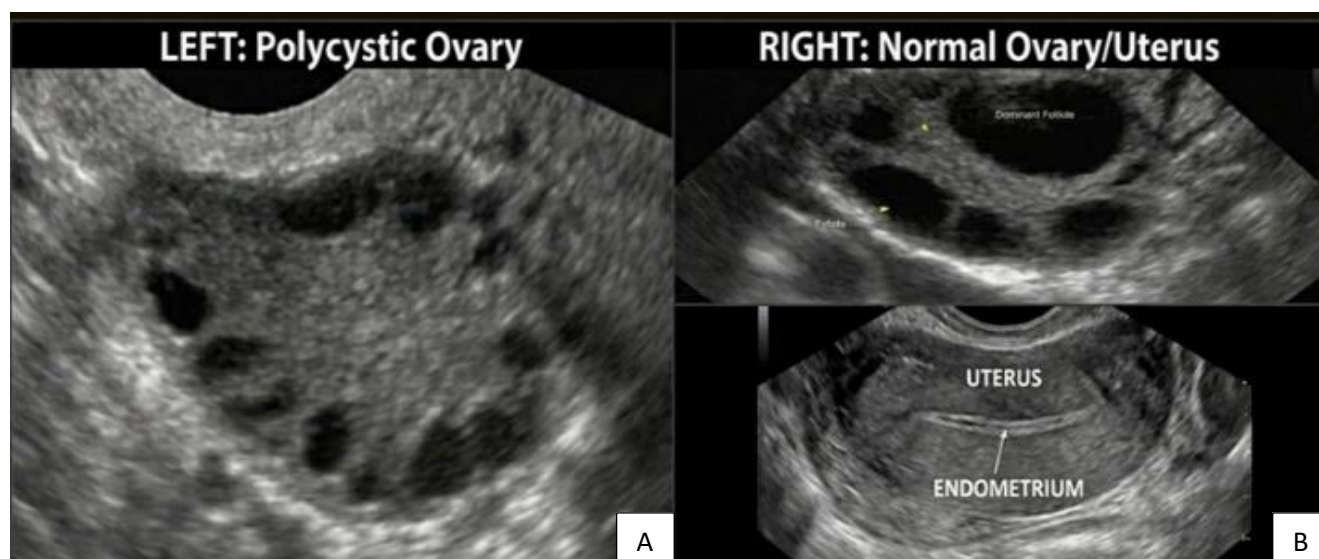


Figure 1 (A and B): Transvaginal ultrasound comparison. (Left) polycystic ovarian morphology showing multiple peripheral follicles ('string of pearls'). (Right) normal ovarian morphology/uterus (Illustrative purpose only).

Diagnostic hysteroscopy

Given the failure of withdrawal bleeding despite a normal endometrial echo on USG, a structural defect (e. g., Asherman's syndrome or infectious endometritis) was suspected. The patient consented to a diagnostic hysteroscopy.

Intraoperative findings

Cervical canal

It was normal.

Uterine cavity

The cavity was partially obliterated by filmy fundal synechiae (adhesions).

Endometrium

The mucosa appeared unhealthy, with scattered pale patches and areas of fibrosis, distinct from the lush appearance of a healthy estrogenized endometrium.

Hysteroscopic view of the uterine cavity showing filmy fundal synechiae (adhesions) and pale, unhealthy

endometrium characteristic of early tubercular endometritis.

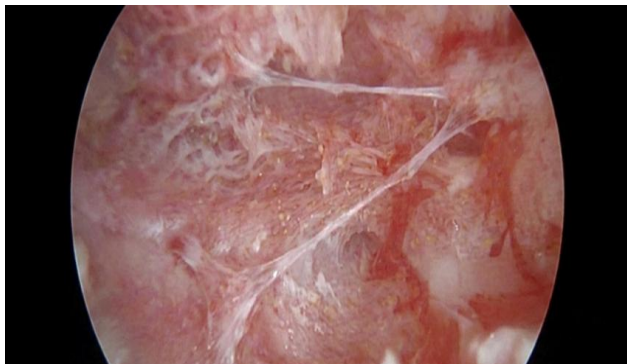


Figure 2: Hysteroscopy reveals filmy adhesions, caseating granulomas, pale and hyperemic endometrium suggestive of endometrial tuberculosis.

Histopathology and microbiology

Endometrial curettage samples were sent for comprehensive analysis:

Histopathology

Microscopic examination revealed the hallmark of tuberculosis: caseating epithelioid-cell granulomas interspersed with Langhans giant cells.³

Microbiology

Ziehl-Neelsen (ZN) stain

Negative for acid-fast bacilli (AFB), a common finding in genital TB due to the low bacterial load (paucibacillary nature).

TB PCR (Polymerase chain reaction)

Positive for *Mycobacterium tuberculosis* DNA, confirming the diagnosis.

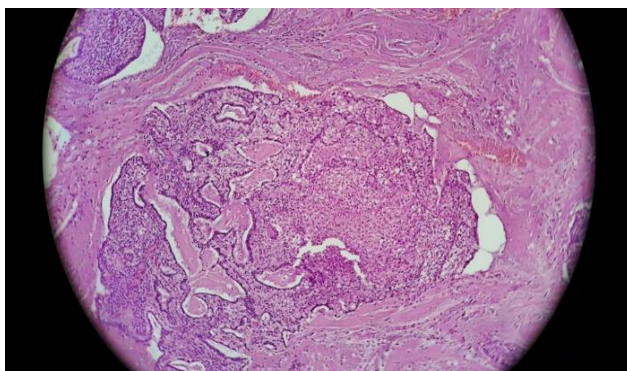


Figure 3: Histopathological picture shows caseating granuloma with langhans giant cells; Z-N stain.

Histopathological photomicrograph of endometrial biopsy showing a caseating epithelioid-cell granuloma with Langhans giant cells (H and E stain).

Treatment and outcome

The patient was initiated on ATT under the NTEP guidelines.

Regimen

The 2 months of intensive phase (Isoniazid, rifampicin, pyrazinamide and ethambutol) followed by 4 months of continuation phase (Isoniazid, rifampicin and ethambutol).⁶

Outcome

The patient tolerated the therapy well with no adverse events. Upon completion of the six-month course, her menstrual cycles resumed spontaneously and became regular, indicating the restoration of endometrial function.

DISCUSSION

This case highlights the “diagnostic trap” of attributing all menstrual irregularities in PCOS patients to anovulation. The key clinical pivot point in this case was the negative progesterone withdrawal test.

In typical PCOS, the endometrium is thickened due to unopposed estrogen.⁴ Administering progesterone and then withdrawing it triggers shedding (menstruation). Failure to bleed implies one of two things: either hypoestrogenism (unlikely in PCOS) or an outflow tract defect. In this patient, the persistent amenorrhoea despite OCP administration (which provides both estrogen and progesterone) strongly pointed to endometrial end-organ damage-in this case, caused by *Mycobacterium tuberculosis*.

A significant learning point from this case is the deceptive nature of ultrasound findings in FG TB. The patient had “normal endometrial thickness”. Unlike classic Asherman’s syndrome, which may show a thin, irregular line, tuberculous endometritis can create a pseudo-normal appearance due to caseous material or inflammatory debris mimicking healthy tissue. This underscores that a normal USG does not rule out FG TB.

Hysteroscopy was the gold standard in this case, allowing direct visualization of the “filmy synechiae” and “pale patches” characteristic of early-stage TB. Furthermore, while ZN staining was negative (consistent with the paucibacillary nature of FG TB), PCR provided the definitive etiological confirmation.⁵

CONCLUSION

FGTB should be strongly suspected in women with PCOS who present with “resistant” amenorrhoea that does not respond to hormonal withdrawal. This case demonstrates that early intervention with diagnostic hysteroscopy and molecular testing (TB PCR) is essential to prevent permanent reproductive morbidity. Successful treatment with ATT can reverse menstrual dysfunction if the structural damage is detected early.

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