



TWO FACES OF AMENORRHEA: PREMATURE OVARIAN INSUFFICIENCY VERSUS INTRAUTERINE ADHESIONS - A CASE REPORT.

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ABSTRACT

Background: Premature ovarian insufficiency (POI) causes cessation of ovarian function before 40 years and may present with amenorrhea and vasomotor symptoms. However, uterine pathology, including intrauterine adhesions, can also cause menstrual cessation and may complicate diagnosis. **Case Presentation:** A 40-year-old P2L2 woman with previous two caesarean sections, type 2 diabetes mellitus, hypertension, obesity and metabolic syndrome presented with amenorrhea, hot flushes, mood irritability and insomnia. She had no history of uterine curettage, manual removal of placenta or postpartum haemorrhage. Three-dimensional ultrasonography showed a small uterus ($4.7 \times 3.4 \times 2.2$ cm), thin endometrium (2.3 mm) and bilaterally normal ovaries. Hysteroscopy-guided biopsy revealed intrauterine adhesions, with only the right tubal ostium visualized. TruNAAT and Mantoux tests were negative. The coexistence of vasomotor symptoms, a small uterus and intrauterine adhesions raised the diagnostic dilemma of ovarian insufficiency versus uterine pathology. Serum FSH and further evaluation for secondary causes of amenorrhea were planned. **Conclusion:** Amenorrhea with menopausal symptoms requires careful evaluation of both ovarian function and uterine pathology. Intrauterine adhesions should not be assumed to indicate ovarian failure. A structured assessment incorporating biochemical confirmation of ovarian insufficiency and evaluation of the uterine cavity is essential for accurate diagnosis and appropriate management.

KEYWORDS : Premature Ovarian Insufficiency; Amenorrhea; Intrauterine Adhesions; Vasomotor Symptoms; Estrogen Deficiency; Asherman Syndrome; Menopause; Hysteroscopy; FSH.

INTRODUCTION

Premature ovarian insufficiency (POI) is characterized by impaired ovarian function before 40 years of age, presenting with menstrual disturbance and symptoms of estrogen deficiency. It has important implications for fertility, bone, cardiovascular and psychological health, and requires biochemical confirmation rather than symptom-based diagnosis alone.¹

Amenorrhea has a broad differential, including intrauterine adhesions, which may follow uterine instrumentation, infection or pregnancy-related complications. Hysteroscopy provides direct assessment of the uterine cavity and adhesions.^{5,6}

This case highlights the diagnostic challenge of distinguishing ovarian insufficiency from intrauterine pathology in a woman presenting with amenorrhea and vasomotor symptoms, emphasizing the need for systematic evaluation before diagnosing POI.

Case Presentation

A 40-year-old P2L2 woman with two previous lower-segment caesarean sections was evaluated in the gynecology department. Her medical history included type 2 diabetes mellitus, systemic hypertension, obesity, metabolic syndrome, and a previous clinical diagnosis suggestive of premature menopause.

She reported hot flushes, mood irritability and insomnia, with no history of night sweats, palpitations, significant anxiety or depression, vaginal dryness, dyspareunia, postmenopausal bleeding, urinary symptoms, cognitive impairment or

musculoskeletal symptoms.

Both previous caesarean deliveries were reportedly uneventful. There was no history of dilatation and evacuation, manual removal of placenta, postpartum haemorrhage or other known intrauterine instrumentation.

She was subsequently evaluated following hysteroscopy-guided endometrial and cervical biopsy. Clinical examination included assessment of BMI, blood pressure, thyroid, breast, abdomen and pelvis, with particular attention to features of estrogen deficiency and metabolic comorbidities.

Diagnostic Assessment

3D ultrasonography revealed a small uterus measuring $4.7 \times 3.4 \times 2.2$ cm, with an endometrial thickness of 2.3 mm and bilaterally normal-appearing ovaries. No Müllerian anomaly was identified. Normal ovarian morphology does not exclude POI.

Hysteroscopy-guided endometrial and cervical biopsy demonstrated intrauterine adhesions, with the right tubal ostium visualized and the left ostium not visualized, confirming significant intrauterine pathology that could contribute to menstrual disturbance.

Other investigations: TruNAAT and Mantoux tests were negative.

Planned Biochemical Evaluation

As POI requires biochemical confirmation, serum FSH was planned. Current guidelines recommend considering POI in women with ≥ 4 months of menstrual disturbance and FSH

>25 IU/L; if uncertain, FSH should be repeated after 4–6 weeks. Serum estradiol may support the diagnosis but should not be used alone.¹

Pregnancy testing, TSH and serum prolactin were planned where clinically indicated to exclude other causes of amenorrhea. Following confirmation of non-iatrogenic POI, appropriate genetic and autoimmune evaluation may be considered based on the clinical context.¹

Differential Diagnosis

Differentials included POI, early menopause, intrauterine adhesions (Asherman syndrome), thyroid dysfunction, hyperprolactinemia, hypothalamic–pituitary disorders, pregnancy, and other causes of secondary amenorrhea.

The main diagnostic challenge was distinguishing ovarian dysfunction from intrauterine pathology. Intrauterine adhesions may cause amenorrhea but do not by themselves confirm ovarian failure; POI requires biochemical confirmation.

Therapeutic and Clinical Management

Management was individualized according to biochemical confirmation and clinical findings. If POI is confirmed, hormone therapy should be considered until the usual age of menopause, unless contraindicated, with careful assessment of her metabolic and cardiovascular comorbidities.^{1,3}

Bone health should be optimized with calcium, vitamin D, weight-bearing exercise and BMD assessment when indicated. Cardiometabolic risk factors, including diabetes, hypertension, obesity and dyslipidemia, should be appropriately controlled. Mood and sleep symptoms should be monitored.

Intrauterine adhesions should be managed according to their extent, symptoms and reproductive goals, with hysteroscopic adhesiolysis considered when indicated.^{5,6}

DISCUSSION

POI causes ovarian estrogen deficiency before 40 years and may present with amenorrhea, hot flushes, sleep disturbance and mood changes. Current guidelines emphasize menstrual disturbance with biochemical confirmation, with FSH >25 IU/L supporting the diagnosis.¹

This case highlights the diagnostic challenge of distinguishing ovarian dysfunction from intrauterine pathology. Although the patient had symptoms suggestive of estrogen deficiency, hysteroscopy revealed intrauterine adhesions, which can cause hypomenorrhea or amenorrhea. Hysteroscopy is valuable for direct assessment of such adhesions.^{5,6}

Her diabetes, hypertension, obesity and metabolic syndrome further emphasize the need for individualized cardiovascular and metabolic risk assessment. If POI is confirmed, management should address vasomotor symptoms as well as bone, cardiovascular and psychological health.^{1,6}

Normal-appearing ovaries on ultrasonography do not exclude POI, while intrauterine adhesions and a thin endometrium do not establish ovarian failure. Biochemical assessment is therefore essential. Finally, the exact age at onset should be confirmed, as ovarian dysfunction before 40 years is termed POI, whereas onset between 40 and 45 years is classified as early menopause.

Strengths and Limitations

Strength: The coexistence of estrogen-deficiency symptoms and hysteroscopically confirmed intrauterine adhesions highlights the diagnostic challenge in evaluating amenorrhea.

Limitation: FSH/estradiol results and a clearly documented menstrual timeline were unavailable. Therefore, POI cannot be definitively diagnosed until biochemical criteria are fulfilled.

CONCLUSION

This case emphasizes the need for a systematic evaluation of amenorrhea with estrogen-deficiency symptoms. Intrauterine adhesions may contribute to menstrual cessation and should be distinguished from ovarian failure. Biochemical confirmation of POI, accurate menstrual history, and evaluation of endocrine and structural causes are essential. Confirmed POI requires comprehensive long-term care addressing bone, cardiovascular, metabolic and psychological health.

Learning Points

- POI requires biochemical confirmation and should not be diagnosed based on symptoms alone.
- Age and menstrual history are essential to distinguish POI from early menopause.
- Intrauterine adhesions should be considered in women with amenorrhea, even with estrogen-deficiency symptoms.
- Normal-appearing ovaries on ultrasound do not exclude POI.
- Confirmed POI requires long-term multidisciplinary care, including bone, cardiovascular, metabolic and psychological health.
- Hormone therapy should be individualized in women with significant metabolic and cardiovascular comorbidities.

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