Combined Reproductive and Multiple Endocrinopathies in a Single Case

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INTRODUCTION

Polycystic ovary syndrome (PCOS) is a prevalent endocrine disorder that leads to anovulatory infertility. PCOS is commonly linked to insulin resistance, substantially elevating the risk for metabolic disturbances, such as Type 2 diabetes, dyslipidaemia, and cardiovascular disease.[1,2] Pregnant women with PCOS are at a higher risk for gestational diabetes, which can result in fetal macrosomia and complications, such as neonatal hypoglycaemia and long-term metabolic sequelae.[3,4]

While the Rotterdam criteria for PCOS diagnosis require the exclusion of hyperprolactinaemia and hypothyroidism, these conditions may coexist with PCOS and further contribute to anovulation and polycystic ovarian morphology.[2] Notably, prolactinoma and Hashimoto's thyroiditis have been implicated in increasing the risk of pregnancy-related complications.[5,6]

CASE REPORT

A 31-year-old female with a 2-year history of infertility was initially diagnosed with PCOS. During her infertility workup, hormonal analysis revealed significantly elevated serum prolactin concentrations, prompting further evaluation. MRI of the pituitary gland revealed a macroadenoma. Additional endocrine assessment confirmed isolated hyperprolactinaemia, with no other pituitary hormone abnormalities identified. The patient was started on cabergoline to reduce prolactin levels and shrink the pituitary macroadenoma. After 18 months of treatment, radiological analysis confirmed a reduction in tumour size.

The patient was additionally diagnosed with Hashimoto's hypothyroidism and started on levothyroxine therapy. Upon achieving hormonal normalisation, the patient conceived. Cabergoline was maintained until 9 weeks of gestation. Her concomitant medical conditions exerted no detrimental impact on glucose tolerance or fetal development.

Upon early postpartum follow-up, given her history of pituitary adenoma, which may undergo progression due to pregnancy-related hormonal changes, comprehensive postpartum surveillance was undertaken. Re-evaluation of serum prolactin levels demonstrated significantly elevated values beyond the expected postpartum range, and a follow-up MRI revealed adenoma enlargement. Findings indicated the need for reassessment by ophthalmology and re-evaluation of pituitary hormones. Cabergoline therapy was reinitiated at a dose of 1.5 mg weekly; however, hyperprolactinemia persisted despite a reduction in prolactin levels. Therefore, the dose was raised to 2.5 mg per week.

The patient exhibited insulin resistance characterised by impaired fasting glucose and hyperinsulinaemia. Despite being on therapeutic doses of metformin, she

experienced progressive weight gain, indicating suboptimal metabolic control.

Given her medical history, early gestational diabetes screening was conducted at 12 weeks during her second pregnancy. Acknowledging her diagnosis, the patient underwent continuous metabolic assessment throughout her pregnancy, allowing for proactive monitoring, personalised counselling, and timely glycaemic control to improve outcomes for both mother and fetus. After implementing lifestyle changes, the patient's glucose levels stayed within normal ranges, and home readings were consistent. Fetal ultrasonography indicated growth about a week ahead of gestational age. The patient ultimately delivered at 38 weeks, a healthy neonate weighing 3.7 kg and measuring 51 cm.

Cabergoline therapy was promptly reinitiated in the postpartum period to prevent the recurrence of pituitary adenoma enlargement after being discontinued during pregnancy.

CONCLUSION

This case emphasises the challenges in diagnosing and managing concomitant endocrine disorders in reproductive-age women. Standard guidelines often fall short in the presence of comorbidities, underscoring the importance of individualised management and multidisciplinary care. Early detection and intervention are critical for improving outcomes and preventing complications.

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