Health Dynamics
eISSN: 3006-5518
Publisher: Knowledge Dynamics
DOI: https://doi.org/10.33846/hd20404

*Case Report*

# Spontaneous Conception and Favorable Obstetric Outcome in a Patient with PCOS and Class II Obesity: A Case Report

**Melisa Indah Mustikasari[1,*] Manggala Pasca Wardhana[1], Anin Indriani[1], Alexander Tikara Sugondo[1] and Made Chindy Dwiyanti Marheni Putri[2]**

[1]Medical Faculty, Airlangga University, Surabaya 60132, Indonesia
[2]MSC Ophthalmology, University College London, London EC1V2PD, United Kingdom

**Article history**
Received: 11 April 2025
Revised: 16 April 2025
Accepted: 21 April 2025
Published Online: 28 April 2025

***Correspondence:**
Melisa Indah Mustikasari
Address: East Marina Emas 3 Number 36, Surabaya 60111, Indonesia.
Email: melisaindah5910@gmail.com

**How to cite this article:** Mustikasari MI, Wardhana MP, Indriani A, Sugondo AT, Putri MCDM.Spontaneous Conception and Favorable Obstetric Outcome in a Patient with PCOS and Class II Obesity: A Case Report. Health Dynamics, 2025, 2(4), 160-165. https://doi.org/10.33846/hd20404



**ABSTRACT**

Polycystic ovary syndrome (PCOS) and obesity are prevalent conditions that independently impair fertility and increase the risk of adverse pregnancy outcomes. When coexisting, they present compounded challenges for conception and pregnancy management. We report a case of a 27-year-old primigravida with PCOS and class II obesity who achieved spontaneous conception after three years of infertility and multiple failed assisted reproductive attempts. Preconception lifestyle modifications and medical management improved insulin sensitivity and restored ovulatory function. Despite being categorized as a high-risk pregnancy, the antenatal course was unremarkable, with appropriate fetal growth and no major maternal or neonatal complications. Vaginal delivery occurred at term, resulting in a healthy newborn. This case highlights the potential for favorable reproductive and obstetric outcomes in women with PCOS and obesity through early diagnosis, lifestyle intervention, and meticulous prenatal care. It underscores the importance of individualized and multidisciplinary management strategies in optimizing maternal and fetal health. Spontaneous conception and favorable pregnancy outcomes are achievable in women with PCOS and obesity through comprehensive care. Early intervention and tailored monitoring play a crucial role in mitigating risks associated with these conditions.

**Keywords:** Polycystic ovary syndrome; obesity; fertility; pregnancy; infant

## 1. INTRODUCTION

Polycystic ovary syndrome (PCOS) is a common endocrine condition, affecting an estimated 6–13% of women of reproductive age worldwide. Notably, up to 70% of affected individuals remain undiagnosed. PCOS is the primary contributor to anovulatory infertility, accounting for approximately 80% of such cases. Its clinical presentation varies and may include menstrual disturbances, elevated androgen levels, and polycystic ovarian morphology. Obesity is frequently observed in individuals with PCOS, with prevalence rates reaching as high as 47%, further worsening the syndrome's reproductive and metabolic complications.[1,2]

Obesity itself is a significant public health concern, with global prevalence more than doubling since 1990. In 2022, it was estimated that 1 in 8 individuals worldwide were living with obesity. Among women of reproductive age, obesity rates are particularly alarming; in the United States, for instance, nearly 25% of pregnant women have obesity.

The concurrence of PCOS and obesity poses compounded risks for adverse pregnancy outcomes, including gestational diabetes mellitus (GDM), hypertensive disorders, and fetal growth abnormalities.[3,4]

Infertility remains a distressing consequence of PCOS, often necessitating assisted reproductive technologies (ART) for conception. However, women with PCOS exhibit a reduced response to ART and lower natural conception rates. The presence of obesity further diminishes fertility potential and complicates pregnancy management. Despite these challenges, lifestyle interventions aimed at weight reduction have been shown to improve ovulatory function and increase the likelihood of spontaneous conception in this population.[4]

This case report details the clinical course of a 27 years old primigravida with PCOS and class II obesity, who achieved spontaneous conception following lifestyle modifications after multiple unsuccessful ART attempts. The report underscores the importance of comprehensive preconception counseling, meticulous antenatal surveillance, and individualized management strategies to optimize maternal and fetal outcomes in pregnancies complicated by PCOS and obesity.

## 2. CASE REPORT

A 27 years old female presented to the emergency room at 39 weeks and 6 days of gestation with complaints of irregular uterine contractions and leakage of amniotic fluid that started at approximately. This was her first pregnancy which was achieved spontaneously following over three years of primary infertility and three failed intrauterine insemination (IUI) attempts at a fertility clinic. The patient had been diagnosed with PCOS three years prior, presenting with oligomenorrhea, hirsutism, and ultrasound confirmed bilateral polycystic ovaries. She had been initiated on metformin (2×500 mg/day), combined with folic acid and counseling on lifestyle interventions. Despite initial treatment, ovulation did not resume, and she required ovulation induction protocols during her fertility treatment. Eventually, with consistent adherence to a healthy lifestyle and improved insulin sensitivity, she began to ovulate spontaneously, resulting in a natural conception.

Her prepregnancy body mass index (BMI) was 37.8 kg/m², consistent with class II obesity. She had no personal history of diabetes mellitus, hypertension, or thyroid dysfunction. Antenatal follow-up was initiated early, and she was classified as a high-risk pregnancy due to obesity and PCOS. Routine antenatal laboratory and screening tests, including a negative OGTT at 28 weeks (OGCT 137 mg/dL), were performed. Hematologic and biochemical parameters remained within normal limits throughout gestation. Serial ultrasound examinations were performed starting from the first trimester. In the mid-trimester scan, there was suspicion of fetal growth restriction, with an EFW below the 10th percentile. To address this, fetal growth was closely monitored every 2–4 weeks using ultrasonography and Doppler velocimetry. The patient was also instructed to maintain adequate nutritional intake with appropriate caloric monitoring. Growth parameters improved over time, and third-trimester scans showed fetal growth within the 50th percentile. Doppler evaluations of the umbilical artery, middle cerebral artery (MCA), and uterine artery consistently demonstrated parameters within normal physiological limits.

Fetal wellbeing was monitored intensively through weekly non-stress tests (NSTs) starting from 36 weeks, given the maternal comorbidities. The patient also underwent regular maternal-fetal assessments to screen for preeclampsia, gestational diabetes, and abnormal fetal biometry. She was advised to monitor fetal movements daily and report any decrease in activity.

Fetal growth chart showing biometric measurements and estimated fetal weight (EFW) over time based on serial fetomaternal ultrasound examinations. Gestational age was assessed based on the date of the last menstrual period (LMP). Fetal biometric evaluation included biparietal diameter (BPD), head circumference (HC), abdominal circumference (AC), femur length (FL), and estimated fetal weight (EFW). The EFW values are expressed both in grams and percentiles. Percentile estimations are derived from the Hadlock fetal growth standard, which is widely used in obstetric ultrasound to assess intrauterine growth patterns. The plotted percentiles reflect how the fetal weight compares to a reference population for the corresponding gestational age. Fluctuations in EFW percentile over time suggest variations in fetal growth velocity, with a notable drop below the 10th percentile at 27 weeks, followed by catch-up growth by 35 weeks, and a subsequent decline to the 21st percentile by 38

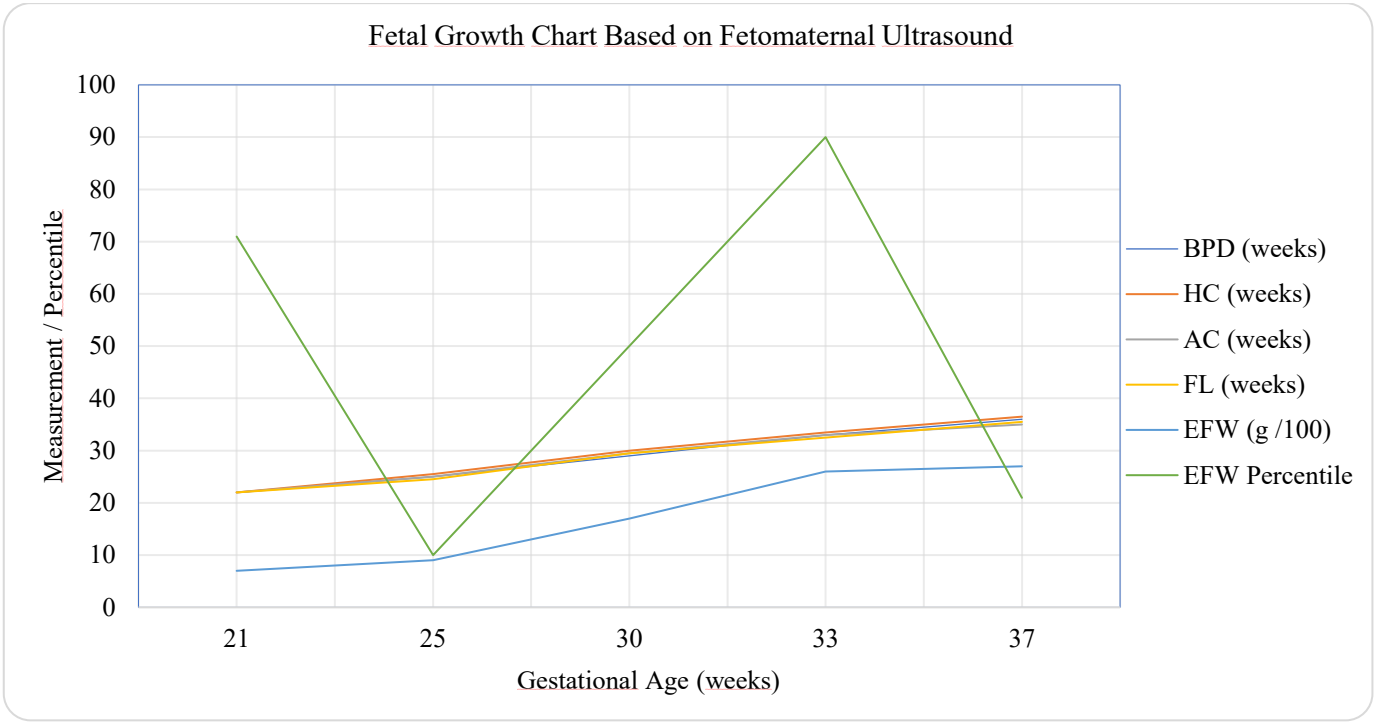


**Figure 1**. Fetal growth chart based on serial ultrasound EFWs, illustrating catch-up growth after the second trimester

weeks, raising clinical consideration for possible intrauterine growth restriction (IUGR) monitoring.

At 39 weeks and 6 days of gestation, she presented with spontaneous rupture of membranes and irregular uterine contractions. On arrival, the cervix was 3 cm dilated, 70% effaced, and the fetus was in cephalic presentation. Pelvimetry was clinically adequate, sacral promontory was not palpable, linea inominata was less than two-thirds, pubic arch >90°, and intertuberous diameter accommodated one fist. Labor was monitored using partographic observation. The patient was kept under continuous fetal monitoring to detect any fetal distress early. She was managed expectantly in early labor and supported through active labor with careful fluid balance, analgesia, and emotional support. After six hours, the patient delivered a healthy female infant vaginally at Kehamilan Sehat Clinic. The baby weighed 3300 grams, length 53 cm, with Apgar scores of 8 and 9 at one and five minutes, respectively. A single loop of nuchal cord was observed without any compromise. Clear amniotic fluid and an intact placenta were noted. A mediolateral episiotomy was performed, followed by successful primary perineal repair. The estimated blood loss was within normal limits, and uterine involution progressed well. The mother and baby were discharged in stable condition after 48 hours.

## 3. DISCUSSION

Polycystic ovary syndrome (PCOS) and obesity are well-established factors contributing to impaired fertility and adverse pregnancy outcomes. Despite these challenges, the presented case demonstrates a spontaneous conception and favorable obstetric result in a patient affected by both conditions. This discussion contextualizes the clinical course with current literature on the reproductive and obstetric implications of PCOS and obesity, including their pathophysiology, management strategies, and long-term outcomes.

### *PCOS as Reproductive Health Issues and Pathophysiology*

PCOS is among the most prevalent endocrine disorders in women of reproductive age, with global prevalence estimates ranging from 5% to 20%.[2,3] It is typified by hyperandrogenism, ovulatory dysfunction, and polycystic ovarian morphology.[4] Obesity, affecting nearly 25% of pregnant women in high-income countries,[3] shares and amplifies the reproductive and metabolic disturbances seen in PCOS.[2,4] Both conditions warrant early diagnosis and a multidisciplinary management approach to mitigate their reproductive and metabolic sequelae.[5,6]

PCOS involves a multifactorial etiology, with genetic, metabolic, endocrine, and environmental contributors. Heightened GnRH pulsatility elevates LH secretion, stimulating excess ovarian androgen production. Concurrently, low or normal FSH impairs follicular maturation. Insulin resistance (IR), frequently present even in lean PCOS patients, promotes hyperinsulinemia, which exacerbates androgen excess by stimulating theca cell activity and reducing SHBG synthesis.[7]

Obesity compounds these derangements by worsening insulin resistance, disrupting hormonal balance, and inducing chronic low-grade inflammation. Inflammatory cytokines (e.g., IL-1, IL-6, TNF-$\alpha$) and immune dysregulation further aggravate metabolic dysfunction and reproductive impairment.[9,10] This results in compromised ovulatory function, poor oocyte quality, and impaired endometrial receptivity.

These abnormalities contribute to anovulation, suboptimal embryo development, and reduced implantation rates, even with ovulation induction. The overlap with metabolic syndrome (MetS) further emphasizes the systemic nature of PCOS,[8] highlighting the importance of individualized and integrated management strategies.[11]

### *Impact of PCOS and Obesity on Fertility and Pregnancy*

PCOS is responsible for approximately 40% of female infertility cases, primarily due to chronic anovulation.[2] Increased GnRH pulsatility causes elevated LH, which disrupts follicular development. High AMH levels reflect numerous immature follicles that fail to mature.[12] IR and hyperinsulinemia worsen androgen excess and reduce SHBG, compounding ovulatory dysfunction.[5,6,9] Obesity intensifies these issues, further impairing ovulation, reducing responsiveness to fertility treatments, and diminishing endometrial receptivity. Consequently, women with both PCOS and obesity often experience reduced spontaneous conception and poorer outcomes with assisted reproductive technologies. They are also at greater risk for OHSS and multiple gestations.[6,9,12,13]

Women with PCOS and/or obesity face higher risks of gestational diabetes mellitus (GDM), preeclampsia, preterm birth, and intrauterine fetal death (IUFD).[13,14] These complications are often driven by IR and systemic inflammation, which negatively affect placental function. Additionally, fetal outcomes are marked by an increased risk of IUGR or macrosomia, both of which are linked to birth trauma and higher NICU admissions.[14,15] Meta-analyses confirm that PCOS significantly elevates the risk of adverse maternal and fetal outcomes, while obesity adds independent and additive risks when coexisting with PCOS.[14,15] These findings highlight the importance of early identification and tailored prenatal care.

### *Management Strategies, Prognosis and Implication*

Management of PCOS and obesity begins with lifestyle modifications. A weight reduction of just 5–10% can restore ovulation and improve fertility outcomes.[7,9,10] Pharmacologic treatments such as metformin and inositol may improve insulin sensitivity. Ovulation induction agents like letrozole (preferred over clomiphene citrate) further enhance conception rates.[10,11] During pregnancy, close monitoring is essential. This includes regular blood glucose checks, blood pressure monitoring, and serial ultrasounds to assess fetal growth and placental function. High-risk cases may require Doppler studies.[10,11,13] A multidisciplinary team—obstetricians, endocrinologists, and nutritionists—is vital for optimal care.[9] Intrapartum management should be individualized. Obese patients or those with macrosomia require careful planning to reduce the risks of shoulder dystocia and cesarean delivery. Anesthesia considerations and delivery planning should be tailored to the patient's risk profile.[9,13] Women with PCOS and/or obesity are at higher risk for long-term metabolic conditions such as type 2 diabetes mellitus (T2DM), cardiovascular disease, and persistent infertility due to chronic anovulation.[6,9,12] IR and inflammation play central roles in these complications. Furthermore, in utero exposure to maternal hyperandrogenism and metabolic dysfunction may lead to epigenetic changes in the offspring, increasing their risk for obesity, IR, and metabolic syndrome.[5,6,10] This highlights the need for ongoing monitoring and preventive care for both mother and child.

### *Case Correlation*

The presented case illustrates a spontaneous conception and favorable obstetric outcome in a patient with PCOS and class II obesity, conditions typically associated with reproductive and metabolic challenges. As described in the literature, PCOS and obesity independently contribute to subfertility through mechanisms involving anovulation, hyperandrogenism,

and insulin resistance.[1,2,5,6] Despite these barriers, our patient achieved spontaneous conception, highlighting that lifestyle factors or underlying variations in phenotypic expression of PCOS may influence fertility outcomes.

From a maternal perspective, the patient carried a high-risk profile for pregnancy complications such as gestational diabetes mellitus (GDM), hypertensive disorders, and cesarean delivery, which are consistently reported in women with PCOS and/or obesity.[6,12,13,15] However, throughout the antenatal period, no major complications developed, possibly due to early and continuous prenatal monitoring, weight stabilization, and metabolic compensation, underscoring the potential benefit of multidisciplinary management in high-risk pregnancies.

Fetal outcomes in such conditions are often complicated by intrauterine growth restriction (IUGR), macrosomia, or neonatal intensive care unit (NICU) admission.[6,12–14] In our case, the estimated fetal weight was within normal range, and no immediate neonatal complications occurred, suggesting that individualized care and close fetal surveillance such as serial ultrasounds and Doppler studies can mitigate these risks.

Moreover, considering the long-term prognosis, the patient may face increased risk for future metabolic syndrome or type 2 diabetes mellitus, as commonly seen in post-partum PCOS women, especially when compounded by obesity.[5,6,9] Additionally, the child may carry a heightened risk of developing obesity or insulin resistance, given the known transgenerational effects of metabolic and hormonal dysregulation during gestation.[5,9]

Overall, this case contrasts with the commonly poor reproductive and obstetric outcomes in women with PCOS and obesity but affirms that favorable results are attainable with timely diagnosis, comprehensive monitoring, and supportive care throughout the preconception and antenatal periods.

# 4. CONCLUSION

Despite the well-established reproductive and metabolic challenges associated with PCOS and obesity, this case demonstrates that spontaneous conception and favorable pregnancy outcomes are achievable. Key contributing factors included early diagnosis, lifestyle modifications, and multidisciplinary prenatal care. This highlights the importance of individualized management and continuous surveillance to optimize maternal and fetal health in high-risk pregnancies involving PCOS and obesity.

## Ethical Approval

Not required.

## Acknowledgement

Thanks to the medical staff of Iskak Regional Public Hospital, as well as the patient for her cooperation and consent, which made this study possible.

## Competing Interests

All the authors declare that there are no conflicts of interest.

## Funding Information

No funds were received for this study.

## Underlying Data

Derived data supporting the findings of this study are available from the corresponding author on request.

# REFERENCES

1. Huo M, Wang Y, Yuan X, Yuan Y, Zhang X. Changing trends in the global burden of polycystic ovarian syndrome-related infertility over the past 30 years: retrospective data analysis of the global burden of disease study 2019. BMC Women's Health. 2025;25(1):35. http://dx.doi.org/10.1186/s12905-024-03537-7
2. Tay CT, Loxton D, Bahri Khomami M, Teede H, Harrison CL, Joham AE. High prevalence of medical conditions and unhealthy lifestyle behaviours in women with PCOS during preconception: findings from the Australian Longitudinal Study on Women's Health. Human Reproduction. 2023;38(11):2267–2276. http://dx.doi.org/10.1093/humrep/dead190
3. Ogunwole SM, Zera CA, Stanford FC. Obesity Management in Women of Reproductive Age. JAMA. 2021;325(5):433. http://dx.doi.org/10.1001/jama.2020.21096
4. McDonnell R, Hart RJ. Pregnancy-related outcomes for women with polycystic ovary syndrome. Women's Health. 2017;13(3):89–97. http://dx.doi.org/10.1177/1745505717731971
5. Patel S. Polycystic ovary syndrome (PCOS), an inflammatory, systemic, lifestyle endocrinopathy. The Journal of Steroid Biochemistry and Molecular Biology. 2018;182:27–36. http://dx.doi.org/10.1016/j.jsbmb.2018.04.008
6. Calcaterra V, Verduci E, Cena H, Magenes VC, Todisco

CF, Tenuta E, et al. Polycystic Ovary Syndrome in Insulin-Resistant Adolescents with Obesity: The Role of Nutrition Therapy and Food Supplements as a Strategy to Protect Fertility. Nutrients. 2021;13(6):1848. http://dx.doi.org/10.3390/nu13061848

7. Marinelli S, Napoletano G, Straccamore M, Basile G. Female obesity and infertility: outcomes and regulatory guidance. Acta Biomedica. 2022;93(4):e2022278. https://doi.org/10.23750/abm.v93i4.13466
8. He Y, Lu Y, Zhu Q, Wang Y, Lindheim SR, Qi J, et al. Influence of metabolic syndrome on female fertility and in vitro fertilization outcomes in PCOS women. American Journal of Obstetrics and Gynecology. 2019;221(2):138.e1-138.e12. http://dx.doi.org/10.1016/j.ajog.2019.03.011
9. Matsuyama S, Whiteside S, Li SY. Implantation and Decidualization in PCOS: Unraveling the Complexities of Pregnancy. International Journal of Molecular Sciences. 2024;25(2):1203. http://dx.doi.org/10.3390/ijms25021203
10. Artini PG, Obino MER, Sergiampietri C, Pinelli S, Papini F, Casarosa E, et al. PCOS and pregnancy: a review of available therapies to improve the outcome of pregnancy in women with polycystic ovary syndrome. Expert Review of Endocrinology & Metabolism. 2018;13(2):87–98. http://dx.doi.org/10.1080/17446651.2018.1431122
11. Mirza FG, Tahlak MA, Rjeili RB, Hazari K, Ennab F, Hodgman C, et al. Polycystic Ovarian Syndrome (PCOS): Does the Challenge End at Conception? International Journal of Environmental Research and Public Health. 2022;19(22):14914. http://dx.doi.org/10.3390/ijerph192214914
12. Wang J, Yin T, Liu S. Dysregulation of immune response in PCOS organ system. Frontiers in Immunology. 2023;14:1169232. http://dx.doi.org/10.3389/fimmu.2023.1169232
13. Bahri Khomami M, Shorakae S, Hashemi S, Harrison CL, Piltonen TT, Romualdi D, et al. Systematic review and meta-analysis of pregnancy outcomes in women with polycystic ovary syndrome. Nature Communications. 2024;15(1):5591. http://dx.doi.org/10.1038/s41467-024-49749-1
14. Farland LV, Stern JE, Liu CL, Cabral HJ, Coddington CC, Diop H, et al. Polycystic ovary syndrome and risk of adverse pregnancy outcomes: a registry linkage study from Massachusetts. Human Reproduction. 2022;37(11):2690–2699. http://dx.doi.org/10.1093/humrep/deac210
15. Zhang Y, Lu M, Yi Y, Xia L, Zhang R, Li C, et al. Influence of maternal body mass index on pregnancy complications and outcomes: a systematic review and meta-analysis. Frontiers in Endocrinology. 2024;15:1280692. http://dx.doi.org/10.3389/fendo.2024.1280692