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*Case Report*

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# A severely obese patient with long-term infertility due to polycystic ovary syndrome who was pregnant after intensive preoperative weight loss treatment, but underwent laparoscopic sleeve gastrectomy unaware of this pregnancy and subsequently delivered a healthy infant

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## Abstract

Obese type-Polycystic Ovary Syndrome (PCOS) is characterized by severe metabolic complications and a low live birth rate. We report a case of a 41-year-old Brazilian woman with severe obesity (BMI, 53.6 kg/m²), hypertension, sleep apnea syndrome, type 2 diabetes mellitus (HbA1c 7.8%) and long term-infertility due to PCOS, which was diagnosed at age 24. The PCOS was treated without successes in the patient's infertility or Body Weight (BW) loss. After intensive nutritional guidance combined with semaglutide/tirzepatide treatment for 7-months, the patient achieved a -9.9% BW loss in anticipation of Laparoscopic Sleeve Gastrectomy (LSG). She became pregnant at 6 weeks pre-LSG, but this was not discovered until the 14th week (8 weeks post-LSG). We changed her post-operative nutritional management to maintain maternal health and fetal growth by increasing her daily energy and each nutrient intake. She delivered a healthy infant via scheduled cesarean section at 38 weeks of pregnancy.

## Introduction

Polycystic Ovary Syndrome (PCOS) is a heterogeneous endocrine and metabolic disorder associated with long-term infertility, which affects women of reproductive age worldwide [1]. Furthermore, women with PCOS are more likely to have certain comorbidities, including obesity, insulin resistance, and type 2 diabetes mellitus (T2DM) [2,3]. Weight reduction is associated with improved infertility in some PCOS patient [4]. A recent review article by Gao et al. [5] clearly describes the 4 phenotype-specific types of PCOS: 1) hyperandrogenic PCOS, which shows the highest risk of second-trimester pregnancy loss and a high incidence of dyslipidemia; 2) Obese type (OB)-PCOS which shows severe metabolic complications involving multiple obesity-associated comorbidities, and the lowest live birth rates, but the highest remission rate; 3) PCOS exhibiting a high sex hormone-binding globulin showing favorable reproductive outcomes and the lowest incidence of diabetes and hypertension; and 4) PCOS with high-luteinizing hormone and anti-Müllerian hormone, which shows the greatest risk of ovarian hyperstimulation and the lowest PCOS remission rate. In this case report, we presented a OB-PCOS patient with a successful but unrecognized pregnancy after pre-surgery Body Weight (BW) loss of -9.9% following intensive medical and nutritional treatments. She underwent Laparoscopic Sleeve Gastrectomy (LSG) for weight reduction, after which it

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was discovered that she was 14-weeks pregnancy. Thus, the specific pre- and post-LSG clinical care in this OB-PCOS patient is valuable as a case report.

## Case report

A severely obese 41-year-old Brazilian female patient visited our obesity outpatient clinic to achieve BW reduction through bariatric surgery. Her BW had gradually increased to 76 kg at age 24, when PCOS was diagnosed after her marriage and thereafter her obesity continuously worsened alongside progression of T2DM and hypertension without medical treatment. Since age 17, she had noticed that her menstrual cycle was irregular and she suffered long-term infertility after marriage without contraception. As shown in (Table 1), she had a BW of 133.7 kg (Body Mass Index [BMI], 53.6 kg/m$^2$) at her first visit, with hypertension, 164/109 mmHg, HbA1c, 7.8%, steatosis, sleep apnea syndrome, and markedly increased subcutaneous and visceral adiposity measured by abdominal Computed Tomography (CT). However, she showed no symptoms related to hyperandrogenism such as acne, hirsutism, or alopecia, which were coincident with normal testosterone levels (Table 2). She showed mild insulin resistance (Homeostasis Model Assessment of insulin Resistance [HOMA-R], 1.94) and marked hyperinsulinemia (HOMA-β, 208) before surgery. She was a good candidate for LSG based on her ABCD score (Age, BMI, C-peptide, Diabetes duration) of 8 points [6]. As shown in Figure 1, we started pre-surgical medical treatment in order to reduce BW by introducing a low-energy (1200 kcal/day) diet consisting of relatively high protein (20%) and fat (40%) and low carbohydrate (40%). To promote glycemic control and BW reduction, we initiated once a week injection of semaglutide (0.25-1.0 mg) and then tirzepatide (2.5-10 mg). At 7 months after those treatments, the patient's BW had decreased by -9.9%. Although she became pregnant following these treatments, she was unaware of the pregnancy. She underwent LSG for BW reduction and shortly thereafter was found to be 14 weeks pregnant. We then changed her nutritional protocol from an intensive low-energy diet to diabetes control during pregnancy, as shown in (Figure 2). The patient's total energy intake was increased from 800 kcal/day to 1300 kcal/day and finally 2,000 kcal/day with increases in all nutrients, especially an increased carbohydrate intake of 240 g/day until delivery. Thus, maternal BW was maintained around 100 kg and fetal growth steadily increased. A healthy female infant was delivered by scheduled cesarean section at 38 weeks of gestation with an infant BW of 2,532 g and an Apgar score of 9/10. Maternal glycemic control (HbA1c, 5.7%), blood pressure (120/50), normal serum lipid levels, and renal and hepatic function tests were all within normal range upon delivery. Menstruation resumed naturally approximately three months after delivery, with a cycle of approximately one month. In a recent gynecological examination, our obstetrician and gynecologist found no PCOS.

**Table 1:** Clinical course of body weight reduction and changes in laboratory data after medical and surgical treatments in morbidly obese patients with polycystic ovary syndrome.

| | Initial examination | 5 months-pre-surgery | 6 weeks-pre-surgery<br>Pregnancy: estimated | 8 weeks-post-surgery<br>Pregnancy: confirmed | 1 year-post-surgery |
|---|---|---|---|---|---|
| Body weight, kg | 133.7 | 125.1 | 120.4 | 103 | 100.6 |
| Body fat, % | 53.1 | 53.5 | 53.1 | 51.2 | 51.5 |
| LBM, kg | 62.71 | 58.17 | 56.47 | 50.26 | 48.8 |
| SBP/DBP. mmHg | 154/110 | 141/99 | 147/98 | 117/71 | 130/90 |
| FPG, mg/dL | 98 | 80 | 78 | - | 92 |
| HbAle, % | 7.8 | 6.3 | 6.3 | 5.5 | 6.2 |
| TG, mg/dL | 135 | 119 | 113 | - | 87 |
| HDL-C, mg/dL. | 46 | 34 | 40 | 52 | 30 |
| LDL-C, mg/dL | 80 | 91 | 75 | 63 | 84 |
| AST, IU/L | 18 | 16 | 16 | 11 | 13 |
| ALT, IU/L | 24 | 15 | 13 | 11 | 10 |
| y-GTP, IU/L | 27 | 14 | 14 | - | 12 |
| Albumin, mg/dL | 3.9 | 3.9 | 4.1 | 3.2 | 3.5 |
| Uric acid, mg/dL | 4.5 | 4.9 | 5.1 | 3.2 | 4.6 |
| eGFR, mL/min/1.73 m$^2$ | 98.9 | 107.8 | 92.5 | 120.4 | 93.7 |
| HOMA-R | | 1.9 | | - | 1.6 |
| HOMA-β | | 207.6 | | - | 86.9 |
| Visceral fat area, cm$^2$ | | 150.7 | | - | 158.2 |
| Subcutaneous fat area, cm$^2$ | | 828.4 | | - | 659.3 |

**Table 2:** No abnormal hormonal characteristics to express hyperandrogenism and LH/FSH ratio in this patient with obese-type polycystic ovary syndrome.

| Characteristics in hormonal levels | 5 months pre-surgery | 1-year post-surgery |
|---|---|---|
| LH, mIU/mL | 3.4 | 4 |
| FSH, mIU/mL | 5.6 | 4.7 |
| E2, pg/mL | 37 | 39 |
| Progesterone, ng/mL | <0.1 | 0.2 |
| Testosterone, ng/mL | 0.18 | 0.12 |
| Antimüllerian hormone, ng/mL | 1.34 | 0.67 |
| Cortisol, µg/dL | 4.76 | 5.5 |
| DHEA-s, ng/mL | 1020 | 663 |
| GH, ng/mL | 0.14 | 0.76 |
| IGF-1, ng/mL | 98 | 105 |
| Prolactin, ng/mL | 12.6 | 17.5 |
| fT4, ng/dL | 1.05 | 1.12 |
| TSH, µIU/mL | 1.96 | 1.03 |

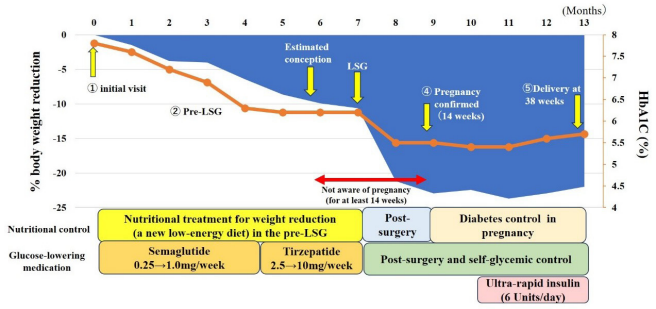


**Figure 1:** Clinical course of body weight and HbA1c levels during the intensive nutritional and pharmacological treatments in a patient with obese-type polycystic ovary symdrome.


| Nutrition | ① Initial visit | ② Pre-LSG | ③Early post-LSG | ④ Pregnancy confirmed | ⑤ At delivery |
|---|---|---|---|---|---|
| Energy intake (kcal/day) | 2200 | 1120 | 800 | 1300 | 2000 |
| Protein (g) | 45 | 65 | 45 | 55 | 80 |
| Fat (g) | 60 | 60 | 30 | 50 | 60 |
| Carbohydrate (g) | 370 | 80 | 80 | 160 | 240 |
| Urine ketone body | - | - or 1+ | 3+ | 1+ | - |

**Figure 2:** Clinical course of maternal and infant body weight changes during pregnancy under intensive nutritional control in a patient with obese-type polycystic ovary syndrome.

## Discussion/Conclusion

A female patient with OB-PCOS achieved a 9.9% reduction in BW through a 7-month treatment including nutritional and pharmacological (semaglutide followed by tirzepatide) treatments, achieving unrecognized pregnancy before LSG. Nutritional approaches, including a Mediterranean or ketogenic diet with very low carbohydrates, are partly effective in short-term treatment for patients with OB-PCOS [7]. However, we used a non-ketogenic low-energy diet with a Protein: Fat: Carbohydrate balance of 20:40:40% to reduce the severe obesity of this OB-PCOS patient in conjunction with pharmacological treatments.

It is generally accepted that PCOS patients should use contraception for at least 1 year pre- and post-LSG because of difficulties related to maternal and fetal nutritional care in the gestational period [8]. In the present study, the patient conceived at -9.9% BW reduction, approximately 6 weeks pre-LSG, but the pregnancy was not discovered until 8 weeks post-LSG. Thus, we were required to change the goal of post-LSG medical care from weight reduction to diabetes control in the gestational period. Fortunately, we were able to maintain a maternal body weight of approximately 100 kg while supporting fetal growth. A healthy infant was delivered by scheduled cesarean section at 38 weeks of gestation.

In the present case, our OB-PCOS patient showed no signs of hyperandrogenism. However, according to the classical Rotterdam criteria [9], 2 of the 3 main characteristics of PCOS (hyperandrogenism, chronic anovulation, polycystic ovary) are sufficient for a diagnosis of PCOS. We diagnosed our patient as phenotype D, which is the obese and non-androgenic type. However, a definitive diagnosis of PCOS in Japanese patients requires all 3 main characteristics [10]. The present Brazilian female PCOS patient had morbid obesity with marked subcutaneous and visceral adiposity, hyperinsulinemia, and multiple obesity-associated comorbidities without signs of hyperandrogenism, which is also extensively discussed by Gao et al. [5].

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