Nulliparous Woman with Polycystic Ovary Syndrome: Case Report

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Abstract. Background: Polycystic ovary syndrome (PCOS) is a heterogeneous disorder characterized by a combination of signs and symptoms of androgen excess (hirsutism, hyperandrogenemia) and ovarian dysfunction (oligo-ovulation, polycystic ovary morphology (PCOM)). In ASRM/ESHRE consensus in 2003, PCOS diagnosis was established by 2 out of 3 symptoms. Anovulatory infertility in polycystic ovary syndrome can be treated with clomiphene citrate and gonadotropins or laparoscopic ovarian surgery in clomiphene-resistant women.

Case Report: Mrs R, 30 years old, P0A0, came to Haji Adam Malik gynaecology polyclinic on Jun 6, 2022, at 09.30 with a chief complaint of irregular menstruation experienced five months ago. The patient had menstrual duration for 10–14 days with a total volume of 4–5 times changing sanitary napkins. On vaginal examination, menstrual blood was found, and on vaginal tussae examination, no abnormalities were found. After evaluating the Tanner stage, the development of pubic hair was found to be stage 4, breast stage 4, with a total Modified Ferriman Gallway Score was 5. On ultrasound examination, polycystic ovary syndrome (PCOS) was found. The patient was diagnosed with anovulation polycystic ovary syndrome (PCOS) and managed with lifestyle improvements.

Conclusion: A patient with polycystic ovary syndrome (PCOS) has been described based on ASRM/EHSRE criteria and treated with lifestyle modification as the primary treatment to improve hormonal and metabolic profiles, as the primary basis for PCOS management with obesity.

Keywords: polycystic ovary syndrome (PCOS) · anovulation · Modified Ferriman Gaallway score · Polycystic ovarian morphology

1 Introduction

Polycystic ovary syndrome (PCOS) is a heterogeneous disorder that affects at least 7% of adult women. According to the National Institutes of Health Office of Disease Prevention, PCOS affects about 5 million women of childbearing age in the United States [1]. In Indonesia, a study by Sumapraja et al. (2011) showed the highest PCOS frequency is in the 26–30 year age range, accounting for 45.7% [2]. The prevalence ranges between 5% and 15% depending on the diagnostic criteria. The diagnosis of PCOS is based on the presence of at least two of three following criteria: chronic anovulation,

M. M. Amin et al. (Eds.): ICoNaP 2022, AHSR 59, pp. 133–139, 2023.
https://doi.org/10.2991/978-94-6463-120-3_20

hyperandrogenism (clinical or biologic), and polycystic ovaries [3]. Some of the most commonly used treatments for chronic management of PCOS include hormonal contraceptives, progestins and metformin. Infertility treatment focuses on ovulation induction therapy, which may involve medications such as letrozole, Clomiphene, or gonadotropin. Weight loss in obese women with PCOS may benefit infertility treatment and long-term management [4].

2 Case Report

Mrs R, 30 years old, P0A0, came to Haji Adam Malik gynaecology polyclinic on Jun6, 2022, at 09.30 with a chief complaint of irregular menstruation that the patient had experienced five months ago. Her menstruation period lasted 10–14 days with a total volume of 4–5 times changing sanitary napkins. In addition, increased appetite and

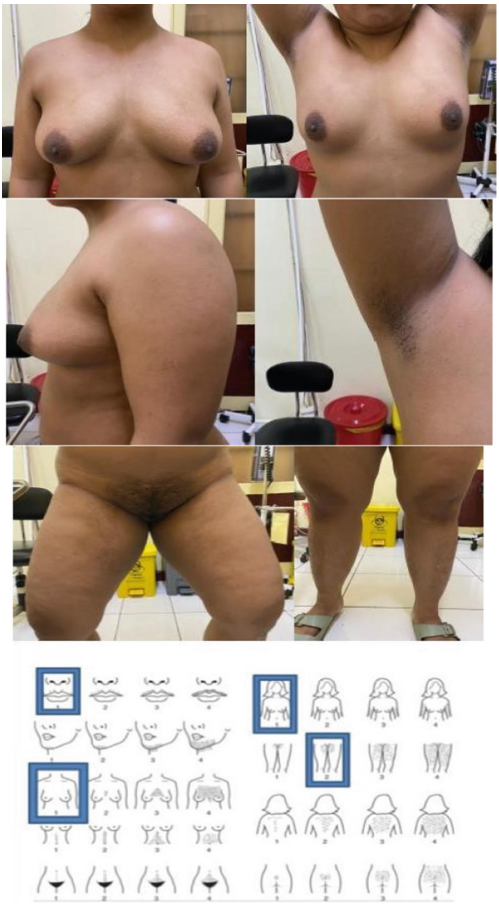

Fig. 1. Physical examination, showing patient's Modified Ferriman Gallway Score was 5.

weight gain of 15 kg in 9 months were found. Menarche was 12years old, with a regular 28-day cycle, for 3–5 days with dysmenorrhea.

On physical examination, the patient was compos mentis. Blood pressure was 119/75 mmHg, with heart rate of 80 bpm, respiratory rate of 20 rpm, and temperature of 36.7 °C. The patient's body mass index was 35.4 kg/m^2, indicating class II obesity. Examination of the head, neck, heart, thorax and extremities were all found within normal limits. Abdominal examination was found to be lax and normoperistaltic, with active bleeding due to menstruation. On vaginal examination, menstrual blood was found, and on vaginal tissue examination, no abnormalities were found. After Tanner's stage, the development of pubic hair was found to be stage 4, breast stage 4, with a total Modified Ferriman Gallway Score was 5 (Fig. 1).

On ultrasound examination, it was found that the bladder was filled, the uterus was ante flexed with the size of 4.50 × 3.67 × 3.07 cm, E-Thickness 8.18 mm, right ovary size 3.36× 2.91 cm, >7 follicles size 3.4 mm, left ovary size 3.02 × 2.90 cm, >9 follicles were found to be 4.0 mm, strongly appear to be typical of polycystic ovary syndrome (PCOS) (Fig. 2).

On laboratory examination found hemoglobin 13.4 g/dl; leukocytes 7,120/μl; hematocrit 42.5%; platelet 326,000/μl, MCV 81 fl; MCH 26.6 pg; MCHC 32.7%; neutrophils 62.4%; lymphocytes 27.1%; monocytes 7.2%; eosinophils 2.7%; basophils 0.6%. Hormonal examination found prolactin 13,84; estradiol 68.89pg/ml; testosterone 0.81 and free testosterone 6.82. Therefore, the patient was diagnosed with polycystic ovary syndrome (PCOS) and primarily managed with life style improvements.

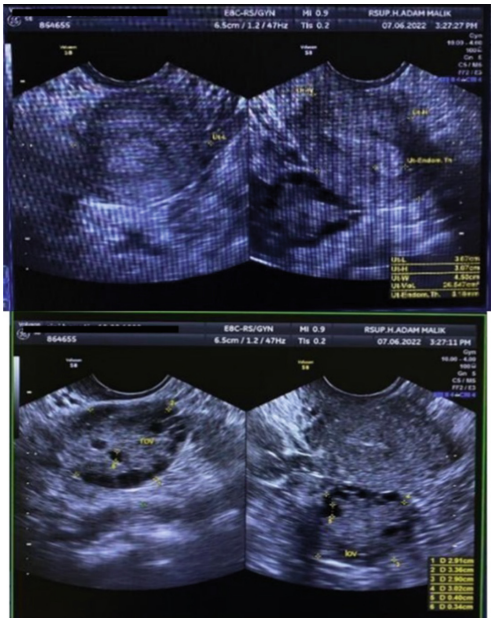

Fig. 2. Ultrasound examination concluded polycystic ovary syndrome (PCOS).

3 Discussions

Polycystic ovary syndrome (PCOS) is a heterogeneous disorder characterized by a combination of signs and symptoms of androgen excess (hirsutism, hyperandrogenemia) and ovarian dysfunction (oligo-ovulatory, polycystic ovary morphology (PCOM)).

PCOS can only be diagnosed when other diagnoses, such as hyperprolactinemia, and non-classical congenital adrenal hyperplasia, have been ruled out [5].

Various sources explain that PCOS occurs due to complex interactions between genetic and environmental factors. Insulin resistance and hyperinsulinemia are critical factors in the pathogenesis of ovulatory disorders and hyperandrogenism in PCOS. Increased adrenal activity is thought to cause phosphorylation of insulin receptors leading to insulin resistance. Hyperinsulinemia also reduces the production of sex hormone binding globulin (SHBG) in the liver, so free testosterone levels will increase [5].

Ovulation disorders in PCOS are caused by increased LH levels and decreased FSH levels. Under normal circumstances, folliculogenesis is facilitated by FSH, which regulates follicular growth and produces dominant follicle that is ready for ovulation. Inhibin secretion is increased indirectly by insulin and suppresses the production of FSH, which has an important role in folliculogenesis. In PCOS patients, hypersecretion of LH and insulin will cause premature luteinization and cessation of the ovarian follicle maturation process. The cessation of the ovarian follicle maturation process in its development will increase follicle number and produce polycystic ovarian morphology. In the ovaries of PCOS patients, there is hypertrophy of theca cell layer, which is in charge of synthesizing androgens [5].

The stimulating effect of LH secreted by the anterior pituitary is currently the proposed mechanism for anovulation and increased androgen levels found in PCOS. This leads to hyper stimulation of the ovarian theca cell that simultaneously increases androgens (e.g. testosterone, androstenedione) release. Due to decreased levels of FSH relative to LH, ovarian granulosa cells cannot convert androgens to estrogens, leading to decreased estrogen levels and consequent anovulation. Growth hormone (GH) and insulin-1 growth factor (IGF-1) may also enhance their effect on ovarian function [6].

Anovulation in PCOS is characterized by cessation of antral follicles, a phenotype that can be salvaged by increasing the circulating FSH concentrations. Abnormalities of regulation, secretion, and action of gonadotropins are implicated in anovulation etiology and local intraovarian factors. Recent studies have involved central and intraovarian actions of anti-Mullerian hormone (AMH) in PCOS aetiology and anovulation mechanisms [7].

The ASRM/ESHRE consensus in 2003 agreed that PCOS diagnosis was established by the presence of 2 out of 3 symptoms; (i) clinical or biochemical signs of hyperandrogenism; (ii) chronic ovulation disorders; and (iii) morphological features of polycystic ovaries found on Ultrasonography (USG). According to the Guastella study, phenotypes A and B (classic PCOS) are a group of patients withabdominal obesity, increased androgen values, LH values and LH/FSH ratio, and increased insulin values and insulin resistance. While phenotype C (ovulatory COP) is a mild form of classic PCOS, nonhyperandrogenic PCOS (phenotype D) is characterized by mild testosterone excess butdoes not show signs of hyperandrogenism [5]. In this patient, ultrasound examination showed an anteflexed uterus size 4.50 × 3.67 × 3.07 cm, E-Thickness 8.18 mm, right ovary size

3.36 × 2.91 cm, > 7 follicles size 3.4 mm, left ovary size 3.02 × 2.90 cm, > 9 follicles size 4.0 mm that are typical for polycystic ovary syndrome (PCOS).

Hirsutism is a marker for hyperandrogenism found in 70% of women with PCOS. However, signs of hyperandrogenemia still need to be evaluated biochemically in all women suspected of PCOS. Examination of hirsutism in PCOS generally uses the modified Ferriman – Gallwey score [5]. Our patient showed that the Tanner stage score of pubic hair was stage 4, breast stage 4, and the total Modified Ferriman Gallway score was 5. Ferriman Gallwey's score of ≤8 shows mild hirsutism, and a score ≥15 indicates severe hirsutism. A study by Karimah conducted on PCOS patients at RSCM obtained a cut-off point of 5, indicating hyperandrogenism [5].

Lifestyle modification is first-line therapy, which includes diet and physical activity interventions. Diet modification in women with PCOS has the effect of improving hormonal and metabolic profiles. The dietary plan must be based on a balanced diet by considering the glycemic index of carbohydrates consumed. Limitations of nutritional intake and exercise are the primary standard of PCOS management with obesity [5]. In this case, the preferred treatment is lifestyle modification. If anovulation persists, then therapy with Clomiphene or other ovulation induction drugs can be considered and if it still fails, in vivo fertilization can be the following approach if pregnancy is expected.

Anovulatory infertility in polycystic ovary syndrome can be treated with clomiphene citrate and gonadotropins or laparoscopic ovarian surgery in clomiphene-resistant women [7]. Several studies have shown that letrozole is better for ovulation induction than clomiphene citrate in PCOS patients [8–12]. Combined oral contraceptives, together with lifestyle modifications, are the first-line medical treatment for long-term management of PCOS [13]. Ovarian laparoscopy has been a successful second-line treatment for ovulation induction in cases of clomiphene-resistant PCOS as an alternative to gonadotropin therapy [14]. Metformin is an anti-insulin drug widely used in women with PCOS to treat metabolic comorbidities and improve ovarian dysfunction in women with PCOS [15]. A study also showed that metformin was effective in PCOS combined with lifestyle interventions [16]. Other studies have found that metformin has an advantage over placebo for live births but has side effects on the digestive system [17].

4 Conclusion

A patient with polycystic ovary syndrome (PCOS) has been described based on ASRM/EHSRE criteria. Lifestyle modification is the primary treatment to improve hormonal and metabolic profiles in obese individuals with PCOS.

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