Nat. Volatiles & Essent. Oils, 2021; 8(5): 991 - 996

NVEO
Natural Volatiles &
Essential Oils

Case Report on an adolescent with polycystic ovarian disorder

Ms. Sanjana Prakash Futane, Ms. Vaishali Balpande, Shital Telrandhe, S. R. Kapse,

1 GNM 3rd year student, Florence Nightingale Training College Of Nursing, Sawangi Meghe Wardha.

2 Nursing Tutor, Florence Nightingale Training College Of Nursing, Sawangi Meghe Wardha. Email: vaishalibalpande@gmail.com

3 Research Consultant, Jawaharlal Nehru Medical College, Datta Meghe Institute Of Medical Sciences , Wardha.

4 Dept. of Computer Technology, Yeshwantrao Chavan College of Engineering, Nagpur. Email: smitakapse@gmail.com

Abstract:
PCOD, or the most frequent endocrine-metabolic illness in reproductive-aged women is polycystic ovarian disorder (PCOD). Polycystic ovarian disorder is a complicated, heavily hereditary condition. PCOD has been linked to pathophysiological changes in gonadotropin production and among other things, ovarian folliculogenesis, steroidogenesis, insulin production and action, and adipose tissue function. A collect history and physical examination, as well as an a hirsutism examination, ovarian ultrasound, and hormonal tests to confirm hyperandrogenism and oligo-anovulation if necessary. In PCOD, therapeutic decisions are based on the patient's phenotype, concerns, and goals, and should aim to inhibit and counteract androgen secretion and activity, improve metabolic status, and improve fertility. Despite tremendous advances in comprehending the causes as well as a diagnosis condition in comparison to last twenty years, many practitioners continue to misdiagnose and misunderstand the disorder. Clinical finding is menstrual abnormalities include irregular menstruation, menstrual absence, Spotting, heavy menstruation, irregular menstruation, brief and moderate menstruation, and spotting are all symptoms of menopause. Therapeutic intervention it is a combined oral contraceptive comprising 20g to 35 g of ethinyl estradiol is the first-line pharmacological management for teenagers with polycystic ovarian disorder. This technique are effective for both hirsutism and acne. Then output is given the considerable decreases in quality of life as it relates to health reported among the affected client, PCOD is becoming an increasingly relevant teenage reproductive health diagnosis. Then conclusion it is in the majority of the research in this review, the IDF definition was used, which is the most recent and is standardised for teenagers by age group. summarises data on metabolic disorder in women including and excluding polycystic ovarian disorder.

Keywords- Ultrasonography, Cyst, Ovary, Menstruations, Hormones.

Introduction:

When you think of ultrasonography, you might think of cysts, overy, menstruation, and harmones.[1] Adolescent girls suffer from anovulatory cycles, obesity, and acne. PCOD can begin in utero, although the hypothalamic–pituitary–ovarian axis matures throughout adolescence, according to the androgen overexposure theory.[2] PCOD affects 11 to 26% of adolescents, with half of them being overweight. Although evidence suggests that ovarian hyperandrogenism and impaired insulin sensitivity are caused by environmental and genetic factors, the pathophysiology of polycystic ovarian disorder is still unknown.[3] Because the underlying causes of PCOD remain unknown, teenage girls' PCOD treatment is symptomatic. Hyperandrogenism is lowered and insulin sensitivity increases when weight loss and physical activity are coupled. In adults, Metformin has been shown to help people with insulin resistance, In a number of

studies, decreased testosterone levels and enhance menstrual cyclicity were found.[4] Metformin's use in the treatment of polycystic ovarian disorder and glucose intolerance in adolescents, however, is lacking in data.

Case report

Patient information:

A 15 year old girl was examined on September 20, 2021, for obesity and amenorrhea were assessed. The patient was the third kid in the family. Hypertension, hypercholesterolemia, and glucose intolerance affect both of her parents. Each of her mother's pregnancies resulted in gestational diabetes mellitus. Two healthy female siblings weighed in at around the same weight.[5]

At the age of eight, the patient began gaining weight fast. By the time of her presentation, her weight had risen from the tenth to the seventy-first centile during next seven years. Her height standard deviation scores (SDS) remained at K0.5. She suffered secondary amenorrhea after reaching menarche at the age of 14.

Medical and family history :

Birth control tablets to regulate periods, metformin to avoid diabetes, statins to reduce high cholesterol, hormones to improve fertility, and surgeries to remove unwanted hair are some of the treatments available.[6]

Relevant past interventions with outcomes :

Not reported

Clinical finding :

Menstrual abnormalities include irregular menstruation, menstrual absence, Spotting, heavy menstruation, irregular menstruation, brief and moderate menstruation, and spotting are all symptoms of menopause.[7]

Obesity, overweight, and weight gain are all synonyms used to describe the same condition.

Infertility, acne, dark skin patches in folds and wrinkles, sadness, unattractive male characteristics, hair loss, oily skin, or unwanted hairs are all common issues.

Timeline :

Patient was polycystic ovarian syndrome with chief complaint of heavy menses, wait gain, and loss of scalp hair, since 5 to 6 month.

Therapeutic intervention :

A combined oral contraceptive comprising 20g to 35 g of ethinyl estradiol is the first-line pharmacological management for teenagers with polycystic ovarian disorder. This technique are effective for both hirsutism and acne.[8]

Diagnostic evaluation :

Blood test :- Hb- 10.2% g

Physical examination - abdominal examination.

Transvaginal ultrasound

Assessment

The patient was extremely overweight, with a stature of 160.8 cm (standard deviation K0.5), a weight of 83.0 kg (71 percentile), a BMI of 32 kg/m2, 85 cm waist circumference and 135/84 blood pressure. Tanner was in pubertal stage B4,P4 when he was discovered. The patient had minor acne and hirsuteness (Ferriman-Gallway score 12). Her acanthosis nigricans had not manifested itself. FSH, oestrogen, and progesterone levels were normal in the blood, but the levels of testosterone and luteinizing hormone were both elevated abnormally high. When the experiment began, TSH, free thyroxine, prolactin, and 24-hour free urine cortisol levels were all within normal ranges. The blood levels of transaminases and creatine were likewise within permissible limits. Throughout the entire test, she developed severe hyperinsulinemia. The serum lipid profile showed an increase in total cholesterol, low density lipoprotein (LDL) cholesterol, triglycerides, and low high density lipoprotein (HDL) cholesterol. A transrectal ultrasonography of the pelvis on both sides revealed polycystic ovaries. PCOD was verified by ultrasound findings of polycystic ovary, secondary amenorrhea with hirsutism, and a high testosterone level. A nutritional therapist and a physiotherapist assessed her and recommended lifestyle changes to help her lose weight and improve physical activity. Metformin was prescribed to assist her begin menstruation and increase her glucose tolerance. Dydrogesterone was developed to increase glucose tolerance and produce menses on days 15 to 20 of the menstrual cycle. Dydrogesterone was developed to increase glucose tolerance and produce menses on days 15–24 of the menstrual cycle. After losing 4.5 kg in six months, her BMI dropped to 30.3 kg/m2. The lipid profile and glycemic tolerance improved somewhat. Hyperinsulinemia was no longer a problem. The LDL-receptor was checked for alterations because of a family history of hypercholesterolemia, but the results were negative. The lipid profile and glycemic tolerance improved somewhat. Hyperinsulinemia was no longer a problem. After dydrogesterone discontinuation, menstruation began, and metformin dosage was raised. Fasting glucose and insulin levels were normal, and the lipid profile had improved. Despite the presence of polycystic ovaries, regular ovulatory menstrual periods were restored without the use of progesterone. The patient is still taking metformin after 4.5 years. She is still overweight and struggles to maintain her fitness. Glucose and insulin levels, on the other hand, have climbed.

Discussion:

PCOD is defined as a disorder of ovarian dysfunction if two or more of the following conditions are present. oligo or an ovulation for more than six months, chemical and biochemical evidence of hyperandrogenism, and polycystic ovaries on ultrasound. Other conditions with symptoms that resemble PCOD should be

cleared out.[9] This patient met all of the PCOD criteria. She was amenorrhoeic, with clinical and biochemical symptoms of hyperandrogenemia and polycystic ovaries, according to ultrasonography. Natural levels of estradiol and gonadotropin ruled out primary ovarian failure as well as hypo- or hypergonadotropic hypogonadism. Since prolactin and the levels of progesterone were within normal limits, Late-onset congenital adrenal hyperplasia and hyperprolactinemia were ruled out. There were no signs of hypercortisolism or thyroid illness, however. PCOD is frequently misdiagnosed during youth. Although clinical and metabolic signs are similar to those seen in adults, distinguishing between a young PCOD lady and a regular adolescent can be challenging.[10] 95 percent of teenagers with irregular periods have PCOD. Many women are using the oral contraceptive pill to mask their condition before trying to conceive. PCOD is often associated with irregular menses, such as periods lasting less than 21 days or more than 35 days. This is something clinicians should be aware of.

Women with PCOD do not have clinical or biochemical symptoms of hyperandrogenism while having polycystic ovaries. Our patient exhibited a high blood testosterone level as well as signs of moderate hyperandrogenemia. The most common circulating androgen in women is testosterone, which is produced from ovarian and adrenal precursors (mostly androstenedione and dehydroepiandrostenedione) and then metabolised in peripheral tissues. Teen PCOD patients may have normal androgen levels and minimal hirsutism when compared to adult PCOD patients.[11] Hyperandrogenemia can be diagnosed with a reliable immunoassay that measures total testosterone. Adolescents with androgen-secreting tumours are rare, but they must be ruled out. Although androgen-secreting tumours are uncommon, they must be excluded. 8 A high increase in blood testosterone (8.7 nmol/l, two to three times the upper normal limit) is strongly predictive of an androgen-secreting ovarian tumour when DHEA-sulfate (DHEAS) levels are normal. An adrenal androgen-secreting tumour is suspected when DHEAS is elevated (O16.3 mmol/l). Insulin resistance is a significant factor to the physiopathology of PCOD.[12] Insulin sensitivity declines throughout puberty, resulting in increased insulin production. Obese adolescent females with PCOD symptoms generated more insulin and exhibited lower insulin sensitivity than weight-matched controls. Prior to treatment, our patient had substantial hyperinsulinemia and poor glucose tolerance, but these problems were alleviated with lifestyle changes and metformin therapy. In a study of highly obese PCOS patients, researchers discovered a high prevalence of impaired glucose tolerance and type 2 diabetes in first-degree relatives, demonstrating that PCOD patients' poor glucose metabolism is genetically influenced. Our patient's parents had metabolic disorder despite their normal weight. Screening is advised for first-degree relatives of PCOD patients due to an increased risk of metabolic disorders.

PCOD is treated symptom by symptom. Lifestyle adjustments are a first-line treatment for overweight PCOD women. Diet and exercise, as well as healthy weight management, can help with glucose intolerance. Metformin improves insulin sensitivity and glucose metabolism in teenagers, as well as lowering hyperandrogenism and irregular menses. Metformin also aids in the maintenance of a healthy lipid profile. During a 4.5-year period, this example, which clearly profited from such treatment, had no negative consequences. On the other hand, questions like how long care should be provided and long-term protection remain unaddressed. Positive effects disappear soon once treatment is stopped, according to Ibanez et al. PCOD is a chronic condition that lasts a lifetime. As a result, patients should be constantly watched throughout their adolescence and maturity.[13, 14] Few studies on related aspects of ovarian disorders were reviewed[15-20].

Learning points

1. If an adolescent's menses are irregular and she is overweight, PCOD should be considered.

2. The metabolic syndrome is linked to PCOD. Obesity needs glucose intolerance and dyslipidemia screenings.

3. For overweight young women with PCOD, lifestyle changes are the first treatment.

4. Metformin treatment in the case of PCOD adolescent is effective and the short-term consequences are uncertain, while the long-term effects are well tolerated.

Conclusion :

In the majority of the research in this review, the IDF definition was used, which is the most recent and is standardised for teenagers by age group. summarises data on metabolic disorder in women including and excluding polycystic ovarian disorder.

References:

1. van Hooff MH, Voorhorst FJ, Kaptein MB, Hirasing RA, Koppenaal C, Schoemaker J. Polycystic ovaries in adolescents and the relationship with menstrual cycle patterns, luteinizing hormone, androgens, and insulin. Fertility and sterility. 2000 Jul 1;74(1):49-58
2. Jardim N. Fix Your Period: Six Weeks to Life-Long Hormone Balance. Random House; 2020 Apr 30.
3. Ibáñez L, Oberfield SE, Witchel S, Auchus RJ, Chang RJ, Codner E, Dabadghao P, Darendeliler F, Elbarbary NS, Gambineri A, Rudaz CG. An international consortium update: pathophysiology, diagnosis, and treatment of polycystic ovarian syndrome in adolescence. Hormone research in paediatrics. 2017;88:371-95.
4. Conway G, Dewailly D, Diamanti-Kandarakis E, Escobar-Morreale HF, Franks S, Gambineri A, Kelestimur F, Macut D, Micic D, Pasquali R, Pfeifer M. The polycystic ovary syndrome: a position statement from the European Society of Endocrinology. European journal of endocrinology. 2014 Oct 1;171(4):P1-29.
5. National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. diabetes. 1979 Dec 1;28(12):1039-57.
6. Setji TL, Brown AJ. Polycystic ovary syndrome: update on diagnosis and treatment. The American journal of medicine. 2014 Oct 1;127(10):912-9.
7. Somwanshi SB, Gaikwad VM, Dhamak KB, Gaware VM. WOMEN‟ S HEALTH ISSUE: A BRIEF OVERVIEW ON IRREGULARITIES IN MENSTRUATION.
8. Krowchuk DP, Lucky AW. Managing adolescent acne. Adolescent Medicine Clinics. 2001 Jun 1;12(2):355.
9. Williams T, Mortada R, Porter S. Diagnosis and treatment of polycystic ovary syndrome. American family physician. 2016 Jul 15;94(2):106-13.
10. Williams T, Mortada R, Porter S. Diagnosis and treatment of polycystic ovary syndrome. American family physician. 2016 Jul 15;94(2):106-13.

11. Chang RJ. A practical approach to the diagnosis of polycystic ovary syndrome. American journal of obstetrics and gynecology. 2004 Sep 1;191(3):713-7.
12. Lowenstein EJ. Diagnosis and management of the dermatologic manifestations of the polycystic ovary syndrome. Dermatologic therapy. 2006 Jul;19(4):210-23.
13. Teede H, Deeks A, Moran L. Polycystic ovary syndrome: a complex condition with psychological, reproductive and metabolic manifestations that impacts on health across the lifespan. BMC medicine. 2010 Dec;8(1):1-0.
14. Pasquali R, Gambineri A. Glucose intolerance states in women with the polycystic ovary syndrome. Journal of endocrinological investigation. 2013 Sep;36(8):648-53.
15. Acharya, N., Acharya, S., Shukla, S., Joshi, K., Gopal, U., 2020a. Polycystic ovarian syndrome (Pcos) in obese metabolic phenotypes. International Journal of Current Research and Review 12, 16–18. https://doi.org/10.31782/IJCRR.2020.SP62
16. Deshpande, S.S., Phatak, S.V., 2019. A rare case of bilateral multiple ovarian dermoids with uterine fibroid and ectopic kidney. Journal of Datta Meghe Institute of Medical Sciences University 14, 39–41. https://doi.org/10.4103/jdmimsu.jdmimsu_79_18
17. Laishram, S., Gupta, V., Bhake, A., Wankhede, A., Agrawal, D., 2019. To assess the utility of proliferative marker Ki-67 in surface epithelial ovarian tumor. Journal of Datta Meghe Institute of Medical Sciences University 14, 6–10. https://doi.org/10.4103/jdmimsu.jdmimsu_71_18
18. Tapadia, S., Phatak, S., Singh, V., 2020a. Ovarian torsion: Ultrasonography and color doppler imaging. Journal of Datta Meghe Institute of Medical Sciences University 15, 465–467. https://doi.org/10.4103/jdmimsu.jdmimsu_100_20
19. Chadha, A.M., Salve, M., Shrivastava, D., Deo, A.A., 2019. Osseous endometrial metaplasia in a case of primary infertility. International Journal of Infertility and Fetal Medicine 10, 52–53. https://doi.org/10.5005/jp-journals-10016-1189
20. Abbafati, C., Machado, D.B., Cislaghi, B.,2019 D., 2020d. Global age-sex-specific fertility, mortality, healthy life expectancy (HALE), and population estimates in 204 countries and territories, 1950–2019: a comprehensive demographic analysis for the Global Burden of Disease Study 2019. The Lancet 396, 1160–1203. https://doi.org/10.1016/S0140-6736(20)30977-6