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CLINICAL, METABOLIC, ENDOCRINE AND SONOGRAPHIC CHARACTERISTICS OF OBESE ADOLESCENT WITH PCOS

Gynaecology

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ABSTRACT

Introduction- PCOS is a complex metabolic, endocrinopathy and reproductive disorder that results in the production of androgens and is associated with insulin resistance.
Adolescents with PCOS are more obese than normal adolescents and have an increased risk of metabolic syndrome.
In obese adolescent increasing abdominal adiposity, worsening insulin sensitivity and Dyslipoproteinemiadyslipoproteinemia give rise to NAFLD in adulthood.
This study aimed to investigate the association between PCOS, obesity, NAFLD and Metabolic syndrome in adolescents with PCOS.
MATERIAL AND METHOD- Retrospective study of 24 obese adolescent PCOS patients. with mean BMI 32.89 kg/m2. Data were taken from 1st January 2015 to 31st December 2020. PCOS patients were diagnosed according to Rotterdam 2003 criteria. Data were collected from the medical records of the patients including clinical history, height, weight, blood pressure, waist circumference and Modified Ferriman-Gallway score for hirsutism. Laboratory values were obtained ultrasound finding of polycystic ovarian disease and fatty liver were obtained from the records.
RESULTS:- In our study, the mean age was 15.2 yr The mean body mass index was 32.89 kg/m^2 and the mean age of menarche was 12.3 yr. Hirsutism was present in 75% by modified FG Score system, Family history of PCOS was present in 29.16%, Family history of Cardiovascular disease was present in 12.5%. A family history of diabetes mellitus was present in 20.83%. The majority 87.5% of the obese adolescent girl's were presented with menstrual problems. mostly 70.83% with a history of Oligomenorrhea. Waist circumference, >0.80 was present in 87.5%, 50% of patients were hypertensive. 20.83% were prediabetes, 54.16% of patients were found to have dyslipidaemia and 45.83% of patients had Metabolic syndrome. In our mean LH was 10.6 IU/L, mean FSH was 5.8 IU/L, mean Serum testosterone 18.5 ng/ml, mean Serum prolactin was 21.2 ng/mL and mean Serum TSH was 3.3 µIU/mL.75 % of patients had Polycystic appearing ovarian morphology in USG.45.83 % of obese PCOS patient had NAFLD in USG and 33.33% of obese patients had abnormal liver enzymes with NAFLD in USG finding.
CONCLUSION:- In our study, we found both metabolic syndrome and NAFLD were frequent in patients with obese adolescent PCOS confirming a relevant clinical association between these three conditions.
This study highlights the importance of preventing obesity during the management of adolescent PCOS. The importance of obesity counselling in obese adolescent women is a must to reduce the risks associated with metabolic syndrome.
The therapeutic intervention combined with lifestyle modification may provide better treatment for adolescent PCOS.

KEYWORDS

Polycystic syndrome, Metabolic Syndrome, Hirsutism, Obese.

1. INTRODUCTION

The polycystic syndrome is a heterogeneous endocrine condition that affects approximately 5-10% of women in the reproductive age group.[1]

Different studies in India on PCOS have reported a prevalence of 3.7% to 22.5% and even up to 36% in adolescents.

The worldwide prevalence of polycystic ovarian syndrome ranges from 2.2 to 26% [2]The rates of polycystic ovarian syndrome have been reportedly high among Indian women compared to their Caucasian counterparts[3], with an estimated prevalence of 9.13% in Indian adolescents [2,1]

PCOS is a multi-system endocrinopathy involving women from menarche to menopause. In essence, PCOS is a complex metabolic, endocrinopathy and reproductive disorder that results in the production of androgens and is associated with insulin resistance.

In 2003, The Rotterdam consensus defined that the presence of at least two out of the three main characteristics was necessary to confirm the diagnosis. (Table 1.)

Rotterdam Criteria for Diagnosis of PCOS (PCOD) (2003)[3]
• Oligo-ovulation or anovulation. • clinical of biochemical evidence of hyperandrogenism. • polycystic ovarian morphology on ultrasonography is defined as >12 follicles with a 2 to 9 mm diameter or an ovarian volume <10ml. the three sets of criteria are illustrated in the table. with the exclusion of medical conditions such as congenital adrenal hyperplasia, androgen-secreting tumors of Cushing's syndrome.

Consensus on the women health aspect of PCOS has suggested different criteria for diagnosis of PCOS in adolescents from those used for adults. According to its suggestions, PCOS in adolescent should include all three elements of Rotterdam criteria in which oligomenorrhea should be present after two years of menarche or primary amenorrhea at the age 16 years; polycystic ovaries on ultrasound along with ovarian size of more than 10 cn3 and hyperandrogenemia should be present.[4]

However, Androgen Excess and PCOS Society (AEPCOS) in 2006 tightened the criteria and suggested including all the three Rotterdam Criteria to make the diagnosis of PCOS.

Clinical presentations of the polycystic ovarian syndrome include abnormal facial and skin hair growth (hirsutism), acne and irregular or absence of, menstrual periods.[5] However acne is most common during the adolescent phase of life and there is limited literature on adolescent androgenic alopecia.[4]

The determinants of polycystic ovarian syndrome have been linked to both hereditary and environmental factors.[6] the attributed hereditary factors include early age of sexual maturation, premature fetal development, and family history of PCOS among first-degree relatives.[6,7]Studies have reported an earlier age at diagnosis of PCOS (9-12 years). among adolescent females with earlier maturation of sexual characteristics compared to their later counterparts (13-18 years)[7] This has been attributed to an increased androgen secretion associated with early onset of puberty.[6]It has been reported that premature fetal development leads to an earlier and more rapid onset of puberty with an increased risk of developing PCOS.[8]Clinical manifestation of associated symptoms such as hyperinsulinemia have also been observed in offspring of PCOS affected women long before the onset of puberty affirming the role of family history.[8]

The associated environmental factors reported include physical

inactivity, obesity, and its associated insulin resistance[5] **Insulin resistance which is of high prevalence in the Indian Population.[5] has been consistently reported as a strong determining factor for the occurrence of PCOS in Indian adults and adolescents**.[6]

It is proven that insulin resistance and resultant hyperinsulinemia are observed in 50-70% patients of with PCOS Hyperinsulinemia results from increased basal secretion of insulin and decreased hepatic clearance of insulin.[9]

Insulin also stimulates LH to cause hyperplasia of ovarian theca cells which then secrete more androgen, testosterone and epi androstenedione. High androgen levels inhibit the growth of the dominant follicle and prevent apoptosis of smaller follicles. The persistence of many small follicles is a hallmark of PCOS.[9]

PCOS leads to several long term sequelae like dyslipidemia, diabetes, hypertension, cardiovascular disease (CVD), pregnancy-associated disorders, malignancies, sleep disorders, non-alcoholic fatty liver disease and metabolic syndrome. [11]

A metabolic syndrome is a group of metabolic dysfunctional features, including abdominal obesity, insulin resistance, hyperglycemia due to impaired glucose metabolism, high blood pressure, and abnormal blood lipid levels.

Adolescents with PCOS are more obese than normal adolescents and have an increased risk of metabolic syndrome. It is suggested that abdominal adiposity increases the risk of metabolic syndrome by inducing various cytokine secretions.

Recent studies have demonstrated an association between PCOS and other metabolic complication like nonalcoholic fatty liver disease (NAFLD). (NAFLD) occurs as a result of abnormal lipid handling by the liver, which sensitizes the liver to injury and inflammation. NAFLD is more common in PCOS because of insulin resistance and hyperandrogenemia.[10]In adolescents, The overall prevalence of Fatty liver remains lower than in adult women.

Diagnosing polycystic ovary syndrome(PCOS) during adolescence is challenging because features of normal pubertal development overlap with adult diagnostic criteria.
Differential diagnosis of PCOS includes congenital adrenal hyperplasia (late-onset) hyperthecosis, Cushing syndrome, hyperprolactinemia, hypothyroidism, and ovarian and adrenal androgen-secreting tumors.[4]

Therapeutic strategies to decrease abdominal obesity and reduce insulin resistance and dyslipidemia early in the course of PCOS in youth may halt future NAFLD in adulthood. The increased risk of developing Type 2 diabetes and its associated comorbidities during later years can be controlled by identifying high-risk populations and implementing preventive measures. The therapeutic intervention combined with lifestyle modification provides effective treatment for adolescent PCOS.

2. MATERIALAND METHODS

This is a retrospective study of 24 obese adolescent PCOS patients with a mean BMI of 31.39 kg/m2 from 1st January 2015 to 31st December 2020. PCOS patients were diagnosed according to Rotterdam 2003 criteria. Data were collected from the medical records of the patients including clinical history, height, weight, blood pressure, waist circumference and Modified Ferriman-Gallway score for hirsutism. Laboratory values were obtained like, Fasting Glucose level, fasting insulin level, Amino transference level AST, ALT level, AST/ALT Ratio, Total cholesterol, HDL, LDL and Triglyceride level. Hormone values like LH, FSH, LH/FSH ratio, Free testosterone, TSH and prolactin were also obtained from the records.Abdominal ultrasonography was used to determine the presence of NAFLD. Ovarian ultrasound completed trans abdominally or transvaginally if a patient is sexually active. (Polycystic ovarian morphology was seen.) Presenting complaints were menstrual disorders, hirsutism, obesity, infertility, etc which were noted.

Oligomenorrhea was defined as menstrual cycle >35 days, or less than eight cycles per year after 2 years of menarche; primary amenorrhea as amenorrhea by age 15 years, or >3 years post thelarche; secondary amenorrhea as the cessation of menstruation for at least 6 months in a previously menstruating girl; and polymenorrhea as menstrual cycle <21 days.

Assessment of hirsutism was done by the modified Ferriman–Gallwey (FG) score; a score of ≥8 was the cutoff point. Oral glucose tolerance test (OGTT) with a 75-g glucose load was carried out in all participants after overnight fasting for at least 8 hours; fasting plasma glucose (FPG) and plasma glucose 2 hours after OGTT (PG 2H-OGTT) were measured by the glucose oxidase method using a fully automatic biochemistry analyser. Diagnosis of prediabetes and diabetes mellitus was established according to the criteria described by the American Diabetes Association.

Metabolic syndrome diagnoses by both the definition proposed by the National Cholesterol Education Program (NCEP) and the International Diabetic Federation (IDF). Metabolic syndrome was diagnosed using the International Diabetes Federation consensus definition of metabolic syndrome in children and adolescents; abdominal obesity plus two or more of:

- Raised blood pressure: systolic BP≥130 mmHg or diastolic BP≥85 mmHg, or treatment of previously diagnosed hypertension;
- Impaired fasting glucose: FPG≥5.6 mmol/L, or previously diagnosed type 2 diabetes;
- Raised tirglycerides (≥150 mg/dL); or reduced high-density lipoprotein (HDL)-cholesterol (<50 mg/dL), or specific treatment for these lipid abnormalities.

Ultrasound finding of polycystic morphology - Ovaries are enlarged in volume (≥10 $cm^{3)}$ Increased number (≥12) of peripherally arranged follicles per ovary (2-9mm) are seen.

For adolescents aged 17-19 years, awaits circumference ≥of 80cm was defined as abdominal obesity. In the adolescents (aged 10-16 years), waist circumference ≥90^{th} percentile (or adult cut off is lower) was used.

Fatty liver was diagnosed with increased hepatic brightness or hyperechogenicity in USG.

All the data were analyzed using IBM SPSS Ver.20 software. Cross tabulation and frequency distribution were used to prepare tables. Data are expressed as numbers, percentage and mean.

3. RESULTS

Table 1: Demographic and clinical characteristics.

Age (yr)	Mean 15.2
BMI (kg/m^2)	Mean 32.89
Age at menarche (yr)	Mean 12.3
F-G score	
Absent (<8)	6 (25%)
Mild (9-16)	3 (12.5%)
Moderate (17-25)	13 (54.16%)
Severe (>25)	2 (8.33%)
Presence of Acanthosis nigricans (%)	5 (20.83%)
Family history	
FHof PCOS	7 (29.16%)
FH of Cardiovascular disease (%)	3 (12.50%)
FH of Diabetes mellitus (%)	5 (20.83%)

In our study, the mean age was 15.2 yr. The mean body mass index was 32.89 kg/m2 and the mean age of menarche was 12.3 yr.

Hirsutism was present in 75% of cases estimated by the modified FG Score system. Acanthosis nigricans was present in 20.83%. Family history of PCOS was present in 29.16%, Family history of Cardiovascular disease was present in 12.5%. A family history of diabetes mellitus was present in 20.83%.

Table:- 2 Chief complaints of the patient attending the clinic

Symptoms	Number	Percentage
1. Menstrual Problem	**21**	**87.5%**
Oligomenorrhea	17	70.83%
Polymenorrhea	3	12.50%
Secondary amenorrhea	3	12.50%

Primary amenorrhea	1	4.16%
2. Hirsutism	18	75%
3. Obesity	24	100%
4. Acne and oily skin	6	25.00%
5. Others-Acanthosis Nigerians, Loss of scalp hair, sleep apnea, snoring and other gynec problems, depression and anxiety.	8	33.33%

The majority of the obese adolescent girl's were presented with menstrual problems 87.5%(mostly with a history of Oligomenorrhea 70.83% followed by secondary amenorrhea 12.5%, polymenorrhea 12.5% and primary amenorrhea 4.16%).75% of patients had hirsutism. obesity was present in 100% of patients. 25% of patient had acne or oily skin and 33.33% of patient had other symptoms.

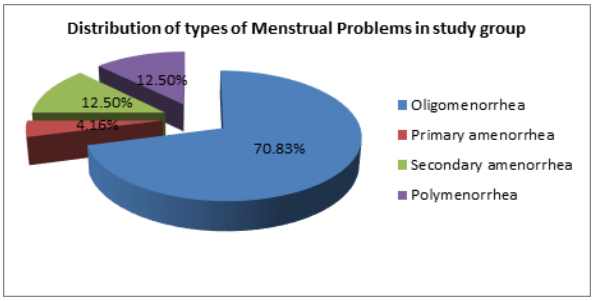

Figure 1: Distribution of types of menstrual problems in the study group

Oligomenorrhea was the main menstrual problem.

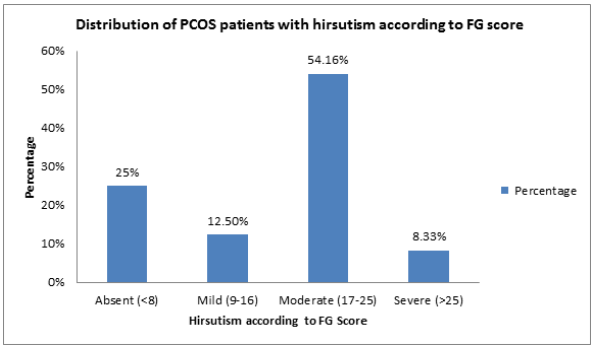

Figure 2:Distribution of PCOS patients with hirsutism according to FG score.

Most of the patients showed moderate hirsutism.

Table 3: Metabolic characteristics of study participants

Metabolic Characteristics	Number	Percentage
Waist circumference, >0.80	21	87.5%
BP status, n (%) Raised blood pressure: systolic BP≥130 mmHg or diastolic BP≥85 mmHg, or treatment of previously diagnosed hypertension;		
Normal	8	33.33%
previously diagnosed hypertension	4	16.66%
HTN	12	50%
Glycaemic status, n (%) Increased fasting glucose: >110Mg/dl or previously diagnosed type 2 diabetes; OGTT- 2 hr glucose- Impaired- 140 to 199 mg/dl Diabetes- >200		
Normoglycaemia	17	70.83%
previously diagnosed diabetes	1	4.16%
Prediabetes (Impaired OGTT)	5	20.83%
Diabetes mellitus, n (%)	1	4.16%
Dyslipidaemia, n (%) Raised tirglycerides (≥150 mg/dL); or reduced high-density lipoprotein (HDL)-cholesterol (<50 mg/dL)		
Present	13	54.16%
Absent	11	45.83%
Metabolic syndrome, n (%)		
Present	11	45.83%
Absent	13	54.16%

In our study, Waist circumference, >0.80 was present in 87.5%, hypertensive patients were 50%, 70.83% were found to have normoglycaemia and 20.83% had prediabetes, 54.16% of patients were found to have dyslipidaemia, 45.83% patients had Metabolic syndrome.

Table:- 4 Hormonal characteristics of the study participants

Hormone	Mean Value
Luteinizing hormone (IU/L)	10.6
Follicle-stimulating hormone (IU/L)	5.8
Serum testosterone, (ng/ml)	18.5
Serum prolactin (ng/mL)	21.2
Serum TSH (µIU/mL)	3.3

Mean LH was 10.6 IU/L, mean FSH was 5.8IU/L, mean serum testosterone 18.5ng/ml, mean Serum prolactin was21.2 ng/mL and mean Serum TSH was 3.3 µIU/mL.

Table:-5 Ultrasound characteristics
OVARIAN MORPHOLOGY

Ovarian morphology on USG	Number	Percentage
Polycystic appearing ovaries in USG in per cent	18	75
Enlarged mean ovarian volume >10 cm^3	15	62.5

75 % of patients had polycystic appearing ovarian morphology in USG whereas- 62.5% had increased ovarian volume.

Table 6: PCOS patient showing NAFLD by USG and liver enzymes

NAFLD	Number	Percentage
NAFLD by USG- Increased hepatic brightness of hyperechogenicity in USG (Fatty liver)	11	45.83
NAFLD by Liver biochemistry- Patient with Abnormal liver Chemistry (Higher Alanine aminotransferase (ALT)	8	33.33

45.83 % of obese PCOS patient had NAFLD in USG and 33.33% of obese patients had abnormal liver enzymes with NAFLD in USG finding.

4. DISCUSSION

PCOS is associated with multiple abnormalities including obesity, hypertension, dyslipidemia, and insulin resistance[12]

In our study, the mean age was 15.2 yr The mean body mass index was 32.89 kg/m^2 and the mean age of menarche was 12.3 yr.

Hirsutism was present in 75% by modified FG Score system, Acanthosis nigricans was present in 20.83%. Family history of PCOS was present in 29.16%, Family history of Cardiovascular disease was present in 12.5%. A family history of diabetes mellitus was present in 20.83%. In one another study in 22 obese PCOS mean age was 15.5 ± 1.4 (range, 12.9-18.3), the mean BMI was 35.9 ± 6.2 (range, 25.5-48.2), mean age at menarche was 11.9 ± 1.4 (range, 10-15). in that study acanthosis nigricans was present in (91%) which was higher than our study.[13]Family history of PCOS, cardiovascular disease and diabetes mellitus was higher in other studies.[14]

The majority of the obese adolescent girls presented with menstrual problems 87.5% (mostly with a history of Oligomenorrhea 70.83% followed by secondary amenorrhea 12.5%, polymenorrhea 12.5% and primary amenorrhea 4.16%). 75% of patients had hirsutism. 54.16% obesity was present in 100% of patients. 25% of patient had acne or oily skin and 33.33% of patient had other symptoms. In another study initial reason for the visit included irregular menses in 75.3%, acne or hirsutism in 7.1% and other reason in 17.5%. In a study from Bangladesh, 88% of the patient had oligomenorrhea, 2.3% had primary amenorrhea, 6.9% had secondary amenorrhea (6.9%) and 2.9% had polymenorrhea.[14]In our study hirsutism was present in 75% of a patient while other studies showed 85.7%, 94.9%, similar to our study results.[15,14]

In our study waist circumference, >0.80 was present in 87.5%, hypertensive patients were 50%, 70.83% were found to have normoglycaemia and 20.83% had prediabetes, 54.16% of patients were found to have dyslipidaemia, 45.83% patients had Metabolic syndrome. In one study from Bangladesh, a total of 64.7% had abdominal obesity,20% of participants had pre-hypertension and 3.4% were hypertensive. Around 24% had abnormal glucose tolerance (prediabetes 21.1%, diabetes 2.9%) and the majority (90.9%) had dyslipidaemia.[14]In another study it was found that adolescent PCOS patients had higher triglyceride and LDL level and lower HDL level. [16]Presence of metabolic syndrome was higher in our study in comparison to other studies in which it was 42.3% because our study had 100% obese patients.[14, 17]The prevalence of metabolic syndrome in the obese girls with PCOS was strikingly higher than the prevalence in the general obese adolescent population in NHANES.[18]In one study the prevalence of metabolic syndrome in overweight / obese girls is not increased in the presence of PCOS or polycystic ovary morphology.[19]The incidence of metabolic syndrome in PCOS increased with age and body mass index (BMI), with the most prevalent condition being low/high-density lipoprotein-cholesterol.[20]

In one study the obese adolescent PCOS group had significantly higher fasting insulin, low-density lipoprotein cholesterol, free testosterone levels and 2-h glucose during the oral glucose tolerance test.[21]

Mean LH was 10.6 IU/L, mean FSH was 5.8 IU/L, mean Serum testosterone 18.5 ng/ml, mean Serum prolactin was 21.2ng/mL and mean Serum TSH was 3.3 µIU/mL. In one another study mean LH in obese PCOS was 9.5, mean FSH 5.5.which was comparable with our study but free testosterone level was found much higher than our study 49.8(ng/dl) in obese PCOS patient.[13]

75% of patients had Polycystic appearing ovarian morphology in USG whereas- 62.5% had increased ovarian volume. In one another study, 75% of obese PCOS had Polycystic appearing ovarian morphology in USG and mean ovarian volume (ml) 8.2 ±4.4.[13]

In one other study, polycystic ovaries by ultrasound were found in 83.3% of obese girls with PCOS.[19]

45.83 % of obese PCOS patient had NAFLD in USG and 33.33% of obese patients had abnormal liver enzymes with NAFLD in USG finding. In another study prevalence of NAFLD in obese cohorts of PCOs is estimated to be between 60%-90%. Many studies showed increased NAFLD in obese PCOS patients.

5.CONCLUSION

Obesity is a leading precursor of poor metabolic health and is an emerging problem in India. The application of this retrospective study is to alert the gynaecologist and physicians to use some easy and accessible tools to diagnose metabolic risk early and prevent future co-morbidities.

Obesity and Insulin Resistance are important risk factors for metabolic syndrome in PCOS. Considering the long–term health risk, it is necessary to identify metabolic disorders in adolescents with PCOS as early as possible. We concluded that: Obesity, metabolic disorders and PCOS in adolescents are associated. Obesity exacerbates metabolic disorders in adolescent PCOS.

In obese adolescent increasing abdominal adiposity, worsening insulin sensitivity and Dyslipoproteinemia give rise to NAFLD in adulthood. NAFLD can lead to hepatitis and or cirrhosis, so it is important to screen for it early.

This study indicates that obesity should be prevented during the management of adolescent PCOS. The importance of obesity counselling in obese adolescent women is a must to reduce the risk associated with metabolic syndrome. The therapeutic intervention combined with lifestyle modification may provide better treatment for obese adolescent PCOS.

6. REFERENCES

1. Azziz R. Woods KS, Reyna R, et al. the prevalence and features of the polycystic ovary syndrome in an unselected population. J Clin endocrinil Metab. 2004;89 (6): 2745-9.
2. Zawadzki JK, Dunaif A. Diagnostic criteria for polycystic ovary syndrome: towards a rational approach. In: Dunaif AGJ, Haseltine F (eds). Polycystic Ovary Syndrome, Boston MA: Blackwell Scientific, 1992;377–84.
3. Rotterdam ESHRE/ASRM-Sponsored PCOS Consensus Workshop Group. Revised 2003 consensus on diagnostic criteria and long-term health risks related to polycystic ovary syndrome. Fertil Steril. 2004;81:19–25.
4. Ding T, Hardiman PJ, Petersen I, et al. The prevalence of polycystic ovary syndrome in reproductive-aged women of different ethnicity: a systematic review and meta-analysis. Oncotarget. 2017;8:96351–8.
5. Bridges NA, Cooke A, Healy MJ, et al. Standards for ovarian volume in childhood and puberty. Fertil Steril. 1993;60:456460.
6. Fauser BC, Tarlatzis BC, Rebar RW, et al. Consensus on women's health aspects of polycystic ovary syndrome (PCOS): the Amsterdam ESHRE/ASRM-Sponsored 3rd PCOS Consensus Workshop Group. Fertil Steril. 2012;97:28–38.
7. Naz MSG, Tehrani FR, Majd HA, et al. The prevalence of polycystic ovary syndrome in adolescents: A systematic review and meta-analysis. Int J Reprod Biomed (Yazd). 2019;17:533–42.
8. Legro RS, Arslanian SA, Ehrmann DA, et al.; Endocrine Society. Diagnosis and treatment of polycystic ovary syndrome: an Endocrine Society clinical practice guideline. J Clin Endocrinol Metab. 2013;98:4565–92.
9. DR J B Sharma, Textbook of Gynecology; first edition;2018, Chapter -6 Polycystic ovarian or syndrome (PCOS) or diseases (PCOS) and hirsutism .106-118
10. Brazozowska MM, Ostapowicz g, weltman, MD. An association between nonalcoholic Fatty liver disease and polycystic ovarian syndrome J Gastroenterol Hepatol 2009;24:243-7.
11. Ajit Virkud; Modern Gynecology 3rd Edition; chapter 27 Polycystic ovary syndrome 313-322.
12. Ehrmann DA, Barnes RB, Rosenfield RL, Cavaghan MK, Imperial J. Prevalence of impaired glucose tolerance and diabetes in women with polycystic ovary syndrome. Diabetes Care. 1999;22.141-6.
13. Miriam E. Silfen Michelle R. Denburg, Alexandra M. Manibo, Rogerio A. Lobo, Richard Jaffe, Michel Ferin, Lenore S. Lvenie, Sharon E. Oberfield Early Endocrine, Metabolic, and sonographic Characteristics of Polycystic Ovary Syndrome (PCOS): Comparison between Nonobese and Obese Adolescents The journal of Clinical Endocrinology & Metabolism, Volume 88, Issue 10, 1 October 2003, Pages 4682-4688, https://doi.org/10.1210/jc.2003-030617 Published: 01 October 2003.
14. ABM Kamrul-Hassan, Fatema Tuz Zahura Aalpona, Shahjada Selim Clinical, Metabolic and Hormonal Profiles Of Bangladeshi Adolescents with Polycystic Ovary Syndrome April 6th2021;17(1):54-8 DOI: https://doi.org/10.17925/EE.2021.1.1.54
15. Akshaya S et al, Int J Reprod Contracept Obstet Gynecol. 2016 Aug;5(8):2530-2533.
16. https://doi.org/10.1016/j.tjog.2019.05.003Get
17. Brooke Rossi et.al. J Clin Prevalence of metabolic syndrome and related characteristics in obese adolescents with and without polycystic ovary syndrome Endocrinol Metab. 2008 Dec.
18. Andrea D. Coviello, Richard S. Legro, and Andrea Dunaif Adolescent Girls with Polycystic Ovary Syndrome Have an Increased Risk of the Metabolic Syndrome Associated with Increasing Androgen Levels Independent Of Obesity and Insulin Resistance Department of Medicine (A.D.C., A.D.), Feinberg School of Medicine, Northwestern University, Chicago, Illinois 60611; and Department of Obstetrics and Gynecology (R.S.L.), Pennsylvania State University Colege of Medicine, Hershey, Pennsylvania 17033. 0021-972X/06$15.00/0 The Journal of Clinical Endocrinology & Metabolism91(2):492-497 Printed in the U.S.A. Copyright © 01 Feb 2006 by The Endocrine Society DOI: 10.1210/Jc.2005-1666.
19. N. Semtaninal, A.Seibokaite l, R. Valickas 2, R. Verkauskiene Polycystic ovary syndrome in overweight and obese adolescent girls and its association with insulin resistance and metabolic syndrome Hospital of Lithuanian University of Health Sciences grant Nr. MIP-039/2013.ea0035p813eposter pdf.
20. Madani T, Hosseini R, Ramezanali F, Khalili G, Jahangiri N, Ahmadi J, Rastegar F and Zolfaghari Z: Metabolic syndrome in infertile women with the polycystic ovarian syndrome. Arch Endocrinol Metab. 60:199-204. 2016. View Article: Google Scholar: PubMed/NCBI.
21. Lili, Qiong Feng, Ming Ye, Yaojuan He Metabolic effect of obesity on polycystic ovary syndrome in adolescents: a meta-analysis Journal of obstetric and gynaecology Published online: 28 Jun 2017Pages 1036-1047.