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"Polycystic Ovary Syndrome in Adolescents: An insight review"

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ABSTRACT:
PCOS, affecting 6-15% of reproductive-age women, is a hormonal disorder with diverse symptoms such as hirsutism, irregular menstrual cycles, and obesity. Diagnostic criteria like NIH, Rotterdam, andAndrogen Excess-PCOS Society consider factors such as hyperandrogenism,chronicanovulation,andpolycystic ovarymorphology.Hirsutism,acne,and seborrhea are common, but alopecia is rare in adolescents. PCOS, found in both normal-weight and overweight women, worsens with obesity, leading to metabolic and cardiovascular complications. Complex pathophysiology involves hormonalimbalances and insulin resistance. Diagnosing PCOS in adolescents is challenging, with limited imaging techniques. Global prevalence ranges from 2.2% to 26%, with higher rates in Indian women. PCOS is linked to diabetes, cardiovascular risks, and gynecological cancers. Early detection and management are crucial to mitigate long-term health risks
Keywords: PCOS, adolescents, hormonal disorder, irregular periods, androgen levels, polycystic ovaries, puberty, diagnosis, lifestyle modifications, diet, exercise, management, healthcare, complications, research, treatment strategies.

I. INTRODUCTION

Polycysticovarysyndrome(PCOS),also knownasStein-LeventhalSyndrome itisa disorderthatstemsfromhormonalimbalancecombined withgeneticandenvironmentalfactors.[1] It affects estimated 6-15 % of women of reproductive age and accounts for 72-84 % of adult hyperandrogenism.[2-4]it is a prevalent endocrine and metabolic disorder affecting 6–20% of Women of childbearing age.[5,6] it is one of the most frequently mentioned causes of infertility accountingforUpto56%ofcases.[7,8]AlthoughtheContributionofgenesisestimatedtobe72%, the genetic loci that have so far been identifiedAs determining the occurrence of this syndrome account for only about 10%.[9,10] It presents with a variety of symptoms such as mild hirsutism, hyperandrogenism, oligomenorrheaordysfunctionaluterinebleedingand obesity.[11,12] In the youngest women, it is Manifested by puberty and menstrual disorders, as well as cosmetic attributes associated With hyperandrogenism. At a later age, problems with ovulation and infertility dominate, followed by metabolic disorders. PCOS is linked to an increased risk of diabetes, Dyslipidemia ,cardiovascular disease, and fatty liver, as well as a higher risk of cancer, Autoimmune diseases, and mental disorders[13,14,15]Three worldwide conferences have developed Somewhat distinctive but covering demonstrative criteria For adult women: the National Institutes of Health (NIH) Conference criteria (1990), the Rotterdam consensus Criteria (2003) (Rotterdam ESHRE/ASRM-Sponsored PCOS ConsensusWorkshop Group 2004),and the Androgen Excess-PCOS Society consensus criteria (2006).[16,17,18]The NIH criteria included hyperandrogenism, Chronic anovulation, and avoidance of other causes of these indications.[1s6]TheRotterdamcriteriaaretheBroadestandincorporatethehighlightsoftheother definitions. They permit PCOS to be analyzed with a combination of Chronic anovulation and polycystic ovary morphology (PCOM) without hyperandrogenism. [19-23]

1.1 ETIOLOGY:

Forthemostpart,PCOSappearstobeacongenitalconditionthatisfirstdiagnosedduring adolescence [24,25] Accumulatingevidenceindicates thatPCOS arises asacomplextrait influenced by hereditary and non-hereditary factors.[26] Familial clustering of cases suggested a genetic basis forthedisease [27,28]

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Manygenesaresaidtobedirectlyorindirectlypromotet heprogressionofthe disease. But not now a penetrant gene was identified.[29]Themostimportantgenestoconsiderwit hintheetiologyofPCOSincludeCYP11A1,CYP17A1 , andCYP19A1. PCOSpatientshaveelevatedserumLHlevels,whichsti mulatesteroidogenesisin androgen-producingthecalcells.Steroidogenesisistheprocessby whichcholesterolismadecells of steroidogenic-specific organs are converted into bioactive compounds. The process is steroidogenicenzymesincludingsteroidreductasessu chashydroxysteroiddehydrogenase(HSD) andcytochrome P450 (CYP) family enzymes. In the ovaries,cholesterol is converted to pregnenolonebyCYP11A1underinfluenceofthelutei nizingHormone(LH).pregnenolone,onhe other hand, is first hydroxylatedto 17-hydroxypregnenolone before being converted to dehydroepiandrosterone (DHEA) by CYP 17A1[30]. Ahigh-calorie diet and a sedentary lifestyle can be possible causes of worsening PCOS.Ahigh- sugar diet can contribute to PCOS by altering gut microflora, causing chronic inflammation, increasinginsulinresistance,andincreasingandrogen production.obesityandweightgainworsen the symptoms of this syndrome. Compared with high glycemic index (HGI) diets, low GI (LGI) dietsreducedfastinginsulin,total andLDLcholesterol,TGvalues,waist circumferenceand total testosterone without changing fasting glucose, HDL cholesterol, body weight orfree androgen index in PCOS. Patients. [31,32]. Several studies have shown that environmental pollutants such as heavy metals, insecticide, and endocrinedisruptingchemicals(EDCs)significantlya ffecthumanhealthandreproductionIndeed, thereis increasingevidencethatenvironmentalpollutants playarole todevelopPCOS.Takeuchi andKandarakietal.foundthatserumBPAconcentratio nsinhyperandrogenicwomenwithPCOS were higher than in non-hyperandrogenic womenwith PCOS and healthy controls [33,34]. A separatestudy found thatitis increasing blood BPAlevels werepositively associated with serum testosterone levels in PCOS women compared to healthy women. Explains the relationships between different environments pollutants and PCOS, Vagi. conducted a case-control study that showed higher serum levels of perfluorooctanoate and perfluorooctanesulfonate in women with PCOS.[35]

1.2 EPIDEMIOLOGY:

ClearlythepredominanceofPCOSwilldepen dtoadegreeonthecriteriautilizedto define This clutter. The predominance of PCOS has been decided in different populations, primarilyofWhiteorCaucasianand,inoneponder,ofD arkraces.Inathinkabout of277womenlookingforapre-employmentphysicalinacollegewithinthesoutheaster nUS, we initially detailed an in general predominance of PCOS analyzed by the NIH 1990 criteria of 4.0%,with no noteworthy distinction between Whites and Blacks[82]consequent and more seriously think about of 400 unselected continuous ladies matured 18–45 a long time in the same setting (223 Dark, 166 White, and 11 of other races), the predominance of PCOS was watched to be 6.6%, and still not essentially distinctive between Blacks and Whites(8.0 and 4.8%, separately)[36]. PredominanceestimatesforPCOS,ascharacterized bytheNIH/NICHD criteria,demonstrate that PCOS may be a common endocrinopathy affecting 4%–8% of ladies of regenerative age. Recently,afewgroupshaveillustratedthatthepredomi nanceofPCOSshiftsdependingon the symptomatic criteria utilized (see Table 1)

Table:1 Prevalenceofpolycysticovarysyndrome(PCOS)usingdifferentdiagnosticcriteria [37-40]

Source	Population	NIH/NICHD criteria	ESHRE/ASRM (Rotterdam) criteria	Androgen excess and PCOS society criteria
March et al	728 Australian women	8.7%	17.8%	12.0%
Mehrabian et al	820 Iranian women	7%	15.2%	7.92%
Tehrani et al	929 Iranian women	7.1%	14.6%	11.7%
Yildiz et al	392 Turkish women	6.1%	19.9%	15.3%

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II. PATHOGENESIS:

The Pathogenesis of PCOSincludes essential theca cell defects [41]as well as neuroendocrine dysfunction of the hypothalamic-pituitary-ovarian axis leading to hyperandrogenemia [42]. The pathophysiology of these conditions influenced by alterations in steroidogenesis, ovarian folliculogenesis, neuroendocrine function, metabolism, insulin production, insulin sensitivity, adipose cell activity, inflammatory factors, and sympathectomies function [43]. Androgen excess, observed in approximately 60–80% of patients with PCOS, is a key feature of the disorder. Hirsutism and hyperandrogenism are manifestations of the excessive androgen production. Indeed, hyperandrogenism, commonly demonstrated by elevated free(unbound) testosterone in circulation, is the most common abnormality observed in the syndrome and plays a major role in perpetuating the aberrant hormone contributors to the pathophysiology of PCOS. Excessive ovarian androgen production is present in the majority of cases, but excessive adrenal androgen productioncanoccuramongsome.Theelevatedandrogenconcentrationssuppresssex hormone bindingglobulin(SHBG)concentrationscontributingtohigherfreetestosterone concentrations [44]

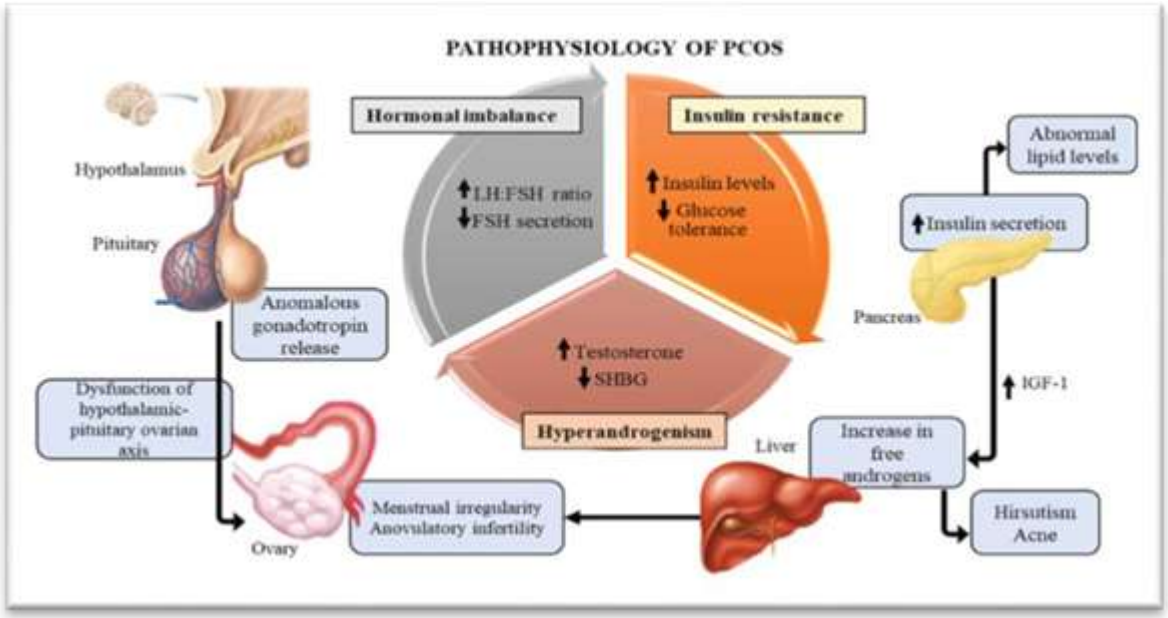

Fig:1PathogenesisofPCOS

2.1 InsulinresistanceinthepathogenesisofPCOS:

The role of insulin resistance in the pathogenesis of PCOS is supported by observations thatimprovinginsulinsensitivityinthiscondition(throughweightlossordrugtherapy)improves reproductive, hyperandrogenic and metabolic features. [45] The Following interaction with its receptor,intracellulareffects ofinsulinfollow twomain pathways via:(i)phosphatidylinositol3- kinase (PI3-kinase) and (ii) mitogen-activated protein kinase (MAP kinase). Each of these pathways mediates disparate cellular effects of insulin stimulation. The PI3-kinase pathway mediates metaboliceffects (including glucose disposalinto skeletal muscle) and theMAPkinase pathwaymediatescellgrowth andsteroidogeniceffects.[46]

InPCOS,itappearsthat onlythePI3- kinase pathway is dysfunctional, with the MAP kinase pathway functioning normally. [47] The result is divergent cellular responses to insulin, with resistance to its metabolic effects and concurrent enhancement of steroidogenesis manifesting as metabolic dysfunction,

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hyperandrogenemia and reproductive dysfunction.[48].

TheintactMAPkinasereceptorpathwayisimporta nt inthe mechanismbywhich insulinresistance influencesdevelopmentofhyperandrogenemiain PCOS.Inacomparisonbetweenurinarysteroid profilesinwomenwithPCOS(n=178)andBMI-matchedcontrolwomen(n=100),ourowngroup demonstrated enhanced 5α reductase activity associated with PCOS. Resulting enhanced conversionoftestosteronetothemorepotentandro gen,5α-dihydrotestosteronelikely contributes to the hyperandrogenism of PCOS.

A further effect of enhanced 5α reductase activity is the breakdown of cortisol with reduced negative feedback at the pituitary. Consequently, the hypothalamo–pituitary adrenal axis becomes overactive in PCOS, thereby further stimulating adrenal androgen production [49]

2.2 GenesinvolvedinpathogenesisofPCOS:

GeneticfactorsinvolvedininheritancePCOS (Table2).However,thegreatheterogeneity ofdescribed genomes does Unequivocal determination ofPCOS genotype is not possible [50] althoughthecontributionofgenesisestimatedtobe72 %ofthegeneticlociidentifiedsofar,the detection rate of this syndrome is only about 10%[51,52].

Table 2: The main genes involved in pathophysiology of PCOS and their metabolic and fertility impact.[50]

Gens Involves in the Pathophysiology of PCOS				
Steroidogenesis	**Insulin Secretion and Action**	**Effect of Steroid Hormones**	**Gonadotropin Regulation**	**Others**
CYP21, CYP11a, CYP19, CYP17	*IRS* group, *INSR, CAPN10, FTO*	AR, SHBG, *DENND1A*	FSHR, *LHCGR*, AMH, *HOXA* group, BMP	PAI-1
Hyperandrogenism	Diabetes, obesity Oxidative stress	Hyperandrogenism	Infertility	Infertility
Ovulation and Implantation Disorders				
Infertility/Cycle Disorders				

CYP group: cytochrome family p450; *INS*: insulin gene; *IRSR*: insuline receptor substrate; *CAPN10*: caplain—10; *FTO*: fat mass obesity; AR: androgen receptor; SHBG: sex hormone binding globulin; *DENND1A*: connecdenn-1; FSHR: follicle-stimulating hormone receptor; *LHCGR*: lutein hormone gen receptor; AMH: anti-Mullerian hormone; *HOXA*: gen responsible for succsesful implantation; BMP: bone morphogenetic protein; PAI-1: plasminogen activator inhibitor 1.

ThemajorityStudiesshowthatthemechanismsthattrig gerPCOSareduetoepigeneticchanges, Includingglycationofcertaingeneendproducts.Those aftertranslationthechangesdependon theenvironmentofthemotherorganism,whichismorea ndmorenumerousCivilizationalfactors thatcauseobesity,as well as hormonaland immunologicalfactorsDisorders associatedwiththe pathogenesis of PCOS in the developing fetus (Figure 3) [53].

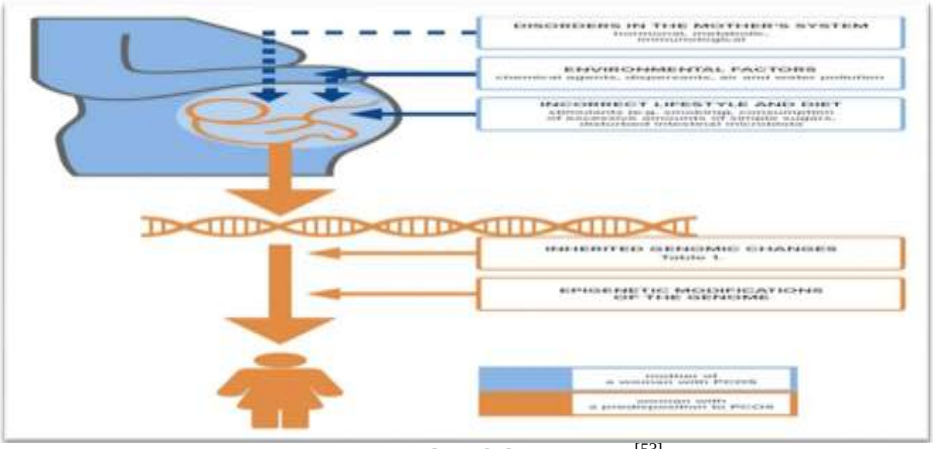

Fig:2GeneticvulnerabilitytoPCOS.[53]

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Additionally, there is evidence of a genetically male counterpart to PCOS that rules it outthe starting point of this syndrome is the ovaries [54]. Men turned out to be tallwith genetic risk, the likelihood of developing the "male equivalent" of PCOSincreasedon the development of obesity, diabetes, cardiovascular diseases and male pattern baldness [55].It is possible that the reproductive problems associated with PCOS may be due to biological causesMechanisms common tomen and women [56].

III. DIAGNOSIS:

Thefirstdiagnosticcriteriafor PCOSinadultwomenwereestablishedbyaconsensus meeting at the National Institutes of Health (NIH) in 1990 [57] Including NIH criteria clinicalorbiochemicalevidenceofhyperandrogenism andovulationdisorders[58].PCOM was not part of their criteria because it was already known at the time that polycystic ovarian morphologyis observed in 20–30% of healthy women [59]. the NIH criteria were the standard for diagnosing PCOSfor more than a decade until 2012when the NIH recommended theuseoftheRotterdamcriteria.TheRotterdamcriteria wereanotherstepforwardinthediagnosis ofPCOSbyaddingPCOMasadiagnosticcriterion. PCOSisdefinedaccording totheRotterdam Agreement twoof the following three criteria: oligo/anovulation, hyperandrogenism, and polycystic ovaries [60]

Table3: DifferencesincriteriaforPCOSdiagnosisinadolescentpatients. [61,62]

Criteria	Definition	
Menstrual Irregularity	Irregular menses/oligomenorrhea 2 years post-menarche. Menstrual cycles > 90 days 1-year post-menarche. Primary amenorrhea in girls with completed puberty.	Irregular menstrual cycles < 1-year post-menarche represents a normal pubertal transition. >90 days for any one cycle > 1-year post-menarche. Cycles< 21 or >45 days >1 to <3 years post-menarche. Cycles < 21 or >35 days 3 years post-menarche. Primary amenorrhea by age 15 or >3 years post-thelarche.
Hyperandrogenism	Evidence of hyperandrogenism: a. Biochemical—no clear testosterone concentration cut-offs; confirmation of biochemical hyperandrogenism in symptomatic adolescents. b. Clinical—hirsutism and/or moderate or severe inflammatory acne, especially if unresponsive to topical therapy.	Evidence of hyperandrogenism: a. biochemical—no clear testosterone concentration cut-offs; calculated free testosterone, free androgen index, or bioavailable testosterone evaluation with high-quality assays. b. clinical—hirsutism assessed with standardized visual scales e.g., the Ferriman–Gallwey scale and/or moderate or severe comedonal acne (i.e., 10 or more facial lesions), or moderate to severe inflammatory acne.
Polycystic Ovary on Ultrasound	The presence of PCOM in an adolescent who does not have hyperandrogenism/oligo-anovulation does not indicate a diagnosis of PCOS.	Pelvic ultrasound should not be used for the diagnosis of PCOS in those with a gynecological age of <8 years.

3.1 PolycysticOvaryonUltrasound:PCOM

The presence of enlarged ovaries with increased stroma and several small peripheral cystsknown as PCOM (Polycystic ovary morphology). PCOM is associated with hyperandrogenism,butnotalwaysincludedinthediagn osticpartofPCOS.PCOMisan inconsistentfinding inhealthygirls[63]andadults,butthepersistenceofPC OMisgreater time is observed in hyper androgenic adolescents [64]in addition, the defining criteria for an ultrasound examination modification of thePCOS model is ongoing [65]. The anatomical appearance of the ovary changes with age[66]. Ovarian volume increases during pubertyandreachesadultnumbersintheyearsafterthe onsetofmenstruation.Itremainsstableand declines in young adulthoodafter the middle of the fourth decade of life.[67] Follicles size also changeswithageandmaximumamountduringpuberty smallfolliclesareobservedandasayoung adult, and thenumber of follicles decreases significantly read by age.[68]

AnultrasounddiagnosisofPCOMismadesta ndardizedforadultsusingthetransvaginalroute.In thecaseofyoungpeople,themajorityoftheexamsaresti llsuccessfulTransabdominalroutewith a high physiological follicle number can make follicle count an unreliable criterion to diagnose PCOM.ImportanceofuseappropriatediagnosticcriteriaforPCOMinyouthexistisemphasized becausetheapplication ofadultcriteriacan lead to afalsely elevated prevalenceof PCOM (30- 40%

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region) [69,70]

Table4: SuggestedcriteriaforthediagnosisofPCOSinadolescence[71]

Required	Optional[a]	Not recommended[b]	Comments
1. Irregular menses/ oligomenorrhea 2. Evidence of hyperandrogenism: a. Biochemical b. Clinical (e.g., progressive hirsutism)	1. PCOM 2. Severe cystic acne	1. Obesity 2. Insulin resistance 3. Hyperinsulinemia 4. Biomarkers (e.g., AMH, T/DHT ratio) 5. Acanthosis nigricans	1. Must generally be 2 years post-menarche 2. Must rule out other disorders of hyperandrogenism (e.g., NC-CAH, Cushing syndrome)

PCOS; polycystic ovary syndrome; PCOM, polycystic ovarian morphology; AMH, anti-Müllerian hormone; T/DHT, testosterone to dihydrotestosterone; NC-CAH, non-classical congenital adrenal hyperplasia. [a] These criteria are often used in concert with the required criteria, but should not be used independently as diagnostic features. [b] These criteria have been associated with PCOS but are not diagnostic.

3.2 Menstrualirregularities:

One of the most important problems faced by healthcare providers in particular anovulatory dysfunction is diagnosed in primary care based on the irregular cycles of the adolescent. This has long been understoodThat theperiod between periods is accompanied by maturationHypothalamic-pituitary-ovarian (HPO) axis and period establishing aregular ovulation cycle. For this therefore, the average cycle for an adult can last 28 days (range 24-35). some time starts right after menstruation, with a significant difference in cycle length and a high ratioAnovulatory cycles, especially in the first year after menarche [72]Guidelines recommend considering PCOS based on cyclonic irregularityin the third year after menstruation.Atthispoint,95%ofcyclesfail21-45daysandperiodslast2-7days.Menstruation irregularities at this age have been shown to be highly correlated suffers from oligomenorrhea at the age of 18 years [73]Children can be especially overweight Tendency to premature pubic arch andpubicarchandinturnmaybemorepronetoprematurmenstruationandPCOS;Theyshould beconsideredahigh-riskpopulationandreceivesspecialattentionthecourse ofthepostmenstrual cycle[74,75,76]. BecausePCOSisadiagnosisofexclusion,thereareothercausesofirregularcycles (suchashypothyroidismorhyperprolactinemia)mustbeconsideredandruledoutbefore diagnosing PCOS.Astudy of menstrual disorders after two yearsirregular cycles seem to have become the norm worldwide [77,78,79].

IV. TREATMENT OF ADOLESCENT PCOS:

Adolescents with pre-existing PCOS symptoms [80]often require treatment to manage their symptoms. For adolescents with a clear diagnosis of PCOS, treatment should include education about PCOS and lifestyle interventions. These interventions can be tailored to address themostcommoncomplaintsandsymptoms. Interventionsmayinclude: Metformin; Combinedoral contraceptivepills(COCP);Spironolactone;localHirsutismandAcneTreatment.Managementof Comorbid Conditions; regular Follow-Up; Planning for Transition toAdult Care Providers [81]

4.1 Oralcontraceptives(OCPs):

OCPsareatypeofmedicationthatcanbeusedasafirstlineoftreatmentforwomenwho areunabletoovulateduetomenstrualirregularities.Theyareavailableintwoforms:progesterone only pills and combined pills that contain both estrogen (estradiol dose up to 50µg) and progesterone (norethisterone, desogestrel) [82]. OCPs reduce the circulating androgen levels by increasing theSHBG (seeFig:6). [83] WomenwithPolycysticOvarySyndrome(PCOS)are more likelytodevelopcancers,however,OCPsmayreducetheriskofovariancancer [84].OCPsarenot knowntoaffectinsulinresistance,however,theymaydemonstratevariabilityinlipidprofilesthat may lead to metabolicdisorders [85].Therefore, OCPs should beused based on therisk gradeand should be discontinued immediately if any discrepancies are observed.

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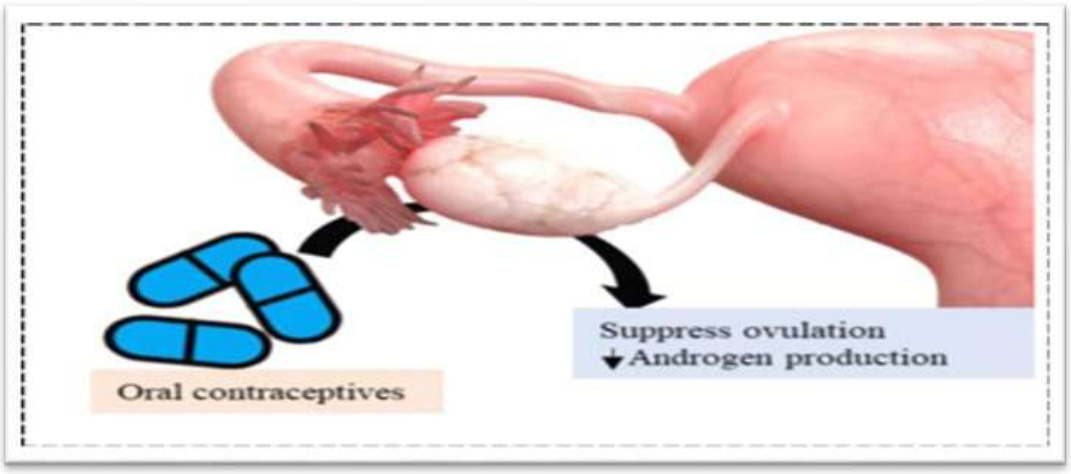

Fig:3.MechanismofOCPs.[86]

4.2 Antiandrogens:

Theuseofeffectiveformsofcontraception,including antiandrogens,maybeconsideredas atreatmentforhirsutismandandrogen-relatedalopecia.However,thepotentialteratogeniceffects ofantiandrogensonmalefetuses,aswellastheirinterferencewithexternalgenitaldevelopment, necessitate the use of effective contraception. This recommendation, based on a conditional evidence-based review with aGRADEof very low,was primarily madein adultwomen and was based on the use of flutamide or finasteride, spironolactone's, alone or in conjunction with a diet intervention.[87] This group includes: Spironolactone ,Flutamide ,Cyproterone acetate Which reduces androgen secretion byinhibitionbyandrogenreceptorantagonistsandispreferredasthefirst-linedrugin hirsutismtreatment.[88] Spironolactoneisananabolicagentthatproducesanantiandrogeniceffect whentakenathighdoses.Whentakenalone,itleadstomorefrequentperiods,soitisusuallyused incombinationwithOCPstocreatesynergyandovercometheproblem.[89]Flutamideisanorally- tolerated anabolic agent used to treat prostate cancer and has the same efficacy as spironolactone in the treatment of hirsutism.[90,91,92] Flutamide is commonly used in conjunction with metformin due to its potential to cause hepatotoxicity when taken on its own. [93] Additionally, Cyproterone Acetate is a powerful antiandrogen with a progestogenic effect. When used in conjunction with Ethinylestradiol, it can beusedasatreatmentforacneandhirsutism.]Finasteride isaninhibitorof5-S-reductase,which has been shown to reduce hirsutism cores.However, its use in women is restricted due to its teratogenic properties. It is recommended for postmenopausal women and those who are unable to ovulate.[94]

4.3 Insulinsensitizers:

This class of medicinal products is typically utilized to address metabolic co-marks associated with Polycystic Ovary Syndrome (PCOS) by reducing insulin resistance and restoring insulin levels to a normal range. By reducing the Internal Repute Ratio (IR), the associated androgen levels will decrease, resulting in an improvement in menstrual cycles.[90]

4.4 Metformin:

Metformin has been shown to increase insulin sensitivity in liver by reducing the activity of gluconeogenic enzymes such as Pele glycoprotein catheters, Biases, and glucose-6-phosphatases, as well as inhibiting the liver's uptake and conversion of lac-tate to alanine and glucose to alanine. Metformin also increases peripheral glucose uptake, reduces fatty acid oxidation,anddecreasesglucoseabsorptionfrom the intestine.

At the cellular level,theeffectsof metformin on AMPK are mediated by phosphorylation, which modulates the activity of both catalytic a1s and a2s ofAMPK. This results in improved muscle glucose uptake when insulin is present. In mice skeletal muscle cell lines, the effect of metformin is mediated by threonine a2-residue a2-phosphorylation, which is maintained upon discontinuation of the medication.[95] The use of metformin has been shown to have a modest beneficial effect on the lipid profile of patients with PCOS.[96] Additionally, it does not appear to have

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a teratogenic effect when used during pregnancy. Furthermore, it has been shown to reduce inflammation and complications associated with pregnancy.[97]
Furthermore,whenusedinconjunctionwithClomipheneCitrate,ithasbeenfound toincreasetheovulationrateandpregnancyrateofInfertilepatientswithPCOS [98].Furthermore, when combined with antiandrogens such as flutamide, it has been observed to have a synergetic effect in women with PCOS who are obese, although this effect is not observed to be safe for laboratory animals.[99] Furthermore, a beneficial effect was observed to improve hyperan- dragonish of PCOS women with the use of metformin and lifestyle modification [100]

V. CONCLUSION:

In conclusion, addressing polycystic ovarian syndrome (PCOS) in adolescents requires a multidisciplinary approach. Early recognition, coupled with lifestyle interventions, formsthecornerstoneof management. Regularmonitoringand individualizedcarearecrucialfor mitigatinglong-termcomplications.Furtherresearchisneededtoenhanceourunderstandingof PCOSinadolescentsandrefinetreatmentstrategies,ensuringcomprehensiveandeffectivesupport for this population.Oral contraceptiveslike norethisterone, desogestrel, are commonly prescribed for PCOS to regulate menstrual cycles, reduce androgen levels, and manage symptoms. While effective for many, individual responses vary, and potential side effects should be discussed with a healthcare provider for personalized guidance and monitoring.Antiandrogens, likespironolactone, can be effective in managing PCOS symptoms likeacne and hirsutism by blocking androgen effects. However, individual responses and potential side effects vary, necessitating careful consideration and monitoring under the guidance of a healthcare provider.Insulinsensitizers,likemetformin,areusedin PCOStoimproveinsulinresistance.Theycan help regulate menstrual cycles and manage metabolic aspects. While generally effective, individual responses vary,andconsultingwithahealthcareprovideris crucial toassess suitability,potential side effects, and monitoring needs.

Compliance with ethical standards

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Disclosure of Conflict of Interest
No conflict of interest to be declared.

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