PCOS: Diagnosis and Management in Adolescents: Transition of Care

SAHM 2015 WORKSHOP: Paula Hillard, MD, Neville H Golden, MD, and Sophia Yen, MD
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Case 1, Erin.

14 yo F presents to the ER with a 16 day H/O painless vaginal bleeding. Menarche was at age 13yrs. Menses have been irregular. She denies sexual activity. No h/o trauma

PE: Ht. 63ins (50th %), Wt. 115lbs (65th %), BMI 20.3 (64th %)
BP 90/50, P 120/m
Pale. No evidence acne or hirsutism
Skin – no petechiae or ecchymoses
Breasts- Tanner 3, Genital- Tanner 4
Pelvic: No clitoromegally
   Active bleeding noted
   No foreign body or lacerations
   Speculum Deferred
WORKSHOP: PCOS and TRANSITION OF CARE

- CASE: Does this patient have PCOS?
- INTRODUCTION: PCOS in TEENS
  - P Hillard
- CASE:
- Menstruation: What’s normal in teens
  - N Golden
- Transition of Care: Principles of Transition
  - S Yen
- Resources

OBJECTIVES

At the conclusion of this program, the attendee will be able to:
- Recognize clinical signs and symptoms that would suggest the diagnosis of PCOS in an adolescent
- State the arguments for and against making a diagnosis of PCOS in an adolescent
- Be prepared to transition care of an adolescent with PCOS to a clinician who cares for adult women
Pediatrician/Primary Care—"Normal adolescence" (common signs and symptoms of acne, irregular periods, obesity, pre-diabetes, hyperlipidemia, metabolic syndrome)
Dermatologist—Acne, Alopecia, Hirsutism, (Acanthosis Nigricans)
Endocrinologist—Diabetes, metabolic syndrome, obesity
Gynecologist—Irregular periods, heavy bleeding, amenorrhea (primary and secondary), (Infertility)

**CONSIDER DIAGNOSIS OF PCOS**

- What would make you think PCOS?
  - Obesity
  - Hirsutism
- What should make you think PCOS?
  - Irregular periods
  - Amenorrhea—Secondary or Primary
  - Frequent bleeding
- FH—PCOS, irregular periods as teen, obesity/hirsutism, anovulatory infertility; (early male balding)
- Former SGA
- History of premature adrenarche

**WHAT IS PCOS?**

- Most common endocrinopathy in women
  - 5-10% of adult women
  - *SM women
- Most symptoms begin during adolescence
- Overlooked, under-recognized clinically
- Heterogeneous disorder—a SYNDROME
- Genetic component with epigenetic and environmental factors
- Phenotypic subtypes
  - Obesity
  - Normal weight or lean
- Consensus definitions
Pathogenesis of PCOS

Genetic predisposition to hyperandrogenism
Genetic predisposition to hyperinsulinism

Environmental Factors

PCOS

What is PCOS? Clinical Signs & Symptoms

• COMMON Presenting Signs & Symptoms in teens
  • Irregular periods (ovulatory dysfunction)
  • Weight gain
  • Ovarian cysts (polycystic ovarian morphology; functional cysts)
  • Associated Signs & Symptoms (not typically presenting concern in teens)
  • Excess hair growth on face/body (Hirsutism)
  • Thinning scalp hair
  • Mental health problems (depression, body image, poor self esteem)
  • Fertility impairment
  • Insulin resistance
  • Associated risks of: DM, hypertension, hyperlipidemia
  • Potential opportunity to mitigate risks with management

What’s in a NAME?

• Current terminology:
  • Difficult to say
  • “Polycystic Ovary Syndrome”
  • “P-C-O-S”
  • “Pee Cos”
  • Inappropriate focus on ovaries
  • Patients equate this with ovarian cysts that cause pain, grow, and
    that may require surgery
  • National/International movement to change the name
    • “Hormone Imbalance”
### PCOS—Diagnostic Criteria

<table>
<thead>
<tr>
<th>Diagnostic Criteria</th>
<th>Strengths</th>
<th>Limitations</th>
</tr>
</thead>
<tbody>
<tr>
<td>Androgen Excess</td>
<td>Included as a component in all major classifications, a major clinical concern for patients, animal models emulating androgen excess resemble but do not fully mimic human disease</td>
<td>Measurement is performed only in blood, concentrations differ during time of day, concentrations differ with age, normative data are not clearly defined, results are not standardized across laboratories, clinical hyperandrogenism is difficult to quantify and may vary by ethnic group, tissue sensitivity is not assessed.</td>
</tr>
<tr>
<td>Ovulatory Dysfunction</td>
<td>Historically associated with syndrome, may be associated with hyperstimuli to ovarian stimulation</td>
<td>Normal ovulation is incompletely understood, normal ovulation varies over a woman's lifetime, ovulatory dysfunction is difficult to measure objectively</td>
</tr>
<tr>
<td>Polycystic Ovarian Morphology</td>
<td>Historically associated with syndrome</td>
<td>Technique dependent, difficult to obtain standardized measurement, lack of normative standards across the menstrual cycle and lifespan (notably in adolescence) as ovarian morphology varies with age, technology required to accurately image not universally available, prognosis poorly characterized in certain circumstances (e.g., adolescence)</td>
</tr>
</tbody>
</table>

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**NIH 1990**
- Chronic anovulation
- Clinical and/or biochemical signs of hyperandrogenism (with exclusion of other etiologies, e.g., congenital adrenal hyperplasia) (Disorder criteria needed)

**Rotterdam 2003**
- Oligo- or anovulation
- Clinical and/or biochemical signs of hyperandrogenism
- Polycystic ovaries
- Hirsutism
- (Two of these criteria needed)

**AE-PCOS Society 2006**
- Clinical and/or biochemical signs of hyperandrogenism
- Ovarian dysfunction (oligopause and/or polycystic ovarian morphology)
- (Three criteria required)
Case 1, Erin.

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Speculum Deferred

Erin, Labs

Hb/HTC 4.9 g/dl / HTC 15.0%
Platelets 200,000/cu mm
PT/PTT 13.2/30.2
U Preg Negative
TFTs WNL
Von Willebrand Panel WNL

Erin, Management
Case 1, Erin.

Erin returns to see you 2 years later. She is now 16 years old. She had been treated for one year with OCPs to control her periods. After stopping the pill she had 3 periods but then her periods stopped. Her last normal menstrual period was > 6 months ago. She denies sexual activity.

PE: Ht. 64ins (50th %), Wt. 145lbs (85th %), BMI 24.9 (87th %)
BP 115/80, P 80/m
Moderate acne of the face and chest. Small amount of hair on her upper lip

Labs:
Testosterone, total: 30 ng/dl (N 40-60)
Free testosterone: 0.65 ng/dl (Normal)
DHEAS: 250 ng/mL (Normal)
17- OHP: (normal)

Controversies about the diagnosis of PCOS in adolescents

• Menstrual irregularity with or without DUB (AUB) in the first few years after menarche is usually secondary to physiological immaturity of the HPO axis, but (as in this case) may be the first presentation of PCOS

• Challenges in the diagnosis of PCOS during adolescence and concerns about over diagnosis have led some to suggest that PCOS should only be diagnosed in adolescents with > 2 years persistence of hyperandrogenism, anovulatory menstrual cycles and ovarian size > 10 cc. There is no harm in starting treatment for milder forms of PCOS at age 18 years. Carmina et al. Am J Obstet Gynecol 2010

• Waiting > 2 years to diagnose and treat PCOS in adolescents may unnecessarily delay treatment and recognition of comorbidities Rosenfeld RL, JCEM, 2013

Clinical, ultrasound and biochemical features of PCOS in adolescents

• Prospective cohort study with 244 unselected post-menarcheal community-based girls in Australia
• Mean age 15.2 years, 91% Caucasian
• Recruited from a large population-based birth cohort
• Methods
  – Menstrual diaries
  – Clinical hyperandrogenism quantified with F-G scores
  – Severity of acne quantified
  – Total testosterone and SHBG measured in early follicular phase (day 2-6) by RIA at the same time of the day. Free T calculated
  – Transabdominal ultrasound examination
  – BMI measured

Hickey M et al, Reproductive endocrinology, 2011
Clinical, ultrasound and biochemical features of PCOS in adolescents - Results

- Menstrual irregularities - 51.7%
- Hirsutism - (F-G score > 5) - 8.2%
- Acne -
  - Mild - 48%
  - Moderate-to-severe - 21%
- Biochemical hyperandrogenism
  - Top 5% calculated free T - 45.6 pmol/l
  - Top 10% free T - 34.5 pmol/l (range of values higher than adults)
- PCO morphology - 35.4% met adult sonographic diagnostic criteria for PCOS
- BMI
  - 21% overweight
  - 8% obese

Menstrual dysfunction and adolescents

- Median age of menarche 12.4 years (range 9-15)
- In the first year after menarche 85% of menstrual cycles are anovulatory, 59% in the 3rd year and 25% by the 6th year but 85% fall within the range of 21-45 days
- By the third year after menarche, 60%-80% of menstrual cycles are 21-34 days long
- It takes approximately 5-7 years for the HPO axis to mature


Clinical, ultrasound and biochemical features of PCOS in adolescents - Results

- NIH Criteria
  - 3.1 % met criteria for PCOS
- Rotterdam Criteria
  - 18.5 % met criteria for PCOS
- Androgen Excess Society Criteria
  - 5 % met criteria for PCOS
- Conclusion
  - Diagnostic features of PCOS used in adult women may be of limited use in adolescents

Hickey M et al, Reproductive endocrinology, 2011
Menstrual dysfunction and adolescents with PCOS

- 75% - 85% of adults with PCOS have menstrual dysfunction
- 75% - 100% of adolescents with PCOS present with menstrual dysfunction

Clinical characteristics of adolescents who met diagnostic criteria for PCOS

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>% of Adolescents</th>
<th>Reference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated total testosterone</td>
<td>67</td>
<td></td>
</tr>
<tr>
<td>Elevated free testosterone</td>
<td>63</td>
<td></td>
</tr>
<tr>
<td>Hirsutism</td>
<td>69</td>
<td></td>
</tr>
<tr>
<td>Clinical hyperandrogenism with PCOS based on sign or symptom</td>
<td>22</td>
<td></td>
</tr>
<tr>
<td>Acne</td>
<td>40</td>
<td>90147</td>
</tr>
<tr>
<td>Acneiform lesions</td>
<td>48</td>
<td>90147</td>
</tr>
<tr>
<td>Total cholesterol &gt; 200 mg/dL</td>
<td>15</td>
<td>1134</td>
</tr>
<tr>
<td>LDL &gt; 130 mg/dL</td>
<td>19</td>
<td>1134</td>
</tr>
<tr>
<td>HDL &lt; 40 mg/dL</td>
<td>30</td>
<td>1134</td>
</tr>
<tr>
<td>Triglyceride &gt; 150 mg/dL</td>
<td>27</td>
<td>1134</td>
</tr>
<tr>
<td>Ferriman-Gallwey score</td>
<td>20</td>
<td>12269</td>
</tr>
</tbody>
</table>

* The differences in denominators are based on the number of patients who had the test ordered or physical exam finding documented.

Clinical hyperandrogenism and teens

- Acne
  - 20% - 70% of healthy teens have acne
  - 12% - 20% of adults with PCOS have acne
- Hirsutism
  - Hirsutism develops slowly and may take many years to manifest and may not be responsive to treatment
  - Various causes include: androgen excess, obesity, PCOS
  - Distribution and number of terminal hairs influenced by ethnicity (low in Asian)
  - Ferriman-Gallwey score based on studies in adult White women and may have varying cut points in adolescents
  - Ferriman-Gallwey scores based on studies in adult White women and may have lower cut points in adolescents
  - No standardized grading system for use in adolescents
- Male pattern balding
  - Prevalence in PCOS varies but probably around 5%
  - Not commonly seen in adolescents
Ferriman-Gallwey grading system

Biochemical hyperandrogenism and teens

- Lack of normative values of androgens in teens
  - Total Testosterone: Normal upper limit 40-60 ng/dL in adults
  - PCOS: 50-150 ng/dL
  - Virilizing tumor: >200 ng/dL
- Total vs. free testosterone
  - Assessment of free testosterone more sensitive than total T
  - SHBG decreased in obese, insulin-resistant and/or hyperandrogenic individuals
- Testosterone assays
  - Commercial measurement of free T by RIA is highly inaccurate, especially at the lower T levels (<100 ng/mL)
  - Measurement of free T by equilibrium dialysis or mass spectrometry more accurate but not widely available
  - Direct assays for total T are highly variable with large intra- and interassay coefficients of variation
  - Free T usually calculated from total T and SHBG levels
- Timing of the blood test
  - In teens often done in the afternoon. Diurnal variation – should be repeated in the early morning

Metabolic effects – obesity and insulin resistance

- Obesity prevalent in adolescents
  - Between 38%-80% of women with PCOS are obese
- Obesity may itself be a common cause of adolescent ovulatory dysfunction
  - Suppresses gonadotropins
    - Increases insulin resistance
    - Increases androgen levels
- Insulin resistance and compensatory hyperinsulinemia peak in mid-puberty and wane thereafter in parallel with GH production
  - 50%-60% of patients with PCOS have insulin resistance and hyperinsulinism

Ref: Rosenfield RL. Adolescent Anovulation: Maturational Mechanisms and Implications. JCEM, 2013
Trends in standard workup of adolescent PCOS performed by pediatric subspecialists

187 adolescents aged 11-18 years diagnosed with PCOS at a tertiary Children's Hospital in Washington DC


DIAGNOSIS in ADOLESCENTS

- Difficult to apply diagnostic criteria for adolescents
  - US—Ovarian morphology best seen with TVUS
  - Irregular periods and anovulation—developmentally normal, but this doesn’t mean “anything goes”
    - 90 days of amenorrhea is abnormal at any age
    - 1 exception: interval from 1st to 2nd menses
  - Statistical norms = 21-45 days
  - Hyperandrogenism
    - Transient during puberty

PCOS—Ultrasound

- TAU5 vs TVUS

- Ultrasound definition:
  - Presence of 12 or more follicles in each ovary measuring 2-8 mm in diameter, and/or increased ovarian volume (>10 ml)
    - Volume = D1 x D2 x D3 x 0.523
  - Does not apply to women on OCs
  - Recommend TransVaginal approach, in follicular phase or d 3-5 after induced menses
  - If dominant follicle (>10 mm) or CL, repeat in next cycle
WHY Make the diagnosis?

Labeling with a Diagnosis: **PROS**
- Motivating (older teens)
  - Diet
  - Exercise
  - Managing CV morbidities
- "Mom understands even if teen doesn’t"
- a "disease" or "condition" not her fault
- Avoids treatment/management delay for acne, hirsutism, obesity
- Prevent menstrual morbidity with appropriate menstrual management
  - anemia
  - infertility?
- Positively impact QOL/self-esteem
- Potential to impact/minimize future health consequences

Labeling with a Diagnosis: **CONS**
- Features of possible PCOS may be in evolution or transient
- Polycystic ovaries
- Hyperandrogenism of puberty
- Unnecessary labeling, leading to
  - Psychological distress
  - Body image issues
  - Excessive focus on future fertility
    - Risk taking or attempting pregnancy to "prove" fertility
    - "Don’t assume you’ll have problems getting pregnant when you want to"
  - Unnecessary treatment

Associated Medical Conditions

- Obesity
- Hypertension
- Insulin resistance/DM
- Hyperlipidemia
- Fatty liver
- Obstructive sleep apnea
- Hirsutism
- Acne


Adult Consequences of PCOS

- Infertility
- Endometrial Cancer
- CV Disease
- Insulin resistance/Diabetes
- Metabolic syndrome

Reproductive Consequences of PCOS

- Adolescents: “Don’t assume you’ll have difficulties getting pregnant”
- Young Adults: “It’s possible that you may have difficulties getting pregnant, so think about a Reproductive life plan.”
- Subfertility w multiple potential contributing factors:
  - Effects on ovulatory function, oocyte quality, endometrial receptivity and fetal development related to:
    - obesity, and metabolic, inflammatory, and endocrine abnormalities
  - Effects are not universal
- Reproductive life plan needs to take PCOS into account
Goals of Treatment of PCOS

- Normalize Hormonal state and its effects
  - Unopposed estrogen and endometrial CA risk
  - Hirsutism, acne
- Improve metabolic status
  - Treat insulin resistance and its effects via Controlling Weight

Weight loss

- ~76% of women with PCOS in the US are obese
- 5% Weight loss can
  - Decrease circulating androgens
  - Improve glucose and lipid profiles
  - Decrease hirsutism
  - Lead to spontaneous resumption of menses

Weight loss by change in diet & exercise

- Plate Model
  - Nutrition videos - Bryan Lian, RD Nourish playlist
- Exercise
  - 8 minutes in the Morning by Jorge Cruise
  - Pedometer 10,000 steps/day
Lifestyle modification program n=8

- 16 sessions with pt and parent/guardian
- Rolling group admission, Support group
- Lecture series - standard nutritional lectures as well as reading food labels, reading a menu, portion sizing, and hands-on kitchen training
- Behavior support – peer counseling, electronic communications
- Structured group exercise weekly
- Goals: aim for 500kcal/d deficit & exercise 30min/day of moderate to intense activity
- Results: increased SHBG 2.2x, but not as good as OCPs

Diet study for 3 months

- N=24, 16 completed the study (67%)
- LowCarb group: ≤/= 20 g/day of carbohydrate for initial 2 weeks. For weeks 3 - 12, carbohydrate was increased to 40 g/day by adding low glycemic index foods, such as nuts, fruits, and whole grains. Minimum fluid intake of 50 oz per day, MVI, and a KCl tablet to avoid hypokalemia
- LowFat hypocaloric diet <40 g per day of fat, 5 servings of starch /day and an ad libitum intake of fat-free dairy foods, fruits, and vegetables, for 12 weeks. A serving of starch = 15 g of carbohydrate per serving, and consumption of whole grains was encouraged.
- And 30 min of aerobic exercise 3x/wk, RD monitoring every 2 weeks
- Results: total group: 6.5% weight loss from baseline

Weight loss by meds/surgery

- Orlistat BMI -4.5%, weight -3.8%
- Metformin BMI -4.5%, weight -5%
- Bariatric surgery in adults
  - Results:
    - 77% moderate or complete resolution of hirsutism
    - 62.5% reduction in free Testosterone
Lifestyle change difficult to sustain

- Adults: 40% drop out in intense lifestyle mod

In Hoeger et al’s PCOS adolescent study, n=79
- 30% dropped out,
- 40% attended < 50% of sessions
  —thus no weight change in those patients

OCPs = 1st line med therapy

- Suppress LH → increase SHBG → decr plasma androgen
- Inhibit 5-alpha-reductase

- 1st line medical therapy in adolescents is COCs
- Control of menstrual cycles
- Decrease risk of endometrial hyperplasia
- Reduced serum Testosterone
- Improved Hirsutism and Acne

- If BMI >30, androgen suppression may be decreased

OCPs mechanism of action

- Decrease T production from ovaries and adrenals
- Increase SHBG production by liver
- Decrease free testosterone as a result of increase in SHBG
- Possible inhibition of 5α-reductase
**Antandrogens in adolescent PCOS**

Symptoms of androgen excess e.g. Acne and hirsutism can be quite distressing

After 6 month OCP trial, may consider adding anti-androgens

- Flutamide (non-steroidal, androgen receptor antagonist) low dose 62.5mg/day
  - Or in combo with metformin 850mg
  - SE as monotherapy and high dose – LIVER TOXICITY
- Spironolactone 50 mg/day
  - SE: hyperkalemia

**PCOS and Insulin**

- Variability in ovarian volume
  - LH accounted for 37%
  - insulin for 18%
- Obesity → acanthosis nigricans (hyperinsulinemia) → hyperandrogenemia
- Role of insulin sensitizers may be limited to:
  - optimizing weight loss
  - tx of metabolic complications e.g. hyperlipidemia and elevated pro-inflammatory cytokines.
  - Factor V Leiden mutations

**Metformin**

- Enhances periph tissue sensitivity to insulin
- Inhibits hepatic gluconeogenesis
- Incr uptake/utilization of glu by muscle
- Reduces: plasma insulin levels, tot Testosterone, sleep disturbance scale, Epworth sleepiness scale
- SE: GI, n/v, bloating, flatulence, diarrhea, lactic acidosis in renal pt
- NOT FDA approved for PCOS
- 850mg BID or 1gm BID
Metformin vs. Metformin+OCP vs OCP 12month adult study

- N=90, 65 completed the study (72%), Denmark, DXA scan
- Metformin 1gm BID, 150mg desogestrel and 30 mg EE
- Metformin vs OCP
  - metformin = improved body composition (decreased weight, BMI, and fat arms, truncus fat, and leg fat) and decreased C-peptide vs OCP. Lower SHBG than OCP.
- Metformin+OCP vs OCP
  - metformin+OCP = improved body composition, improved FG score & decreased insulin and C-peptide vs OCP. Total T and SHBG changes comparable.
- Metformin+OCP vs metformin
  - metformin+OCP: SHBG levels increased, C-peptide decreased vs metformin, total T levels comparable. Metformin alone had improved body composition compared to combo

Metformin + OCPs + lifestyle

- N=32 obese adolescents
- Metformin + OCPs + lifestyle =
  - 4% decrease Waist Circumference,
  - 55% decrease in Tot Testosterone,
  - 46% increase in HDL

Meformin vs. OCP – NOT side by side

<table>
<thead>
<tr>
<th>Metformin</th>
<th>OCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>Increase HDL</td>
<td>Increase SHBG, decrease androgens</td>
</tr>
<tr>
<td>Decrease mean serum testosterone</td>
<td>Decrease glu tolerance</td>
</tr>
<tr>
<td>2.5 RR of menses vs placebo</td>
<td>Unfavorable changes in insulin sensitivity</td>
</tr>
<tr>
<td>Increases HDL by 7mg/dl vs. -2.33 in placebo</td>
<td>No effect on weight</td>
</tr>
</tbody>
</table>
### Metformin vs. OCP – side by side comparison

N=35 obese, adolescents aged 12-21y/o with PCOS and hyperinsulinism, RCT for 6 months

<table>
<thead>
<tr>
<th>Metformin</th>
<th>OCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>• BMI 37.3 to 36.3</td>
<td>• BMI 40.1 to 38.6</td>
</tr>
<tr>
<td>• Decrease free Test 2.1 to 1.6 pg/ml</td>
<td>• Decrease free Test 1.8 to 0.96 pg/ml</td>
</tr>
</tbody>
</table>

No sig differences in response to treatment in outcome variables.

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### Metformin vs. OCP – side by side comparison

N=43 adolescents PCOS and hyperinsulinism, RCT for 6 months

Lifestyle, OCP, Metformin, or placebo

<table>
<thead>
<tr>
<th>Metformin</th>
<th>OCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>• 25% decrease in TG</td>
<td>• Increase SHBG, reduce free androgen index</td>
</tr>
<tr>
<td>• Decrease in glu but not insulin level</td>
<td>• 14% increase in Tot Chol</td>
</tr>
<tr>
<td></td>
<td>• 40% increase in high sensitivity CRP</td>
</tr>
</tbody>
</table>

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### Metformin vs. OCP – Cochrane

<table>
<thead>
<tr>
<th>Metformin</th>
<th>OCP</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Lower fasting insulin</td>
<td>• Improved menstrual pattern</td>
</tr>
<tr>
<td>• Lower TG</td>
<td>• Decreased androgens</td>
</tr>
<tr>
<td>• 12 mo – lower BMI and weight</td>
<td>• Unfavorable changes in insulin sensitivity</td>
</tr>
<tr>
<td>• Increase HDL</td>
<td></td>
</tr>
<tr>
<td>• SE: GI</td>
<td>• No effect on weight</td>
</tr>
</tbody>
</table>

No difference in hirsutism and acne. No difference/insufficient data on long term for DM development, CV dz or endometrial CA (but only 12 mo studies).
Treatment of Hirsutism

- OCPs
- Anti-androgens:
  - Spironolactone = an aldosterone antagonist
  - Flutamide = an androgen receptor antagonist
  - Efflornithine, the only topical, slows facial hair growth
- Electrolysis and laser treatment

Summary

- Lifestyle modification effective and 1st line
- OCPs = 1st line medication treatment
- Metformin good for metabolic complications

Trends in standard workup of adolescent PCOS performed by pediatric subspecialists

<table>
<thead>
<tr>
<th>Treatment of Patients Who Met Diagnostic Criteria for PCOS by Specialty</th>
<th>Endocrine</th>
<th>Gynecology</th>
<th>Adolescent Medicine</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>113</td>
<td>7</td>
<td>67</td>
<td></td>
</tr>
<tr>
<td>OCP</td>
<td>26% (27)</td>
<td>43% (3)</td>
<td>58% (39)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Medroxyprogesterone acetate</td>
<td>10% (11)</td>
<td>29% (2)</td>
<td>30% (20)</td>
<td>.0017</td>
</tr>
<tr>
<td>Metformin</td>
<td>50% (56)</td>
<td>14% (1)</td>
<td>5% (3)</td>
<td>&lt;.0001</td>
</tr>
<tr>
<td>Spironolactone</td>
<td>13% (3)</td>
<td>0</td>
<td>3% (2)</td>
<td>1.0000</td>
</tr>
<tr>
<td>Lifestyle modification</td>
<td>4% (5)</td>
<td>86% (5)</td>
<td>38% (26)</td>
<td>.0218</td>
</tr>
</tbody>
</table>

Transition of care

Forgotten condition—gets lost in the shuffle.
Review diagnosis periodically with teen and parent to assess understanding.
Chronic condition—labeling may help.

MENSTRUAL FLOW CHART

Please have this chart with you when you call or visit your health care provider.

Patient information

Written—LPCH Epic Micromedix
PCOS for teens
Website—youngwomenshealth.org

What is Polycystic Ovary Syndrome (PCOS)?

PCOS is a hormone imbalance that is very common. 15-21% of all women may have it and often not realize it. In this condition, the ovaries make too much of the "male" sex hormone (testosterone).

Symptoms may include:

- Irregular menstrual periods
- Heavy
- Acne on the face and body (hirsutism)
- Excess hair on the stomach, arms, and legs
- Weight gain or obesity

Diagnosis of PCOS

To find out if you could have PCOS, your doctor will ask questions about:

- Your menstrual periods
- How you are feeling physically and mentally
- Your family history
- Your medical history

To learn more about PCOS, visit www.youngwomenshealth.org.
Preconception Health and Health Care

My Reproductive Life Plan

Thinking about your goals for having or not having children and how to achieve those goals is called a reproductive life plan. There are many kinds of reproductive life plans, your plan will depend on your personal goals and dreams.

How to Make a Plan
First, think about your goals for school, for your job or career, and for other important things in your life. Then, think about how having children fits in with those goals.

If you do want to have children one day, think about when and under what conditions you want to become pregnant. This can help ensure that you and your partner are healthy and ready when you choose to have a baby. If you do not want to have children, then you might think about how you will prevent pregnancy and what steps you can take to be as healthy as possible.

Try to include as many details as possible in your plan. Some people find it helpful to write their plan down on a piece of paper or in a journal. Be sure to talk with your health care provider. Doctors and counselors can help you make your plan and achieve your goals.

Questions to Get Started
When making a reproductive life plan, the following questions might be helpful. These are priority considerations, but you may want to ask yourself if they will help you get started.

- Do you want to have children and when do you want to have them?
- Are you ready to become pregnant now?
- Are you ready to become pregnant in the near future?
- Are you ready to become pregnant at all?
- Are you not ready to become pregnant?
- Do you want to focus on your career first?
- Do you want to delay having children for other reasons?
- Do you want to have children but are not sure how to start?
- Do you want to have children but are not sure if you can afford them?
- Do you want to have children but are not sure if you can support them?
- Do you want to have children but are not sure if you can make a good parent?
- Do you want to have children but are not sure if you can provide a good home for them?
- Do you want to have children but are not sure if you can give them the best possible start in life?
- Do you want to have children but are not sure if you can give them the best possible education?
- Do you want to have children but are not sure if you can give them the best possible opportunities?
- Do you want to have children but are not sure if you can give them the best possible future?
- Do you want to have children but are not sure if you can give them the best possible chance to succeed?
- Do you want to have children but are not sure if you can give them the best possible chances to be happy?
- Do you want to have children but are not sure if you can give them the best possible chances to be healthy?
- Do you want to have children but are not sure if you can give them the best possible chances to be successful?
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Preconception Health and Health Care
Reproductive Life Plan
Tool For Health Professionals

Do you plan to have any (more) children at any time in your future? (Open ended and allows blanking.)
IF YES:
• How many children would you like to have? Encourages the person to calculate their total intended number of children
• How long would you like to wait until you or your partner becomes pregnant? Encourages the person to vision their own future!
Studies have shown an association between shorter birth intervals and increased risk of birth defects. Health guide

PCOS TEMPLATE
• PCOS Template with EPIC
• Dot phrase .pcostransition
• Data populates fields from the EMR
  – Automatically from local labs
  – With shortcut entered manually from outside labs
• Once we establish Variable Names, submit to EPIC HQ in Wisconsin
• Partner institution(s)