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COMMON GYNECOLOGICAL ISSUES IN ADOLESCENTS

SPEAKER

Dr Shikha Mahajan

CHAIRPERSONS

Dr Satinder Walia

Panelists

Dr Alka Sinha, Dr Deepa Passi, Dr Smita Ramachandran



- Director Pediatrics
- Centre for Child Health, BLK-MAX Hospital.
- Adolescent Health Specialist
- National Facilitator for adolescent health
- National ALS Instructor
- Executive Member, IAP Delhi, 2025

- Ph.D, Clinical Psychology
- Masters, School Psychology, Oklahoma USA & Del Univ.
- Over 25 years of professional experience as a child psychologist
- Clinical Psychologist at BLK MAX





- **Director - Gynaecology and Head Laparoscopic Surgery**
- **BLK-Max Super Speciality Hospital, New Delhi.**
- **MD (AIIMS)**
- **Diploma Gyne Endoscopy (Kiel - Germany)**
- **Fellowship in Robotic Surgery (WALS)**





NAME	DR. DEEPA PASSI
DESIGNATION	SENIOR CONSULTANT
CURRENT AFFILIATION	APOLLO HOSPITALS NOIDA & DELHI
ACHIEVEMENTS	PRESENTLY PRESIDENT AHA DELHI 2024 & 2025 2ND INDIVIDUAL AWARD AT NASHIK ADOLESCON 2024 Awarded the special women award in march 2025 PLACED IN APOLLO EXCELLENCE REPORT 2025



NAME	Dr Smita Ramachandran
DESIGNATION	Senior Consultant Pediatric and Adolescent Endocrinologist
CURRENT AFFILIATION	<ul style="list-style-type: none">• Venkateshwar Hospital, Dwarka• BLK-Max Hospital, Pusa Road
ACHIEVEMENTS	Co-Authored Book on Pediatric endocrinology, authored several chapters in endocrinology textbooks Over 35 publications in International and national journals



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Common Gynecological issues in Adolescents : Case based discussion

MODERATOR - Dr Shikha Mahajan

PANELISTS

- Dr Deepa Passi- Adolescent Health specialist
- Dr Alka Sinha- Senior Gynaecologist
- Dr Smita Ramachandran- Pediatric Endocrinologist



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CASE 1

DR DEEPA PASSI

- ✧ 16 years old Rupa
- ✧ Came to OPD with severe pain in lower abdomen during her periods
- ✧ Pain started a day prior to her periods & lasted for 2-3 days
- ✧ Severe enough to miss her school, associated with nausea /vomiting
- ✧ Attained menarche at 12 years age
- ✧ Cycles regular and flow moderate

HISTORY : No Non cyclical pain

Not sexually active

No h/o excessive white discharge, vaginal itching

PHYSICAL EXAM : No abnormalities noted

INVESTIGATIONS : CBC, ESR-Normal

Pelvic USG- Normal

What is the DIAGNOSIS and how would you proceed to manage it

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DYSMENNORHEA

- ✦ PAINFUL MENSTRUATION
- ✦ IT IS ONE OF THE MOST COMMON GYNAECOLOGIC COMPLAINTS
- ✦ **Primary** – menstrual pain occurring with no underlying pelvic pathology.
- ✦ **Secondary** – menstrual pain that occurs with an associated pelvic pathology.

- ✱ NO PATHOLOGY IN PELVIS
- ✱ 20% women have significant impairment of quality of life (interferes with school and work activities)
- ✱ 1-3 YEAR AFTER MENARCHE, 15-25 YEARS
- ✱ Primary dysmenorrhea is thought to occur secondary to the excessive release of prostaglandins ($\text{PGF}_2\alpha$ and PGE_2) by endometrial cells.
- ✱ Prostaglandins cause the muscles and blood vessels of the uterus to contract
- ✱ Level of PG is high on D1 of periods and declines as the bleeding continues and the uterus lining is shed off. So the pain declines after first few days of a period

- ✦ Lower abdominal or **pelvic pain**, which can radiate to lower back or anterior thigh.
- ✦ Pain is crampy in nature. It usually lasts for **48-72 hours** around the menstrual period, and is characteristically worst at the onset of menses.
- ✦ Pain can be associated with other symptoms- malaise, **nausea**, vomiting, diarrhea, dizziness.
- ✦ Abdominal and pelvic examinations (including speculum examination of cervix) are performed but are usually unremarkable.
- ✦ **Uterine tenderness** may be present.

WHO IS MORE LIKELY TO HAVE DYSMENORRHEA

- ❖ Attained menarche before 12 years age
- ❖ Age less than 20 years
- ❖ Heavy periods –last longer than 7 days
- ❖ Cigarette smoking
- ❖ Biological parent who has dysmenorrhea
- ❖ Nulliparity
- ❖ Depression and anxiety

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Primary dysmenorrhoea is diagnosed based on typical history alone

How will you manage Primary dysmenorrhoea

NO PATHOLOGY – SO MAINLY SYMPTOMATIC TREATMENT

Pharmacological

✦ Analgesia (First line):

- ✦ NSAIDs (ibuprofen, naproxen, mefenamic acid). They work by inhibiting the production of prostaglandins; which have been implicated in the pathogenesis of primary dysmenorrhea.
- ✦ And/or paracetamol

✦ 3-6 month trial of hormonal contraception (Second line):

- ✦ Monophasic combined oral contraceptive pill is most commonly used
- ✦ Intrauterine system (e.g Mirena coil) may also be effective.

✦ Non-Pharmacological

- ❖ Local application of heat (water bottles or heat patch)
- ❖ Transcutaneous Electrical Nerve Stimulation (TENS)
- ❖ Exercise –walking ,aerobics (produce chemicals that block pain)
- ❖ Good sleep
- ❖ Meditation and yoga
- ❖ Stop smoking (there is a clear relationship between smoking and dysmenorrhea)

Investigations needed when considering the possibility of
Secondary dysmenorrhoea (rare in adolescent population)

- ❖ Mullerian duct anomalies
- ✱ Endometriosis
- ✱ Adenomyosis
- ✱ Fibroids
- ✱ Pelvic inflammatory disease
- ✱ Adhesions
- ✱ Cervical stenosis
- ✱ Non-gynaecological differentials include **inflammatory bowel disease** and irritable bowel syndrome.

- ❖ Pelvic examination- if history suggestive of secondary dysmenorrhea or not responding to medical management.
- ❖ USG PELVIS
- ❖ If suspected PID- Endocervical or Vaginal swabs
- ❖ If indicated –cervical cytology to r/o cervical malignancy
- ❖ MRI PELVIS (to rule out torsion ,adenomyosis, endometriosis ,inconclusive USG
- ❖ Laparoscopy



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CASE 2

DR SMITA RAMACHANDRAN

A 15 year girl presented with:

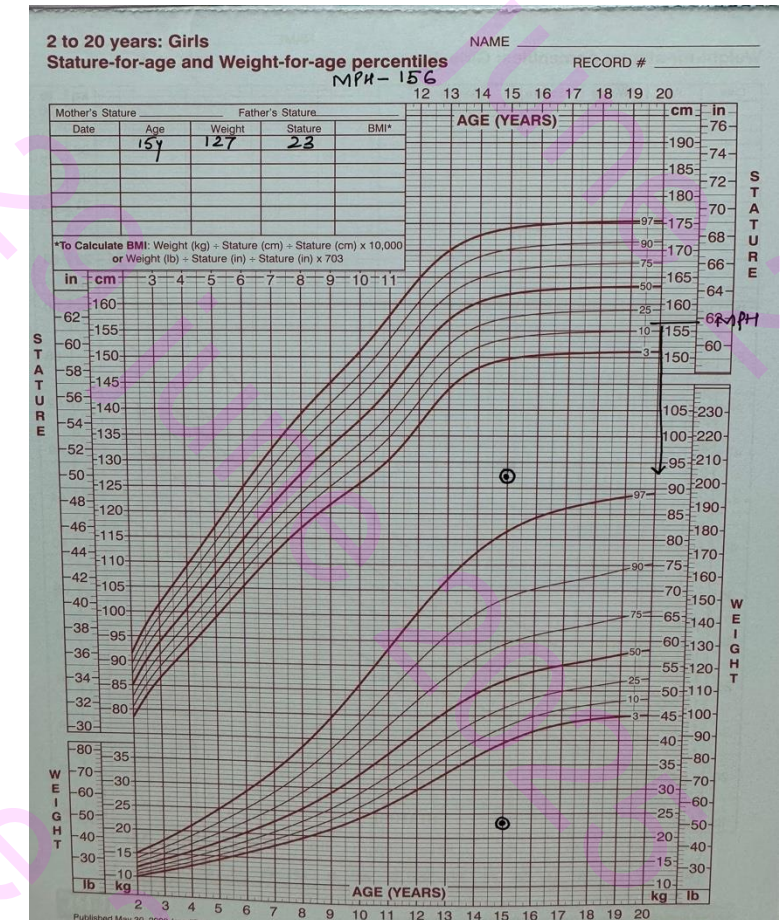
- Poor height and weight gain (noticed from 6yrs onwards)
- Not developing secondary sexual characteristics
- Her scholastic performance was average, with no H/O any acute or chronic illnesses or medications
- She was born of a non-consanguineous marriage / NVD /Term/ birth weight 2.6kg
- Neonatal period uneventful. Physical exam at birth was normal.

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PHYSICAL EXAMINATION

- ❖ Height -127 cm (- 5.5 SDS) , Mid Parental Height- 156cm
- ❖ Weight -23kg
- ❖ Upper : lower segment : 1, with arm span of 131 cm .
- ❖ Blood pressure - 100/70mmHg
- ❖ Pulse rate 86 /min regular with no radio femoral delay.
- ❖ SMR - GBR1L1A1P1 (Stage 1)

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- ✱ Hb-13.8 g/dl
- ✱ Liver and renal function test -NORMAL
- ✱ Blood glucose and calcium profile - NORMAL

Hormonal profile

- ✱ TFT - WNL
- ✱ LH - 33.4mIU/ml, FSH - 120mIU/ml E2 - 5pg/ml
- ✱ Bone age was 13 years

At this stage with these reports and physical findings what is your preliminary diagnosis ?
And how would you proceed to investigate ?

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Physical findings :

- ✱ Auxology : Height -5.5SD
- ✱ Tanner stage 1
- ✱ Bone age 13yrs

Biochemical :

- ✱ LH-high
- ✱ FSH- high
- ✱ E2- low

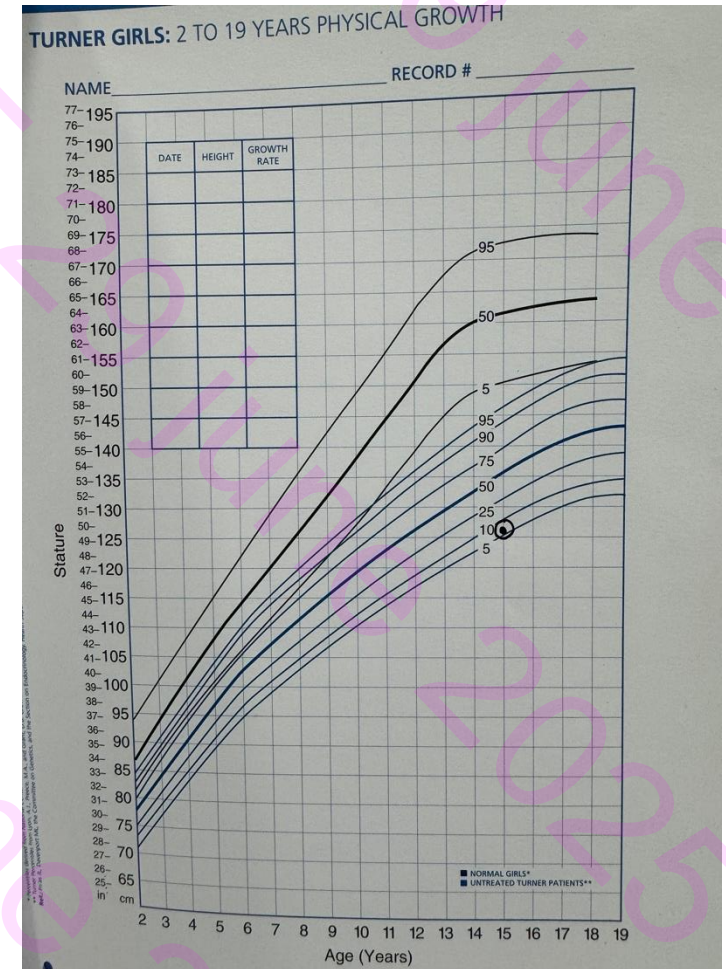
***15yrs old girl with short stature and delayed puberty with
Hypergonadotrophic hypogonadism***

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Her 30 cell karyotype was 45 X0

TURNER SYNDROME

- ✿ Serum TTG IgA – Normal
- ✿ USG abdomen revealed horse shoe kidney and hypoplastic uterus .Ovaries could not be visualized
- ✿ 2D echo Normal



- ✱ When do you label a child as Delayed puberty ?
- ✱ And what is the first line of evaluation ?

- ✱ Absence of any sign of puberty in a child at a chronological age $>2SD$ above the mean age of pubertal development in a given population
- ✱ For boys testicular volume <4 ml at 14 years of age or failure to achieve adult testicular volume 4-5 years after onset of puberty (2,3)
- ✱ For girls absence of thelarche at 13 years of age or no menarche after 16 years or 3 years after thelarche.



<i>Variant of normal puberty timing</i>	<i>Hypogonadotropic hypogonadism</i>	<i>Hypergonadotropic hypogonadism</i>
Constitutional delay of growth and/or puberty Sporadic Familial	Congenital Monogenic mutations Kallmann syndrome (e.g., <i>KAL1</i>) Normosmic idiopathic hypogonadotropic hypogonadism (e.g., <i>GNRHR</i>) Gonadotropin gene mutations (<i>LHβ</i> , <i>FSHβ</i>) Multiple pituitary hormone deficiencies Idiopathic Genetic (e.g., <i>PROP-1</i>) Adrenal hypoplasia congenital (<i>DAX1</i>) Obesity-related (<i>LEP</i> , <i>LEPR</i> , <i>PCSK1</i>) Syndromic (Prader-Willi, CHARGE, Lawrence-moon, Bardet-Biedl syndromes)	Congenital Males Klinefelter syndrome Pure gonadal dysgenesis Defects in steroidogenesis Leydig cell hypoplasia (<i>LHCGR</i>) Androgen insensitivity syndromes Testicular regression syndrome or cryptorchidism Noonan syndrome Females Turner syndrome Pure gonadal dysgenesis Gonadotropin receptor mutations (<i>LHCGR</i> , <i>FSHR</i>) Androgen insensitivity syndromes Estrogen receptor-alpha mutation (<i>ESR1</i>) Noonan syndrome
<i>Functional and systemic disorders</i> Nutritional disorders Malnutrition Anorexia nervosa Excessive energy expenditure, exercise Psychological or emotional stress Chronic illness (gastrointestinal disease, renal failure, hepatic disease, hematologic abnormalities, malignancy, pulmonary disease, etc.) Endocrinopathies Diabetes mellitus Growth hormone deficiency Hypothyroidism Hyperprolactinemia Glucocorticoid excess Medication or drug effect (i.e., anabolic steroids, opiates)	Acquired Suprasellar tumors (e.g., craniopharyngiomas) Infiltrative/inflammatory (e.g., histiocytosis X) Effects of radiotherapy Effects of surgery Cranial trauma	Acquired Males Surgical or traumatic castration Bilateral orchitis Chemotherapy, radiotherapy effects Hemochromatosis Females Surgical or traumatic castration Premature idiopathic ovarian failure Autoimmune ovarian failure Fragile X (<i>FMR1</i>) premutation carrier Chemotherapy, radiotherapy effects Galactosemia

1. X ray of hand and wrist for Bone age
2. CBC, electrolytes, creatinine, bicarbonates, alkaline phosphatase and other relevant test to rule out chronic illnesses.
3. Basal FSH, LH (LH is better predictor for onset of puberty than FSH)
4. Serum estrogen in females
5. IGF -1 levels for detecting growth hormone deficiency.
6. Thyroid function test.
7. MRI brain and pituitary to rule out CNS lesions
8. Gonadotropin release hormone GnRH test
9. Serum Prolactin levels
10. Genetic testing recommended in patients with features indicative of specific syndromes.

- ✧ How do you manage these cases ?
- ✧ What additionally may you need to do in Turner syndrome ?

Hormone replacement therapy

- ✦ Start with low dose estrogen which is slowly built up to maximum dose by 2 yrs
- ✦ Progesterone is added at full dose or if menstruation starts

In Turner 's syndrome additionally

- ❖ Would require growth hormone for short stature and annually need to be followed up for other complications.

Follow Up in Turner Syndrome

	At diagnosis	After diagnosis (childhood)	After diagnosis (adults)
Weight/BMI	Yes	Every visit	Every visit
Blood pressure	Yes	Every visit	Every visit
Thyroid function (TSH and (free) T4)	Yes	Annually	Annually
Lipids			Annually if at least one cardiovascular risk factor ^a or regional recommendation
Aminotransferase, GGT and alkaline phosphatase		Annually after 10 years of age	Annually
HbA1c with or without fasting plasma glucose		Annually after 10 years of age	Annually
25-Hydroxyvitamin D		Every 2–3 years after 9–11 years of age	Every 3–5 years
Celiac screen		Starting at 2 years; thereafter every two years	With suggestive symptoms
Renal ultrasound	Yes		
Audiometric evaluation	Yes*	Every 3 years	Every 5 years
Ophthalmological examination	Yes [#]		
Dental evaluation	Yes, if no previous care has been established		
Clinical investigation for congenital hip dysplasia	Yes, in newborns		
Skin examination	At diagnosis	Annually	Annually
Bone mineral density			Every 5 years and when discontinuing estrogen
Skeletal assessment		5–6 years and 12–14 years (see 6.1.10.)	



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CASE 3

DR ALKA SINHA

DR SMITA RAMACHANDRAN

15 year old girl presented with :

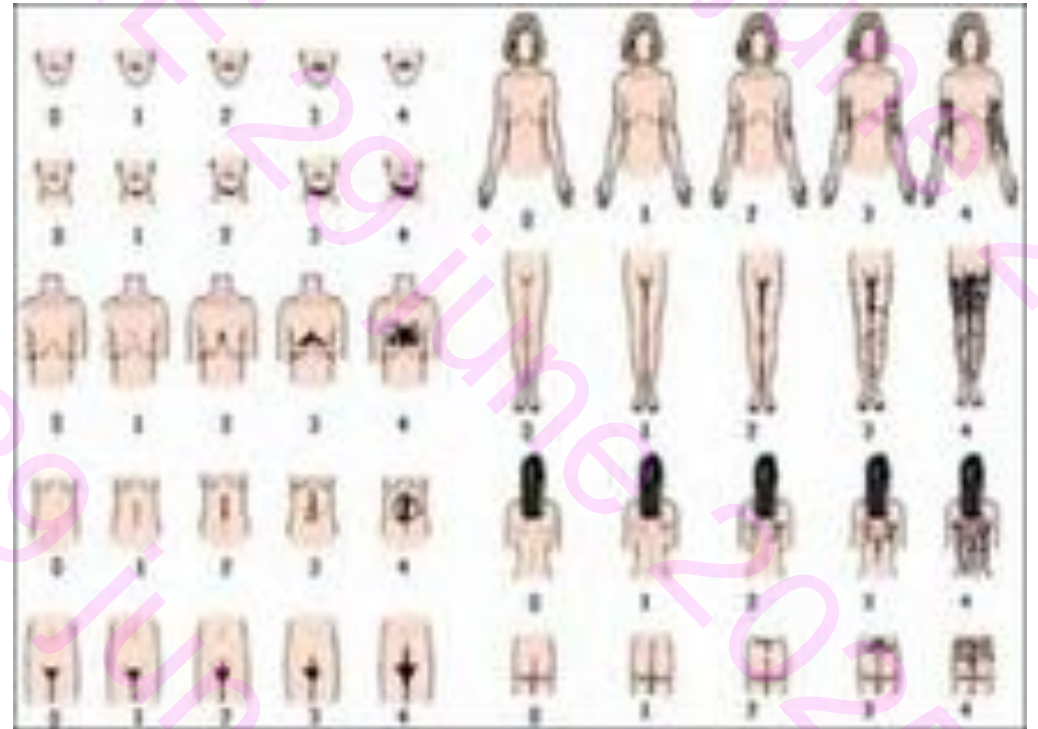
- ✱ Complaints of Amenorrhea for 4 months
- ✱ Followed by continuous bleeding for last 15 days
- ✱ Previous cycles irregular since menarche. But irregularity increased for last 1 year : cycles every 1-3 months. Bleeding sometimes heavy.
- ✱ Menarche 4 years back at 11 years of age

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EXAMINATION

- ✦ Height 5 feet
- ✦ Weight 68 kgs
- ✦ BMI-29.5
- ✦ Modified Ferriman gallaway score 8
- ✦ Acanthosis nigricans +

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- ✱ What is your primary interpretation at this stage based on history and examination ?
- ✱ How would you like to proceed ?

DIFFERENTIAL DIAGNOSIS

- ✱ PCOS
- ✱ NCCAH
- ✱ Cushings disease

- ✱ CBC WNL
- ✱ TSH 2.3
- ✱ Serum Prolactin (Pooled)-17.6
- ✱ Total testosterone- 73 ng/dl
- ✱ OGTT-normal
- ✱ Fasting blood sugar- 86 mg%
- ✱ Insulin Fasting : 23 mIU/L
- ✱ Ultrasound- Endometrial thickness 17 mm. Ovaries normal
- ✱ Lipid profile

- ✱ Cortisol – 15ug/dL
- ✱ 17 OHP- 2nmol/L

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CASE 3 --- POLYCYSTIC OVARY SYNDROME

The diagnostic criteria of PCOS has evolved from

- NIH criteria in 1990
- Rotterdam classification in 2003
- Androgen excess society 2006, to the current classification.

The Criteria suggest for PCOS by international consensus

Required	Optional
1. Oligomenorrhea or Irregular menses	polycystic ovarian morphology
2. Evidence of hyperandrogenism (Clinical or biochemical)	Severe cystic acne

- ✦ PCOS should be diagnosed using the **revised consensus Rotterdam criteria**
- ✦ In adolescents this requires the presence of both
 - ✦ clinical/biochemical hyperandrogenism,
 - ✦ ovulatory dysfunction
- ❖ There are no definitive criteria to define polycystic ovary morphology (PCOM) on ultrasound in adolescents, hence it is not recommended in adolescents
- ❖ Serum AMH should not be used in adolescents

Irregular menstrual cycles are defined as:

- ✱ Normal in the first year post menarche as part of the pubertal transition
- ✱ 1 to < 3 years post menarche: < 21 or > 45 days
- ✱ 3 years post menarche to perimenopause: < 21 or > 35 days or < 8 cycles per year
- ✱ 1 year post menarche > 90 days for any one cycle
- ✱ Primary amenorrhea by age 15 years or >3 years post thelarche

- ✱ Severe acne and hirsutism in adolescents
- ✱ A modified Ferriman Gallwey score of > 4 to > 6 should be used to detect hirsutism
- ✱ New-onset severe or worsening hyperandrogenism, including hirsutism, requires further investigation to rule out androgen-secreting tumours

- ✦ Elevated Total or free testosterone levels in PCOS
- ✦ Free testosterone can be estimated by the calculated free androgen index.
- ✦ Androstenedione (ANSD) and dehydroepiandrosterone sulfate (DHEAS) should be considered if total or free testosterone is not elevated.

✱ How do you manage such adolescents ?

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SHORT TERM GOALS

- ✱ Regulation of menstruation
- ✱ Control of hirsutism
- ✱ Reduction of weight
- ✱ Psychological issues

LONG TERM GOALS

- ✱ These are not of immediate concern to the adolescent but are important for later life
- ✱ Prevention of Metabolic risk factors,
 - Diabetes
 - Endometrial hyperplasia,
 - Cardiovascular risk
 - Infertility

EXERCISE

- ✱ Lifestyle intervention (exercise alone or multicomponent diet combined with exercise and behavioural strategies) should be recommended for all women with PCOS
- ✱ Adolescents should aim for at least 60 minutes of moderate- to vigorous-intensity physical activity per day including activities that strengthen muscle and bone, at least three times per week.

DIET

PSYCHOLOGICAL COUNSELLING

- ✱ Should screen for depression in all adolescents with PCOS and manage accordingly.
- ✱ PCOS can influence psychosexual function

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✱ MENSTRUAL IRREGULARITY – Combined Oral Contraceptive pills (COCP)

✱ HIRUSITISM- COCP

Laser Therapy

Photo epilation

ANTI ANDROGENS (SPIRINOLACTONE)

✱ METABOLIC SYNDROME – METFORMIN

- ✦ For adolescents who have features of PCOS, but do not meet diagnostic criteria, an 'increased risk' could be considered and reassessment advised at or before full reproductive maturity, 8 years post menarche.
- ✦ This includes those with PCOS features before combined oral contraceptive pill (COCP) commencement, those with persisting features and those with significant weight gain in adolescence.

- ✦ Important not to miss the diagnosis of PCOS in adolescent girls as timely intervention can prevent lot of morbidity
- ✦ Pediatricians, Family physicians ,Dermatologists who take care of adol must be well aware of this condition for diagnosis and early referral
- ✦ Any girl with Acne,obesity, Hirsutism and menstrual abnormalities should be screened for PCOS
- ✦ Look out for Acanthosis nigricans –a marker of insulin resistance and associated metabolic syndrome