AMENORRHOEA
Primary & Secondary

DR. katayoun Arfaie
PRIMARY AMENORRHOEA

1. No menstruation by the age of 13 years accompanied by failure to grow properly or develop sec. sexual characteristics.

2. No menstruation by age of 15 when growth and sexual development are normal.
SECONDARY AMENORRHOEA

Secondary absence of menses for six months (or greater than 3 times the previous cycle interval) in a woman who has menstruated before.

- Pregnancy, lactation or hysterectomy must be excluded
- Prepubertal and post-menopausal conditions are also to be excluded as physiological causes
CLINICAL APPROACH

There is a difference of opinion about the age at which Primary Amenorrhoea should be investigated → 18 yrs. often suggested.

Provided the patient has developed normal sec. sex. Characteristics and cryptomenorrhoea has been excluded.
There is a difference of opinion about the age at which **Primary Amenorrhoea** should be investigated → 18 yrs. often suggested.

Provided the patient has developed normal sec. sex. Characteristics and **cryptomenorrhoea** has been excluded.
While those patient with Primary amenorrhoea and sexual infantilism should be investigated at $\geq$ age of 15 years or 16 years (may be earlier).
• Accurate, adequate **history** is essential to reach a firm diagnosis

• Specific questioning is necessary to establish diagnosis of Primary or Secondary amenorrhoea

• Is the amenorrhoea is truly secondary (e.g. prev. menses were actually steroid – induced)
• Careful physical examination aids in reaching a fairly firm provisional diagnosis

• In minority, there is a need to go beyond simple out-patient investigation
CAUSES OF AMENORRHOEA

A. Disorder of outflow tract and or uterus
B. Disorders of ovary
C. Disorders of Ant. Pituitary
D. Disorders of Hypothalamus
Evaluation and Management of Primary Amenorrhea

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University of Nebraska Medical Center
Causes of Primary Amenorrhea

- American Society of Reproductive Medicine classifies causes of primary amenorrhea into three distinct groups

  - Primary Amenorrhea with:
  - Breast Development (30%)
  - No breast development AND high FSH (40%)
  - No breast development AND low FSH. (30%)

ASRM Practice Committee.
Causes of Primary Amenorrhea

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Mullerian Agenesis

- Mayer-Rokitansky-Kuster-Hauser (MRKH) syndrome
- Complete absence of uterus, cervix and the upper 2/3 of the vagina
  - Incidence 1/5000 (1/4000-1/10,000 female newborns)
  - Normal XX Karyotype
  - Normal ovarian function
  - Otherwise normal pubertal development
- Causes
  - Mutations in Antimullerian Hormone or Antimullerian Hormone receptor
  - Association with Wnt gene

Mullerian Agenesis

• Evaluation: Normal breast development, normal secondary sexual characteristics
• Laboratory: Normal XX karyotype, normal LH, FSH
• Pelvic Exam:
  • Normal external genitalia
  • absence of internal midline structures
  • + vaginal dimple
Diagnosis

Left: MRI showing absence of uterus and vagina
Mullerian Agenesis

• Associated Conditions
  • 1/3 concurrent urinary tract anomalies
    • Ex: Ectopic kidney, renal agenesis, horseshoe kidney
  • 12% associated skeletal anomalies
    • Ex: spinal anomalies, absent digits, webbed fingers, toes
• Part of work up needs to include an abdominal CT to evaluate for renal anomalies
Mullerian Agenesis: Treatment

- Dilators (Frank and Ingram)
  - Dilate at a 15 degree angle daily after warm bath for 20 minutes.
  - Progressively work up to larger dilators
  - Success defined as non-painful intercourse or vaginal length of 7cm
  - Studies demonstrate up to an 88% success rate at 19 months of use.
McIndoe Neovagina

• Use a skin graft or artificial skin placed over a mold forming a tube with one closed end
• Incision made in the vaginal dimple and cavity dissected to level of peritoneum.
• Labia majora are sewn together.
• Bed rest for 7 days and then mold removed.
Transverse Vaginal Septum

• Failure of canalization of distal third of the vagina
  • Most common in upper and middle third of vagina

• Diagnosis
  • Usually present after puberty with amenorrhea and pelvic pain
  • Can present with hematocolpos, hematometra
  • Does not bulge with valsalva maneuver
  • MRI helps with diagnosis
Vaginal Septum

Illustration by John Parker. Wheeless, CR. And Roenneburg, M.L. Atlas of Pelvic Surgery
Imperforate Hymen

- Most common obstructive lesion of the female genital tract
  - 1/1000 female births
- Classic appearance of bulging, blue-domed, translucent membrane
- Can present with hematocolpos or urinary retention
- Differs from vaginal septum in that an imperforate hymen bulges with valsalva
- Treatment: Surgical Resection
  - Hymenectomy versus hymenotomy
Androgen Insensitivity

• Incidence 1/60,000
  • although 9% of causes of primary amenorrhea
• Genetics: X-linked recessive
• Phenotype: Female; Genotype: XY
  • Female external genitalia with small vaginal dimple
  • Absent uterus and cervix.
  • Cryptorchidic gonads
  • Absent axillary and pubic hair
  • +Breast development
• Cause: Mutations in the androgen receptor
Androgen Insensitivity

• Physical Exam
  • Slim and taller than average female
  • Large breasts with juvenile nipples
  • Absent pubic/axillary hair, no acne or other signs of androgen action
  • May have inguinal hernia
  • Normal external genitalia, blind vaginal pouch and absence of midline structures
• Laboratory: Testosterone in the normal to high male range
Androgen Insensitivity: Removal of Gonads

- Location of testicular gonads is variable
  - Intrabdominal cavity
  - Labialscotal folds
  - Inguinal region
- Recommend removal after complete pubertal development
  - Enhance bone maturation and puberty
  - Recommend at age 16-18
  - Once testes removed, treat with hormone replacement therapy
- Incidence of neoplasia
  - 22% incidence of malignancy
  - Most common histology is Leydig cell hyperplasia
CONSIDER THE DIAGNOSIS IN A FEMALE CHILD:

1. With inguinal hernia
2. With $1^0$ amenorrhoea and absent uterus
3. When body hair is absent

MANAGEMENT:

- These patients are female.
- The gonads must be removed after puberty → then HRT started

- Rare cases of incomplete test. feminization do occur → have variable degrees of masculinization
## Causes of Primary Amenorrhea

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### Hypergonadotrophic Hypogonadism

Gonadal dysgenesis

Turner’s Syndrome

- Classically 45 XO or mosaic
  - Incidence 2,500-10,000 live borns
  - 99% of pregnancies affected end in SAB
- Cause: Absence of ovarian determinant genes result in premature loss of germ cells
  - Fetuses with Turner’s have the same amount of germ cells at midgestation as do 46, XX
  - As gestation continues, accelerated loss of germ cells occurs
  - Many XO individuals lose all germ cells prior to birth; less than 15% have enough germ cells to start pubertal process by adolescence
Turner’s Syndrome

- Short stature
- Low hairline
- Shield-shaped thorax
- Widely spaced nipples
- Shortened metacarpal IV
- Small finger nails
- Brown spots (nevi)
- Characteristic facial features
- Fold of skin
- Constriction of aorta
- Poor breast development
- Elbow deformity
- Rudimentary ovaries
- Gonadal streak (underdeveloped gonadal structures)
- No menstruation
Turner’s Syndrome

- Associated Abnormalities
  - Cardiac Anomalies
    - Coartation of aorta in 30% of patients, also bicuspid aortic valve, mitral valve prolapse
    - Recommend echocardiography be performed every 3-5 years
  - Renal Anomalies
    - Horseshoe Kidney
    - Need retroperitoneal ultrasound once diagnosed
  - Hypothyroidism
    - 10% of patients with Turner’s Syndrome
    - Recommend yearly screening of T4/TSH and antibodies
  - Deafness (audiometry)
- Streak ovaries present
- Gonadotrophins ↑↑
- ↓ Estrgoens

- Mosaic Chrom. Pattern
  (e.g. XO/XX) → lead to various degrees of gonadal dysgenesis and sec. amen. + premature menopause

- If Y-Chrom is present in the genotype → risk of gonadal malign. makes gonadectomy advisable
Gonadal Dysgenesis: 46XX

- Refers to a number of conditions in which abnormal development leads to streak gonads
- Incidence: <1/10,000 in women less than 30
- Inherited
  - Familial inheritance 7-30%
  - Premutations in the FMR1 gene (Fragile X Syndrome)
    - 15% of carriers have POF
  - Associated with autoimmune diseases (18-30%)
    - Hashimoto’s Thyroiditis, Addison’s disease, hypoparathyroidism, vitiligo
- Acquired
  - Radiation, chemotherapy
  - Environmental
    - Childhood viruses
Gonadal Dysgenesis: 46XX

- **Diagnosis:**
  - >3 months of amenorrhea + FSH in the menopausal range
  - Ultrasound; >60% of patients have undetectable ovaries by ultrasound. Majority show no follicular growth
  - DEXA scan in addition to screening for autoimmune diseases

- **Hormone Replacement**
  - Low-dose estradiol(1/2 mg/day and step up) for 12-18 months before addition of progestogenic agent
  - Add progesterone in order for regular menstruation.

- **Fertility**
  - 5-10% of spontaneous pregnancy as patients with gonadal dysgenesis will cycle inconsistently.
  - Recommend OCPs in adolescent population to prevent unwanted pregnancy.
Gonadal Dysgenesis: 46 XY Swyer Syndrome

- Cause: Associated with mutations in the SRY gene.
- Streak gonads present; No testes formation
  - Therefore Anti-Mullerian hormone and testosterone are not produced thus
    - Normal uterus and fallopian tubes, female external genitalia
  - Estrogen also not produced from streak gonads therefore breast development does not occur
- Elevated FSH/LH
- Streak Gonads need removal as they are at increased risk (25%) for germ cell tumors: most common gonadoblastoma.
Swyer Syndrome

Dysgerminoma in an adolescent patient with Swyer Syndrome
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</tr>
<tr>
<td>Other CNS</td>
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</tr>
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<td>3</td>
</tr>
<tr>
<td>PCOS</td>
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<td>Other</td>
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**Hypogonadotrophic Hypogonadism**

Prolactinoma

- Most common cause of pituitary related amenorrhea
- Mechanism
  - Elevated PRL levels can suppress hypothalamic GnRH secretion
  - Higher the PRL level, the greater disruption of the menstrual cycle
  - Rule out hypothyroidism!
  - Medications?
- Imaging
  - MRI of pituitary fossa if PRL is >100ng/mL OR if visual symptoms
- Treatment
  - Bromocriptine/Cabergoline
  - 80-90% of hyperprolactinemia will resolve and 80% of microadenomas will shrink
  - Resort to transsphenoidal surgery if medical therapy fails.
Prolactinoma

**Fig. 2.** a MRI: microadenoma 8 mm (arrow) in a 14-year-old female. b MRI: macroadenoma in a 15-year-old male (pretreatment). c MRI: empty sella post-treatment in the same patient
Other Pituitary Causes of Amenorrhea

<table>
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<tr>
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<th>Systemic Inflammatory Disease</th>
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<td>Nonfunctional Tumors (craniopharyngioma)</td>
<td>Arterial Aneurysm</td>
<td>Panhypopituitarism</td>
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Hypothalamic Amenorrhea

- Functional hypothalamic amenorrhea
  - Stress, nutrition, and exercise related
  - Alterations in normal pulsatile release of GnRH.

- Mechanism
  - Complex interplay between neuropeptides
    - Leptin
      - Lower levels of leptin (malnourished, anorexia) seem to decrease amount of leptin and cause amenorrhea.
    - Cortisol
      - Stress related levels CRH interfere and inhibit GnRH
  - Exercise
    - Chronic imbalance between calorie intake and consumption lead to hypothalamic dysfunction
Hypothalamic Amenorrhea

- Primary Amenorrhea and Eating Disorders
  - Despite treatment, adolescents with eating disorders and primary amenorrhea progress through puberty at a slowed rate
  - Estimate weight at which menarche will resume by prepubertal weight
Female Athlete Triad

- Established as a diagnosis in 1992
- Amenorrhea, osteoporosis, and eating disorder among female athletes
- Most common: gymnastics, ballet, and long-distance running
- Bone Mineral Density evaluation
  - Athletes generally have higher BMD
  - A Z-score less than -1.0 requires evaluation
Estrogen and the Bone

- **Estrogen in females**
  - Deficiency result in an ↑ in osteoclastic bone resorption and osteoporosis

- **Testosterone in males**
  - Deficiency result in low bone mass
Female Athlete Triad

- Counseling about Eating Disorders
  - Screening tests:
    - Eating Attitudes Test
    - SCOFF questionnaires
  - Dietician
    - Minimal goal of 30 kcal/kg of lean body mass
    - Dairy, iron, and protein rich foods
  - Referral to psychologist

- Role of Oral Contraceptives
  - Some improvement in BMD but does not restore bone mass to age–matched controls
  - Need to address underlying pathology—focused counseling with regard to nutrition and psychology
Kallman Syndrome

• Cause
  • X-linked recessive mutation in the KAL gene
  • Codes of an adhesion molecule results in lack of migration of GnRH neurons from the olfactory placode.

• Characteristics
  • Hypogonaotrophic hypogonadism
  • Anosmia
  • Midline facial defects
  • Occasional renal agenesis
  • See absence of pubertal development and primary amenorrhea

• Treatment
  • Hormone replacement therapy to promote sexual maturation
  • Fertility is possible using IM gonadotropins
Kallman Syndrome

Figure 7 - hormones and Kallmann's syndrome

- GnRH
- LH & FSH
- Testes
- Ovaries
- Sperm cells
- Testosterone
- Oestrogen
- Egg cell

Voice "breaks" growth of muscle tissue enlargement of genitalia facial, pubic & axillary hair

Broadening of hips menstruation begins development of breasts pubic & axillary hair
Polycystic Ovarian Syndrome

• 5-10% of adult women and increasing in prevalence in the adolescent population

• Diagnosis
  • At least 2 of the following:
    ◦ Chronic Anovulation
    ◦ Clinical or biochemical evidence of excess androgen
    ◦ Polycystic ovaries on ultrasound
  • Typically present with secondary amenorrhea/oligomenorrhea but represent 3% of diagnoses of primary amenorrhea
  • Important to diagnose given metabolic abnormalities
    ◦ Recent study of adolescent population showed 62% had already developed insulin resistance
    ◦ Dyslipidemia
    ◦ Obesity
Polycystic Ovarian Syndrome

Scoring systems can assess the severity of hirsutism. The Ferriman-Gallwey model quantitates the extent of hair growth in nine key anatomic sites (seven of them are shown). Hair growth is graded using a scale from 0 (no terminal hair) to 4 (maximal growth), for a maximum score of 36. A score of 8 or more indicates the presence of androgen excess. (Adapted from Moncada E. J Clin Endocrinol Metab. 1970 and Ferriman D, Gallwey JD. J Clin Endocrinol Metab. 1961)
Congenital Adrenal Hyperplasia

- Enzyme defect leading to excessive androgen production
- Milder form of disease diagnosed later in life (late onset)
- May present with primary amenorrhea but even more classical: hirsutism, virilization, anovulation
- Most commonly a defect in 21-hydroxylase leading to an accumulation of its substrate 17-hydroxyprogesterone
- Diagnosis:
  - Fasting 17-OHP
  - If >300ng/dL → ACTH stim test
  - Levels >1000 ng/dL are indicative of late-onset CAH
Constitutional Delay

- Puberty occurs at a time greater than 2.5 standard deviations from the mean
- Family history of delayed puberty
- Characteristics:
  - Significantly shorter
  - Bone age lags behind age matched controls
  - Often present at early Tanner stage 2
  - Low gonadotropin levels
  - Diagnosis of exclusion—exclude other reproductive disorders
Review: Causes of Primary Amenorrhea

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Review: Evaluation of Amenorrhea

- Patient History
  - OB/GYN: Pubertal development, premenstrual symptoms, dysmenorrhea/cyclic abdominal pain
  - Past Medical History: chronic illness, exposure to radiation, current medications
  - Social History: exercise, weight loss, illicit drug use
  - Family History: history of pubertal delay, infertility,
  - Review of Systems: anosmia, galactorrhea, headaches, visual changes, hirsutism or acne, s/sx of thyroid disease, vasomotor symptoms
Review: Evaluation of Amenorrhea

• Physical Exam
  • Growth chart/BMI
  • Secondary sexual characteristics: Tanner staging, breast development, pubic hair
  • Dysmorphic features: webbed neck, short stature, widely spaced nipples
  • Hirsute features, Acne
  • Thyroid exam
  • Pelvic exam: rudimentary or absent uterus, transverse vaginal septum, imperforate hymen, virilization, clitiromegaly
History and physical examination completed for a patient with primary amenorrhea

Secondary sexual characteristics present

No

Measure FSH and LH levels.

FSH and LH < 5 IU per L

Hypogonadotropic hypogonadism (Table 4)

Karyotype analysis

46,XX

Premature ovarian failure

FSH > 20 IU per L and LH > 40 IU per L

Hypergonadotropic hypogonadism

Karyotype analysis

46,XY

Androgen insensitivity syndrome

45,XO

Turner's syndrome

Yes

Perform ultrasonography of uterus.

Uterus absent or abnormal

Karyotype analysis

46,XX

Müllerian agenesis

Evaluate for secondary amenorrhea (Figure 2).

46,XY

Androgen insensitivity syndrome

Uterus present or normal

Outflow obstruction

No

Imperforate hymen or transverse vaginal septum

Yes
Case

- 16 year old African American Female presenting to her primary care physician with absence of menarche
- On exam
  - Minimal axillary hair tanner stage II
  - Tanner stage II breast development
  - Pelvic exam not performed
- Laboratory Values
  - FSH >20 and LH >40
  - TSH, PRL normal
- Ultrasound Performed
  - Absence of reproductive organs
Work-up

- What do you want to do now?
  - Karyotype: 46XY
  - Diagnosis: Swyer Syndrome

- Now what?
  - Patient taken to the operating room for bilateral gonadectomy
  - Operative findings: rudimentary uterus, streak gonads, and a 1 cm nodule along the area of the left gonad.
  - Histopathology revealed a seminoma of the left gonad

- Follow up
  - Patient referred to gynecological oncology
    - Negative AFP, B-hcg, LDH, CMP, CXR, Abdominopelvis CT
    - Surveillance
  - On hormone replacement therapy
  - Regular follow up with GYN ONC and REI
• Secondary amenorrhea
4. **ASHERMAN’S SYNDROME:**

Sec. amenorrhoea following distruction of the endomet by over zealous curettage \(\rightarrow\) multip. Synechiae show up on “Hysterography”.

**MANAGEMENT:**

\(\rightarrow\) breakdown intraut. Adhesions through hysteroscope \(\rightarrow\) insert an IUCD to deter reformation \(\rightarrow\) hormone therapy \((E_2 + P)\)

5. **INFECTION**

e.g. Tuberculosis.
B. DISORDERS OF THE OVARIES

1. CHROMOSOMAL ABNORMALITIES

Turner’s syndrome (45 x 0) → gonadal dysgenesis

FEATURES:

i. Amenorrhoea (1°, rarely 2°)

ii. Short stature

iii. Failure of sec. sex. Develop

iv. Webbing of the neck

v. ↑ carrying angle

vi. Shield chest

vii. Coartution of aorta

viii. Renal collecting syst. defects
Abnormality of carrying Angle

Cubitus varus  Normal  Cubitus valgus

Any variation of the angle that is more than 15° is known as cubitus valgus and less than 5° are called cubitus varus.
4. **PREMATURE MENOPAUSE:**
   Ovarian failure….due to
   i. Auto-immune dis. (associated with Addison’s dis. ??)
   ii. Viral infection (e.g. mumps)
   iii. Cytotoxic drugs

5. **PCOs:**
   - Mostly present with classical Stein-Leventhal syndrome (of oligomenorrhea, obesity, hirsuitism, and infertility)
   - However a substantial group will have sec. amenorrhea with no obesity or hirsuitism
   - Diagnosis is made by finding ↑ LH/FSH ratio
   - Confirmation is made by laparoscopy.
   - USS ±
1. Pituitary Tumor causing “Hyperprolactinemia”

\[ \approx 40\% \text{ of women with hyperprolactinemia will have a pituitary adenoma} \]

Pit. Fossa XR is necessary in all cases of amenorrhoea – particular 2°.
FEATURES:
In coned view:
✦ Erosion of clinoid process
✦ Enlarge of pituitary fossa
✦ Double flooring of fossa
➢ If any of above features seen
➢ CT san or MRI + Assessment of visual fields
MANAGEMENT:

🌟 Bromocriptine (Dopamine agonist)
- Suppresses prolactin secretion
- Corrects estrogen deficiency
- Permits ovulation
- ↓ Size of most prolactinomas

🌟 Surgical removal of tumor ➔
- If extracellular manifestation (e.g., pressure on optic chiasma) or if patient cannot tolerate or respond to medical Rx.
2. **OTHER CAUSE OF ↑ PROLACT.**
   - Drugs: e.g. phenothiazines, methyl-dopa, metclopramide, anti-histamines, and morphine.

3. **CRANIOPHARYNGIOMA**
   - Other intracranial tumor

4. **SHEEHAN’S SYNDROME**
   - Necrosis of ant. pituitary due to severe PPH
     - Pan – or partial hypopituitarism
   - It is rare problem today due to better obstetric care and adequate blood transfusion
D. DISORDERS OF HYPOTHALAMUS

♣ Commonest reason for hypogonadotrophic sec. amenorrhoea

♣ Often associated with stress e.g. in migrants, young women when leave home, university students

♣ Diagnosis by exclusion of pituitary lesions.

♣ Hormone therapy or ovulation induction is not indicated unless patient wishes to become pregnant
1. WEIGHT – LOSS ASSOCIATED AMENORRHOEA

A loss of > 10 kg is frequently associated with amenorrhoea

i. In young women and teen ages girls become obsessed with their body image and starve themselves.

ii. Jogger’s amenorrhoea:
   - This is seen frequently in women training for marathon racing, in ballet dancers and other form of athletes.
CAUSES:

+ redistribution between proportion of body fat mass and body muscle mass.

May be also mediated by exercise related changes in β-endorphins.

iii. ANOREXIA NERVOSA
Associated with sec. amenorrhoea
(misnomer no loss of appetite)
2. **AMENORRHOEA AND ANOSMIA:**

rare cause of amenorrhoea of hypogonadotrophic – hypo-gonadism.

(Counterpart in males is Kallman’s syndrome)

**POST-PILL AMENORRHOEA:**

♣ There is no evidence that Est. prog. Contraceptive pills predispose to amenorrhoea.. once pill taking is ceased.

♣
An irregular menstrual cycle frequently precedes pill taking.

♣ If this assumption of amenorrhea being merely an after-effect of pill taking → many cases of hyperprolactinemia will be missed (1:5)
And Premat. ovarian failure will be missed in 1:10 cases

Once other causes are excluded, this type of ameno. Responds well to ovulation induction with Clomiphene citrate if preg. is desired.
INVESTIGATION OF AMENORRHOEA

1. S. Prolactin level and TFT

2. Karyotyping…if chrom. anomaly is suspected on clinical grounds

3. Progesterone withdrawal test….to check endog. estrogen.
   e.g. Provera (medroxy-prog) → if bleeding PV=reactive endom. and patent outflow tract.
• If PRL is norm. + no galactorrhoea ---no need for further investigation for pituitary tumor
• If GALACTOR is present→ further evaluation of pit. gland is necessary .. regardless of level of PRL and menstrual pattern
• If PRL is signific. elevated (excluding stress) → Radiology exam of pituitary to exclude tum.
• Visual fields assessment – if X-Ray abnormal
• **FSH & LH** level… especially if no withdrawal bleeding following prog. Challenge.

• ↓ LH (<5 IU/ml) → hypogonadotrophic-hypogonadism

• ↑ FSH (>40 IU/ml) on successive readings → ovarian failure

If women < 35 years = premet. ovar. failure (menopause) → check **karyotype**. (if Y-Chrom + → high risk of gonadal malignancy)
4. **USS:**

Of uterus and ovaries can be useful to investigating and monitor Rx. Of these women
Patient presenting with secondary amenorrhea; negative pregnancy test

Check TSH and prolactin levels.

Both normal

Normal prolactin level, abnormal TSH level

Normal TSH level, abnormal prolactin level

Thyroid disease

Prolactin ≤ 100 ng per mL (100 mcg per L)

Consider other causes (Table 4).

Prolactin > 100 ng per mL

Perform MRI to evaluate for prolactinoma.

Negative MRI

Consider other causes (Table 4).

Progestogen challenge test (Table 3)

Withdrawal bleed

No withdrawal bleed

Normogonadotropic hypogonadism (Table 4)

Estrogen/progestogen challenge test (Table 3)

Withdrawal bleed

No withdrawal bleed

Check FSH and LH levels.

Outflow obstruction

FSH > 20 IU per L and LH > 40 IU per L

Hypergonadotropic hypogonadism (Table 4)

FSH and LH < 5 IU per L

Perform MRI to evaluate for pituitary tumor.

Normal MRI: hypogonadotropic hypogonadism (Table 4)
FLOW CHAR FOR INVESTIGATING OF SEC. AMENORRHOEA

Complete History
Full Ph. exam., tubal patency + sperm count

Amen. Traumatica

Proof by Hystero gram

 ↑ FSH, LH

Low or N LH, FSH

Clomid

Failed Response

Hyperprolac.

Abn. Fossa

FSH, LH, TSH, P RL, X-Ray of Pit. Fossa

TSH ↑

Tomograms

Thyro xin

Tumor

? Premat Menopause

Biopsy

 Fail ed Response

No Tum

MPS

Srug. & OR ext. radioth.

Repeat MPS

HPG or HMG

Bromocriptine