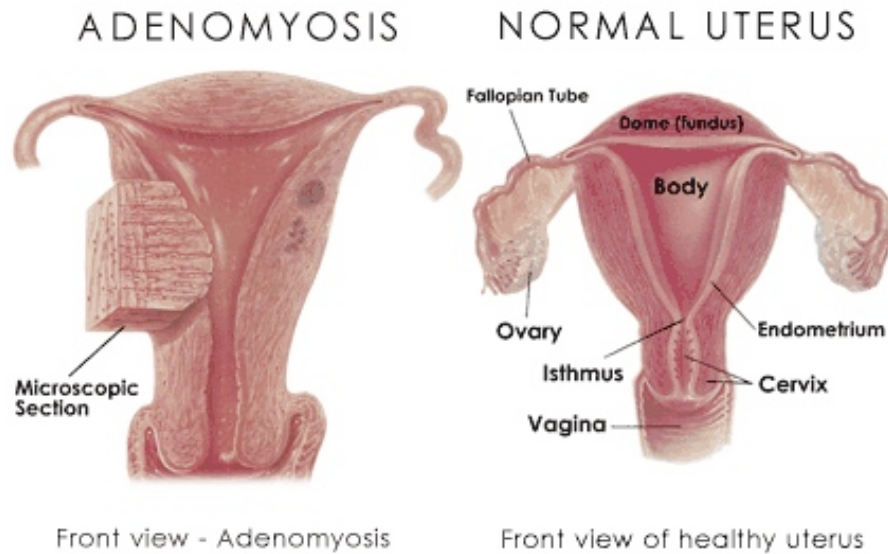


429 PATHOLOGY TEAM



DONE BY:

ALBATOOL ALAMMARI

AMNA ALBALJOUN

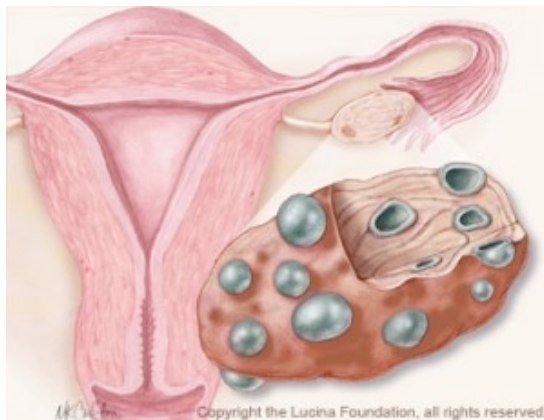
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POLYCYSTIC OVARIAN DISEASE (PCOD)

- Also known as polycystic ovarian syndrome and Stein-Leventhal syndrome.

* What is PCOD?

Bilateral enlargement of polycystic ovaries, **accompanied by** chronic anovulation and clinical manifestations secondary to excessive production of estrogens and androgens, mainly androgens.

* Description of PCOD:

In women without polycystic ovarian syndrome, the ovaries begin to develop 20 eggs every month. These eggs mature in little sacs known as cysts. Over the course of the month, one egg will become dominant and draw most of the hormones being produced, eventually being released by the ovary to be fertilized or shed with your period. Women with PCOS, though, fail to produce the correct balance of estrogen necessary to help one egg become dominant. As a result, the 20 eggs develop but remain as cysts, which in turn results in the production of androgens, or male hormones, and little to no production of progesterone.

Because of the build up of androgens and lack of progesterone, women with PCOS may have irregular periods, fail to ovulate (anovulation), or fail to have a period (amenorrhea) entirely. When a period does occur, many women with PCOS note that their bleeding can be quite heavy.

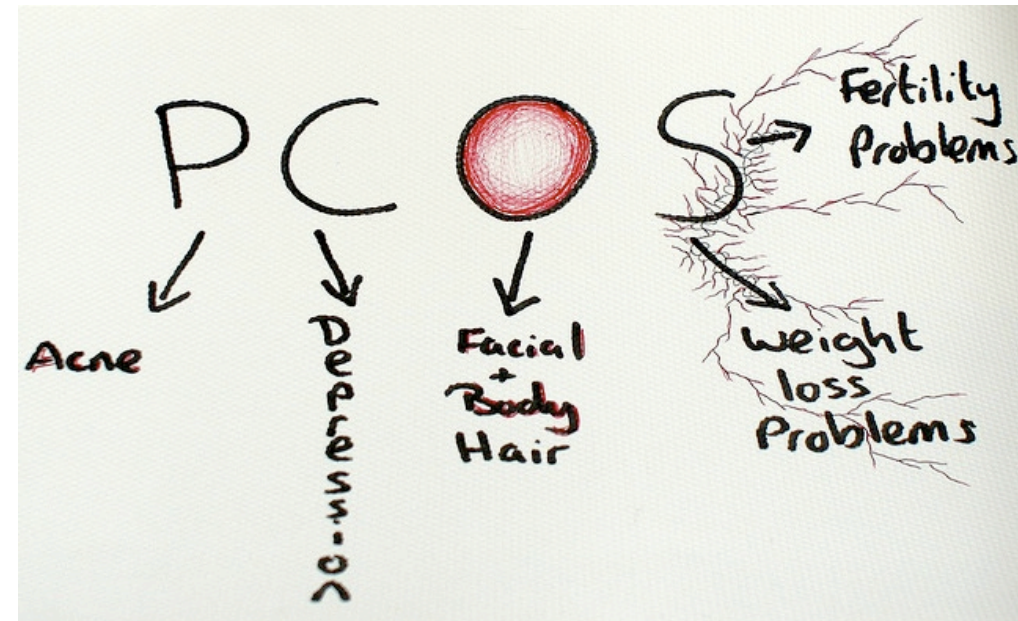
* What is the pathophysiology of PCOD?

1- The initial abnormality resulting in the syndrome is not known but is believed to be related to hypothalamus-pituitary dysfunction leading to oversecretion of luteinizing hormone (LH).

2- LH in turn stimulates the ovary to produce excess androgens. Secretion of follicle stimulating hormone (FSH) is inhibited resulting in repression of ovulation with follicle cyst formation.

* What are the characteristics of PCOD?

- High level of LH and low FSH
- Bilaterally enlarged polycystic ovaries
- Chronic anovulation



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Risk Factors	Clinical Appearance	Grossly	Histology	Clinical behavior
<p>ALL ARE IMPORTANT</p> <ol style="list-style-type: none"> 1. Endometrial hyperplasia and endometrial cancer 2. Insulin resistance/ Type II diabetes. 3. High blood pressure 4. Depression/ Anxiety 5. Dyslipidemia 6. Cardiovascular disease 7. Strokes 8. Weight gain 9. Miscarriage 10. Acanthosis nigricans (patches of darkened skin under the arms, in the groin area, on the back of the neck) 11. Autoimmune thyroiditis 	<p>The usual clinical presentation is a young woman (between 15 and 30 years) with:</p> <ol style="list-style-type: none"> 1. Secondary amenorrhea with anovulation (Due to lack of progesterone) 2. Oligomenorrhea* or irregular menses (Due to lack of progesterone) 3. Infertility (Because they experience infrequent ovulation or lack of ovulation) 4. Hirsutism (High production of androgens "male hormones") 5. Virilism due to excessive amounts or effects of androgenic (masculinizing) hormones 6. Obese (Both high androgen levels and insulin intolerance*) 7. Acne (High levels of DHT) 	<p>Both ovaries are:</p> <ol style="list-style-type: none"> 1. Markedly enlarged usually twice normal in size 2. Have a thickened, fibrotic gray white capsule <u>with</u>: <ol style="list-style-type: none"> a. Smooth pearl-white outer surface, b. Studded with subcortical cysts 0.5 to 1.5 cm in diameter. 	<p>Microscopically:</p> <ul style="list-style-type: none"> - The outer portion of the cortex is thickened and fibrotic - Many follicle cysts lined by granulosa cells are present in the subcapsular cortex. The cysts have prominent outer theca interna layer, which is often luteinized. - Corpora lutea are frequently absent due to the anovulation and occasional focal stromal luteinization is present. - Cortical stromal fibrosis - The chronic anovulation results in unopposed estrogenic stimulation of the endometrium leading to a variety of appearances ranging from mild atypical hyperplasia to well-differentiated endometrial adenocarcinoma 	<ol style="list-style-type: none"> a. Treatment with drugs that either induce ovulation (clomiphene or hCG) or regulate the menstrual cycle restores fertility. b. Reduction of ovarian volume by wedge resection: (Removing a section of the tissue that secretes the hormone produces a sustained reduction in androgen and estrogen levels) is also successful in initiating ovulation and restoring fertility. c. The endometrial changes usually regress once ovulation is achieved.

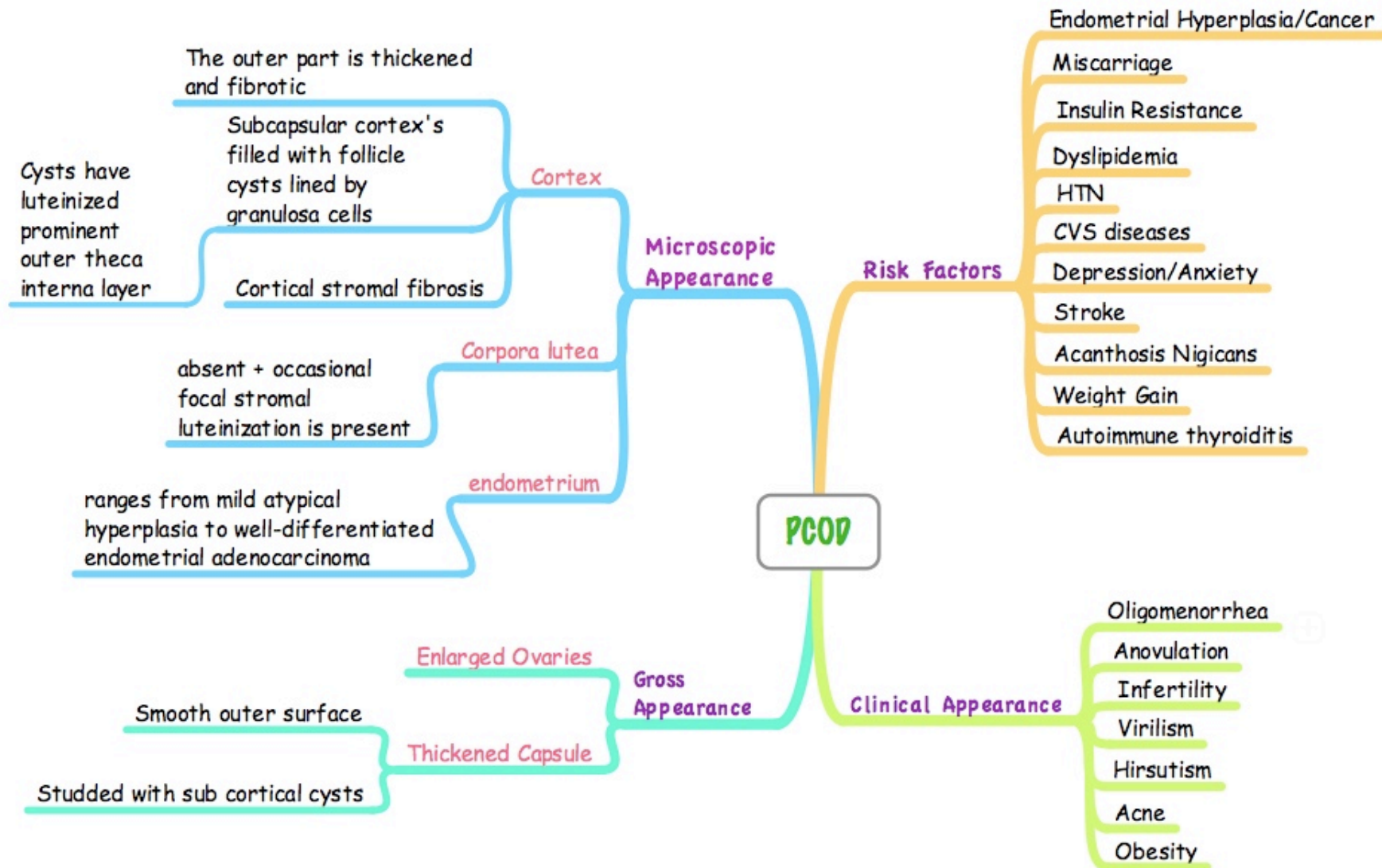
***Oligomenorrhea**: it is menstrual periods occurring at intervals of greater than 35 days, with only four to nine periods in a year.

***Virilism**: the presence of male secondary sexual characteristics in a female

*High androgen levels can make it difficult to lose weight as can insulin resistance. And insulin resistance contributes to obesity by promoting fat storage.

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***Insulin Resistance** vastly reduces the insulin sensitivity of cells, which impairs the processing of glucose through the cell wall for conversion to energy. As a result, glucose remains in the blood stream, causing elevated levels of blood sugar, which are sent to the liver. Once there, the sugar is converted into fat and stored via the blood stream throughout the body. This process can lead to weight gain and obesity, key factors in creating PCOS, which is also referred to as Polycystic Ovarian Disease or PCOD.



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ENDOMETRIOSIS

About	Clinical Appearance	Morphology	Prognosis
<p>Definition: Presence of ectopic endometrial glands and stroma outside the uterus</p> <p>Responsive to: the hormonal variations of the menstrual cycle (just like Endometrial Hyperplasia)</p> <p>Location: the peritoneal surfaces of the reproductive organs and adjacent pelvic organs as: A) Most frequent location: the ovary (approx. 50%) → pouch of Douglas → uterine ligaments B) Occasional sites: the cervix, vagina, perineum, bladder, large bowel and the umbilicus C) Rare lesions: Reaching small bowel, kidneys, lungs and brain.</p> <p>Characterized by: Menstrual Type Bleeding at the site of the ectopic endometrium, resulting in blood filled areas (e.g. chocolate cysts). It has been reported in men. The sites involved have been the bladder, scrotum and prostate.</p>	<p>Depends on the site</p> <ol style="list-style-type: none"> 1) Dysmenorrhea 2) Dyspareunia 3) Results in Infertility 4) Produces cysts <p>* Dysmenorrhea: painful menses * Dyspareunia: painful sexual intercourse</p>	<p>Cysts is produced by: hemorrhage into the foci of the ovary with each menstrual cycle (menstrual-type bleeding) → produce chocolate cysts (contains chocolate-brown material)</p> <p>Gross: – Appears as multiple red/brown cysts (due to hemosiderin) – Dense fibrous adhesions may surround the foci</p> <p>Histology: – Presence of ectopic endometrial glands and stroma (diagnosis is made by microscope) – Macrophages containing hemosiderin (siderophages) may be present in lesions with previous hemorrhage. – When endometriosis develops in a muscular viscus the smooth muscle around it is often hyperplastic.</p>	<p>• Non-neoplastic</p> <p>(Benign with no malignant potential)</p> <p>• May recur after surgical excision but the risk is low</p>

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ADENOMYOSIS

About	Clinical Appearance	Morphology	Prognosis
<p>Definition: Presence of endometrial glands and stroma in the myometrium. a disorder of parous women and occurs infrequently in the nullipara</p> <p>Location: It usually involves posterior wall more than the anterior wall (or both).</p>	<ol style="list-style-type: none"> 1) Menorrhagia 2) Severe dysmenorrhea 3) 1/3rd are asymptomatic (with lesions discovered accidentally) <p>Menorrhagia: severe menstrual bleeding</p>	<p>When extensive: the lesion produce: myometrial thickening + small yellow/ brown cystic spaces containing fluid or blood</p> <p>Adenomyoma: A tumor produced by the proliferation of smooth muscle around a focus of adenomyosis (resembles uterine leiomyoma)</p>	<p>A benign condition with no known malignant potential</p> <p>Regresses after the menopause.</p>

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