SCLEROPOLYKYSTIC OVARIAN DISEASE

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Annotation: *Scleropolykystic ovarian disease (SPOD), more commonly referred to as polycystic ovary syndrome with stromal sclerosis, is a complex endocrine and metabolic disorder affecting women of reproductive age. The condition is characterized by ovarian morphological changes, hyperandrogenism, ovulatory dysfunction, and metabolic disturbances. SPOD represents a more advanced morphological form of polycystic ovary syndrome (PCOS), distinguished by dense ovarian stroma and fibrotic changes that impair follicular maturation. This article analyzes the pathophysiology, clinical manifestations, diagnostic criteria, and current management approaches of scleropolykystic ovarian disease, emphasizing its reproductive and metabolic consequences. Based on literature analysis, the paper highlights the importance of early diagnosis and multidisciplinary management to improve fertility outcomes and reduce long-term health risks.*

Keywords: *Scleropolykystic ovarian disease; polycystic ovary syndrome; ovarian sclerosis; hyperandrogenism; anovulation; infertility; endocrine disorders.*

Scleropolykystic ovarian disease is a chronic gynecological and endocrinological condition that occupies a significant place among causes of female infertility and menstrual disorders. It is closely related to polycystic ovary syndrome but is distinguished by more pronounced structural changes in the ovarian tissue, particularly stromal hyperplasia and sclerosis. These morphological alterations lead to persistent anovulation and hormonal imbalance.

The disease typically manifests during adolescence or early reproductive years and may progress silently for long periods. Women with SPOD often present with irregular menstruation, infertility, hirsutism, acne, obesity, and metabolic abnormalities such as insulin resistance. Due to its multisystem nature, SPOD is not only a reproductive disorder but also a condition associated with long-term cardiovascular and metabolic risks.

Understanding the pathogenesis and clinical features of scleropolykystic ovarian disease is essential for developing effective diagnostic and therapeutic strategies. This article aims to provide a comprehensive overview of the disease based on current scientific literature.

Scleropolycystic ovarian disease, also historically referred to as sclerocystic ovaries or Stein-Leventhal syndrome, is an outdated term for what is now known as Polycystic Ovary Syndrome (PCOS). The "sclero-" prefix referred to the observed thickening or fibrosis of the ovarian capsule in early descriptions, while "polycystic" described the appearance of multiple small follicular structures. Today, PCOS is recognized as a complex endocrine disorder rather than a disease primarily defined by ovarian cysts.

PCOS is one of the most common hormonal conditions in women of reproductive age, affecting approximately 8-13% globally, with higher rates in certain populations. It is characterized by hormonal imbalances leading to a range of reproductive, metabolic, and psychological effects.

Historical context: The condition was first described in 1935 by Irving Stein and Michael Leventhal, who noted enlarged ovaries with a thickened capsule and multiple cysts in women with irregular periods, infertility, and hirsutism (excess hair growth). Surgical wedge resection of the ovaries was once a treatment. Over time, understanding evolved: the "cysts" are actually immature follicles arrested in development, and the thickened capsule is due to collagen deposition from chronic hormonal stimulation.

Pathophysiology: In a normal menstrual cycle, follicles in the ovaries mature, one dominant follicle releases an egg (ovulation), and the rest regress. In PCOS:

- Elevated luteinizing hormone (LH) relative to follicle-stimulating hormone (FSH) drives excessive androgen production by ovarian theca cells.

- High androgens disrupt follicular maturation, leading to accumulation of small antral follicles (2-9 mm).

- Insulin resistance (present in 50-70% of cases, even in lean women) exacerbates androgen production and inhibits ovulation.

- The ovaries become enlarged (often 1.5-3 times normal size) with a thickened, pearly-white outer capsule.

Gross appearance (if surgically viewed): The ovaries are bilaterally enlarged, smooth, and opaque white, with a bosselated surface due to subcortical follicles.

Microscopically: Numerous small follicular cysts lined by granulosa cells, hyperplastic theca cells, and stromal hyperplasia.

Here are ultrasound images showing typical PCOS ovarian morphology:

For comparison, here is an illustrative diagram of normal vs. polycystic ovaries:
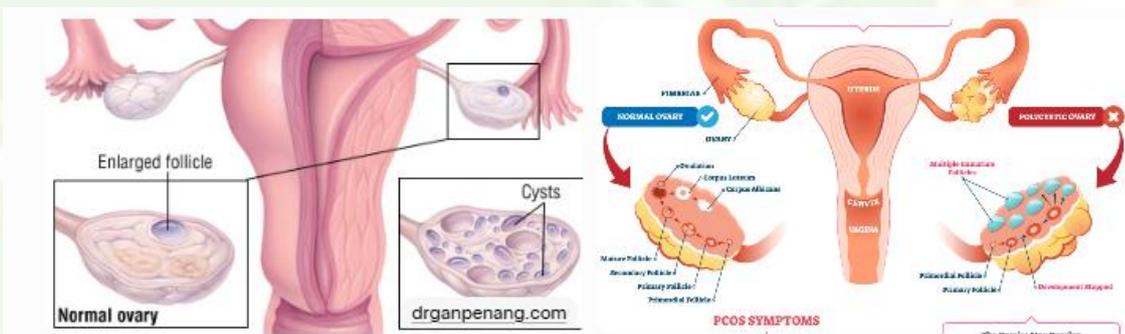
Diagnostic Criteria (Rotterdam Consensus, 2003 - most widely used)

Diagnosis requires at least 2 of the following 3 features, after excluding other disorders (e.g., congenital adrenal hyperplasia, androgen-secreting tumors, Cushing's syndrome):

1. Oligo- or anovulation: Infrequent or absent ovulation, leading to irregular menstrual cycles (fewer than 9 periods/year or cycles >35 days).

2. Clinical or biochemical hyperandrogenism: Excess androgens causing hirsutism, acne, or elevated blood levels (e.g., free testosterone, androstenedione).

3. Polycystic ovarian morphology on ultrasound: ≥20 follicles (2-9 mm) per ovary or ovarian volume >10 mL (updated criteria in some guidelines).

Note: Not all women with PCOS have polycystic ovaries on ultrasound (about 20-30% do not), and some women without PCOS may have this appearance.

Common Symptoms

Symptoms vary widely and may start around puberty:

- Menstrual irregularities (most common)

- Infertility due to lack of ovulation

- Hirsutism (excess hair on face, chest, abdomen)

- Acne and oily skin

- Scalp hair thinning (androgenic alopecia)

- Weight gain or obesity (though 20-50% are lean)

- Skin tags or acanthosis nigricans (dark velvety patches, sign of insulin resistance)

- Fatigue, mood changes, anxiety/depression

- Sleep disturbances

Detailed infographics summarizing common PCOS symptoms:

Causes and Risk Factors

- Genetic: Strong heritability (twin studies show 70% concordance); multiple genes involved in androgen synthesis, insulin signaling.

- Environmental: Fetal exposure to androgens, low birth weight, rapid postnatal weight gain.

- Insulin resistance: Amplifies ovarian androgen production.

- No single cause; it's a syndrome with heterogeneous presentations.

 Long-Term Health Risks

- Infertility

- Type 2 diabetes (3-7x increased risk)

- Cardiovascular disease (due to dyslipidemia, hypertension)

- Endometrial cancer (from chronic unopposed estrogen)

- Non-alcoholic fatty liver disease

- Mental health issues

 Management and Treatment

No cure, but symptoms are highly manageable:

- Lifestyle: First-line – weight loss (5-10% reduces symptoms dramatically), low-glycemic diet, exercise.

- Hormonal regulation: Combined oral contraceptives to regulate cycles and reduce androgens.

- Anti-androgens: Spironolactone for hirsutism/acne.

- Insulin sensitizers: Metformin.

- Fertility: Clomiphene or letrozole for ovulation induction; IVF if needed.

- Monitoring: Regular checks for metabolic complications.

PCOS is a lifelong condition, but with proper management, most women lead healthy lives and achieve pregnancy if desired. Consult a specialist (endocrinologist or gynecologist) for personalized advice, including blood tests and ultrasound.

Conclusion

Scleropolykystic ovarian disease is a complex endocrine-metabolic disorder with significant reproductive and systemic implications. Its defining features include

ovarian sclerosis, hyperandrogenism, chronic anovulation, and metabolic disturbances. Timely recognition of the disease is crucial to prevent infertility and long-term complications.

It is recommended that clinicians adopt a multidisciplinary approach focusing on hormonal regulation, metabolic control, and individualized fertility management. Lifestyle interventions should be considered a foundational component of therapy. Future studies should aim to clarify the molecular mechanisms of ovarian sclerosis and evaluate innovative treatment strategies to improve patient outcomes.

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