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PCOS (Polycystic Ovarian Syndrome)

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Polycystic Ovarian Syndrome (PCOS): A Comprehensive Review of Pathophysiology, Diagnosis, and Management

Introduction

Polycystic ovary syndrome (PCOS) is a multifaceted endocrine disorder that affects 6 to 20% of women reproductive globally, characterized by hyperandrogenism, ovulatory dysfunction and polycystic ovarian morphology [ref_1]. Despite its prevalence, PCO remains underdiagnosed due to heterogeneous clinical presentations and evolutionary diagnostic criteria [ref_2]. This review synthesizes current evidence on PCOS etiology, diagnostic challenges and evidence -based management strategies to guide doctors and researchers.

Pathophysiology and Etiology

PCO arises from a complex interaction of genetic, metabolic and environmental factors. Insulin resistance and hyperinsulinemia are central to their pathogenesis, exacerbating ovarian androgen production and interrupting folliclegenesis [ref_3]. Association studies throughout the genome (GWAS) identified susceptibility locations, including Dennd1a and FSHR, linking SOP to altered steroidogenesis and gonadotropin signs [ref_4]. Adipose tissue dysfunction and low -grade chronic inflammation still contribute to metabolic complications such as type 2 diabetes and dyslipidemia [ref_5].

Clinical Manifestations

PCO has a symptom spectrum, including hirsutism, acne, oligomenorrhea and infertility. Hyperandrogenism, a striking feature, correlates with metabolic risks, while anovulation increases susceptibility to endometrial cancer [ref_6]. Psychological comorbidities, such as depression and anxiety, are predominant, emphasizing the need for holistic care [ref_7]. Notably, phenotypic variability - as a lean versus obesity - highlights the importance of individualized evaluation [ref_8].

Diagnostic Criteria

The criteria of Rotterdam (2003) remain the most widely used diagnostic structure, requiring two of the three characteristics: hyperandrogenism, oligo-/anovulation or polycystic ovaries in ultrasound [ref_9]. However, controversies persist, particularly regarding the usefulness of ovarian morphology in adolescents and overlapping with non -classical adrenal hyperplasia [ref_10]. The anti-müllerian hormone (AMH) emerged as a potential biomarker for ovarian dysfunction, although standardization lacks [Ref_11].

Management Strategies

Lifestyle Modifications

First -rate therapy for overweight/obese women includes weight loss through food changes and exercise, which improves insulin sensitivity and restores ovulation [Ref_12]. Even a 5 to 10% reduction in body weight significantly reduces androgen levels and menstrual irregularity [ref_13].

Pharmacological Interventions

- **Metformin**: Improves insulin sensitivity and reduces hyperandrogenism, particularly in women with glucose intolerance [Ref_14].
- **Oral Contraceptives (OCPS):** suppress androgens' production and regulate cycles, but may worsen metabolic profiles in obese patients [Ref_15].

- **Anti-aircing (eg spironolactone)**: effective for hirsutism, but require contraception due to teratogenicity [ref_16].

- **-Inositois**: The combinations of myio-inositol and d-chiro-ositol improve ovarian parameters and metabolic parameters with minimal side effects [ref_17].

Surgical Options

Laparoscopic ovarian perforation (LOD) is reserved for clomiphene resistant infertility, offering efficacy comparable to gonadotropins, but with greater risk of multiple pregnancy [Ref_18].

Future Directions

Emerging therapies, such as GLP-1 agonists and CRISPR-based gene edition, promise to be directed to insulin resistance and genetic defects [Ref_19]. Personalized medicine, leveraging polygenic risk scores and metabolomic profile, can revolutionize PCO management [Ref_20].

Conclusion

SOP requires a multidisciplinary approach, addressing reproductive, metabolic and psychological dimensions. Early diagnosis and personalized interventions are critical to mitigate long -term complications. Continuous research on molecular mechanisms and innovative therapies will increase patient results.

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