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PREFACE

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Chapter 1

FROM DIAGNOSIS TO TREATMENT: POLYCYSTIC OVARY SYNDROME

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POLYCYSTIC OVARY SYNDROME: A COMPREHENSIVE EXPLORATION

Polycystic ovary syndrome is the most common endocrine disorder affecting reproductive-aged women, marked by a complex and diverse range of symptoms. (1) The heterogeneous nature of PCOS has led to the development of various diagnostic criteria over the years, each with its own unique components and considerations. (1) Despite the wealth of research in this area, the underlying mechanisms of PCOS remain only partially understood, with insulin resistance and hormonal imbalances playing a central role in the pathophysiology of the condition. (2)

The current guidelines for diagnosing PCOS emphasize the importance of carefully evaluating a range of factors, including clinical and biochemical evidence of hyperandrogenism, menstrual irregularities, and the presence of polycystic ovarian morphology on imaging studies. (1) Accurate diagnosis is crucial, as PCOS is associated with a significant burden of both physical and psychological consequences, including infertility, metabolic disorders, and increased risk of cardiovascular disease. (3) (4)

Once a diagnosis of PCOS is established, a comprehensive management approach is essential to address the multifaceted nature of the condition. Treatment strategies often involve a combination of lifestyle modifications, such as weight management and exercise, as well as targeted pharmacological interventions to address specific symptoms and comorbidities.

Epidemiology and Prevalence

Polycystic ovary syndrome is the most common endocrine disorder in women of reproductive age, with a prevalence from 6% to 20% (4). The wide variation in

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Metformin has been shown to improve insulin sensitivity, reduce hyperandrogenism, and restore ovulatory function in PCOS patients. (13) (19)

Additionally, metformin may help reduce the risk of developing type 2 diabetes mellitus and other metabolic complications associated with PCOS. (20)

Other insulin-sensitizing agents, such as thiazolidinediones (e.g., pioglitazone), have also been explored in the treatment of PCOS, with promising results in improving metabolic and reproductive outcomes.

Combination Therapy Approaches

The management of polycystic ovary syndrome often requires a combination of various therapeutic strategies to address the multifaceted nature of the condition.

Combining lifestyle modifications, such as weight loss and exercise, with pharmacological interventions, such as insulin-sensitizing agents and anti-androgen therapies, has been shown to be more effective in improving the clinical and metabolic outcomes in PCOS patients.

The combination of metformin and oral contraceptive pills, for instance, has been found to be more effective in reducing hyperandrogenic symptoms and restoring ovulatory function compared to either therapy alone.

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Chapter 2

CYTOMEGALOVIRUS (CMV) INFECTION AND INTRAUTERINE ULTRASONOGRAPHY FINDINGS

Huriye EZVECİ[1]

INTRODUCTION

Cytomegalovirus (CMV) is a prevalent DNA virus classified under the herpesvirus family. It becomes latent following the primary infection but can reactivate and result in viral shedding. CMV is the most prevalent congenital viral infection globally, with an incidence of roughly 0.67% at birth (1). [1]. Approximately 90% of neonates with congenital CMV infection are asymptomatic at birth, whereas 10% exhibit symptoms. Observations in symptomatic newborns may include petechiae, jaundice, hepatosplenomegaly, and microcephaly.Both symptomatic and asymptomatic neonates face the risk of unfavorable outcomes, with symptomatic newborns being at a greater risk (2). Sensorineural hearing loss is the most prevalent consequence.

Maternal CMV infection can be primary (in individuals who have not been exposed to CMV before) or non-primary (reactivation or re-infection with a different strain in individuals who have been previously infected) (3). Primary infection is usually asymptomatic, but symptoms such as fever, malaise, and lymphadenopathy may be observed, with a 40% chance of transmission to the baby. CMV is transmitted through contact with body fluids and can be transmitted to the fetus via the placenta. CMV seroprevalence is 86% in women of childbearing age worldwide (4).

CMV infection is identified using serological assays (IgG, IgM, IgG avidity) (5). Primary infection is identified through seroconversion, indicated by the transition of IgG from negative to positive. The likelihood of fetal infection escalates in the later stages of pregnancy, although the probability of clinical consequences diminishes (6). The likelihood of fetal infection is minimal in seropositive pregnant women; however, this risk may escalate in pregnant women

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Chapter 3

METABOLIC SYNDROME WITH POLYCYSTIC OVARY SYNDROME

Duygu LAFCI[1]
Akın USTA[2]

Introduction

Metabolic syndrome, which centers on obesity, hypertension, insulin resistance, and dyslipidemia, refers to a combination of factors that significantly increase the likelihood of experiencing serious health conditions, such as heart disease, stroke, and type 2 diabetes (1). Among these factors, insulin resistance plays a central role, often serving as the underlying mechanism for the development and progression of the syndrome. Understanding the multifaceted nature of metabolic syndrome involves not only examining cardiovascular risks but also considering the endocrinological aspects, particularly the significant role insulin resistance (IR) plays in increasing the likelihood of long-term health complications (2).

Polycystic ovary syndrome (PCOS) is a common endocrine disorders, affecting around 11-13% of women globally (1,2). Known by various names, such as Stein-Leventhal syndrome or hyperandrogenic anovulation (HA), PCOS is primarily characterized by a combination of hyperandrogenism, irregular ovulation, and polycystic ovarian morphology. This disorder is associated with a range of long-term consequences, including infertility, cardiovascular disease (CVD), metabolic disturbances, obstructive sleep apnea, psychological issues, and pregnancy complications like preeclampsia and gestational diabetes. PCOS also puts women at greater risk for endometrial cancer and non-alcoholic fatty liver disease (3).

IR is a fundamental factor in PCOS and a key driver of metabolic syndrome development. Consequently, up to 33% of women with PCOS may develop metabolic syndrome

(2). While, PCOS's focus has been on addressing concerns such as infertility, anovulation, and hirsutism, this connection between PCOS and metabolic syndrome is indeed a significant consideration in women's health (1).

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(50,51). Several cross-sectional studies have found that women with PCOS have significantly higher blood pressure compared to healthy controls, regardless of weight or obesity (42,52).

The findings underscore the complex and interconnected nature of metabolic disturbances in PCOS, with androgen excess playing a pivotal role. Addressing these metabolic symptoms is crucial for managing the long-term health risks associated with PCOS, including cardiovascular complications. Lifestyle modifications, such as dietary changes and physical activity, are often recommended as part of the management strategy for women with PCOS to mitigate these metabolic challenges. Additionally, early detection and intervention can be key in preventing or managing the development of metabolic syndrome in women with PCOS.

CONCLUSIONS

Metabolic syndrome is often under-recognized, despite its high prevalence in women with PCOS. The pathophysiology of PCOS is intricate and varies across individuals, making it a challenging condition to diagnose. Although it primarily affects women of reproductive age, PCOS presents diagnostic challenges due to its diverse manifestations and ongoing uncertainties about its causes. It is increasingly acknowledged as a major risk factor for metabolic disorders. Women with PCOS commonly experience hyperandrogenism and insulin resistance, which contribute to both reproductive issues and metabolic dysfunction, creating a cycle that can lead to significant long-term health complications.

This information emphasizes that PCOS has significant effects not only on reproductive but also on metabolic health. Early diagnosis and effective management of PCOS can improve the quality of life of these women and positively influence their long-term health outcomes.

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Chapter 4

TOXOPLASMOSIS INFECTION DURING PREGNANCY

Melike Sevde HARMANCI[1]

INTRODUCTION

Toxoplasma gondii is an obligate intracellular parasite belonging to the phylum Apicomplexa, subclass coccidia. It can take several different forms: oocyst; tachyzoite and cyst. The T gondii genome is haploid, except in cats where sexual division occurs, and contains approximately 8×107 base pairs.(3)

Sources of transmission of Toxoplasma gondii;

- Raw, undercooked or smoked meat or meat products
- Soil or water contaminated with cat feces
- Unpasteurized milk
- •Seafood obtained from contaminated water
- Infected organ transplantation or blood transfusion
- Vertical transmission during pregnancy

There is no evidence that T gondii is transmitted through breastfeeding or direct human-to-human contact. (3) Although infection in healthy nonpregnant women is self-limited and largely asymptomatic, the primary concern is the risk to the fetus through vertical transmission during pregnancy. Congenital toxoplasmosis can cause permanent neurological damage and even serious morbidity, such as blindness. (4) Most (more than 80%) Toxoplasma gondii infections are asymptomatic. Symptomatic patients usually present with mild and nonspecific symptoms such as fever, chills, sweating, headache, myalgia, pharyngitis, lymphadenopathy, hepatosplenomegaly, and generalized maculopapular rash. The febrile attack lasts approximately 2-3 days. Lymphadenopathy is the most common symptom and may last for several weeks. It is typically cervical, bilateral, symmetrical, and nonfluctuating. Rarely, generalized lymphadenopathy may occur. In severe cases, especially those resulting from reinfection, chorioretinitis may be accompanied by floaters and even vision loss.(4) When maternal infection

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avoided. However, if avoidance is not possible, disposable gloves should be worn when doing gardening and when in contact with soil or sand, and hands should be washed with soap and warm water afterwards.(5) It is also important to avoid undercooked, raw or cured meats and raw, unwashed fruits and vegetables. (23) Avoid drinking untreated water, including water from wells, or water that may be contaminated with feces from domestic or feral cats.(5) Proper hand hygiene is essential to reduce the risk of infection.(24)

SUMMARY

Congenital toxoplasmosis is one of the common chronic infections. Screening programs are implemented in various countries depending on the prevalence and virulence of the parasite in the relevant regions.

Diagnosis is confirmed by Toxoplasma DNA PCR at amniocentesis.

Early diagnosis and appropriate antibiotics can reduce the risk of fetal infection.

Primary prevention remains the main intervention to prevent infection and therefore patient education is an important aspect of management.

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Chapter 5

RHD ALLOIMMUNIZATION IN PREGNANCY

Refaettin SAHIN[1]

RHD-negative Women who give birth to a RHD-positive infant or are exposed to RHD-positive blood acquire anti-D antibodies. RHD-positive infants born to these moms may develop hemolytic illness of the newborn. Neonatal hemolytic illness can be effectively managed with proper monitoring and management.

The execution of prenatal and postnatal anti-D immune globulin prophylactic programs has resulted in a notable decrease in the occurrence of RhD alloimmunization and related fetal problems. (1)

The Rh blood group system has around 50 antigens. The predominant antigens that provoke antibody production are D, C, c, E, and e. An RhD-negative mother exposed to fetal red cells with paternal C, c, E, and/or e antigens may produce anti-C, -c, -E, and/or -e antibodies. Consequently, although a RhD-negative mother may get prophylactic anti-D immunoglobulin, this does not avert alloimmunization to alternative Rh antigens (c, C, E, e). The frequency of RhD-negative blood types varies considerably among diverse populations. The highest prevalence is observed among Basques, ranging from 30% to 35%. The prevalence among white North Americans and Europeans is 15%. Other populations exhibit reduced proportions of RhD-negative people.(2)

The RhD antigen is present on the red blood cell (RBC) membrane starting from day 38 of gestation and, in contrast to other antigens (A, B, M, N), is exclusively located on red blood cells. (3) Maternal RhD alloimmunization occurs when the maternal immune system encounters RhD-positive erythrocytes. Assume that anti-D IgG antibodies exist in the maternal circulation. In such instances, they can traverse the placenta and affect fetal red blood cells, resulting in phagocytosis by acrophages and potentially inducing fetal anemia. Transplacental fetomaternal bleeding constitutes the predominant cause of maternal RhD alloimmunization. Research indicates that a minor quantity of fetal red blood cells inadvertently enters the maternal circulation in the majority of pregnancies. The volume and frequency of fetomaternal bleeding escalate as pregnancy advances, peaking in the third

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should be performed for the diagnosis of anemia. If anemia is detected after cordocentesis, intrauterine transfusion should be performed. Regular ultrasonic monitoring during pregnancy and a multidisciplinary approach are crucial for the implementation of all these treatment options. In this way, potential health risks related to RHD alloimmunization can be minimized, and healthy pregnancy processes can be ensured.

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Chapter 6

ECTOPIC PREGNANCY

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1. INTRODUCTION

Ectopic pregnancy is a pathological condition characterized by the implantation of a fertilized ovum outside the uterine cavity, predominantly in the fallopian tubes but also in other extragenital sites such as the abdominal cavity, cervix, or ovaries. This condition constitutes a major cause of maternal morbidity and mortality, necessitating early recognition and intervention to mitigate its potentially fatal complications. Despite advances in imaging techniques and biomarker assessment, ectopic pregnancy continues to challenge clinicians due to its variable presentation and the risk of misdiagnosis(1).

2. HISTORICAL BACKGROUND AND EPIDEMIOLOGY

The first documented cases of ectopic pregnancy date back to the 16th century, but it was not until the 19th century that surgical management became a viable treatment approach. Historically, maternal mortality associated with ectopic pregnancy was exceedingly high due to delayed diagnosis and lack of effective interventions. The advent of ultrasonography and serum beta-hCG measurement significantly improved early detection, while the introduction of methotrexate therapy in the 1980s revolutionized non-surgical management options.

Globally, the incidence of ectopic pregnancy is estimated at 1-2% of all pregnancies, increasing to 2-5% in assisted reproductive technology (ART) users. Risk stratification has revealed that prior pelvic infections, tubal pathology, and certain contraceptive methods significantly predispose individuals to ectopic implantation. Alarmingly, 6% of all maternal deaths related to pregnancy complications can be attributed to ectopic pregnancy(2, 3).

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7.4. Complication Management and Follow-Up

Timely intervention is critical for tubal rupture, hemorrhage, or incomplete β-hCG resolution. Weekly β-hCG monitoring ensures complete trophoblastic regression. Future pregnancies require early ultrasound assessment to confirm intrauterine implantation and mitigate recurrence risks.

8. CONCLUSION

Ectopic pregnancy continues to pose a significant risk to maternal health, necessitating early diagnosis and individualized management strategies. Advances in diagnostic imaging and biomarker research hold promise for further refining treatment approaches. Moving forward, a combination of optimized screening, targeted therapies, and minimally invasive interventions is expected to improve both short- and long-term reproductive outcomes in affected patients. Adherence to clinical guidelines and close patient follow-up remain paramount in optimizing prognosis.

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Chapter 7

UTERINE MYOMA: DEFINITION, CLINICAL CHARACTERISTICS, AND TREATMENT APPROACHES

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1. INTRODUCTION

Uterine myoma, also known as leiomyoma or fibroids, is one of the most prevalent benign tumors of the female reproductive system. It affects approximately 20-40% of women of reproductive age and originates from the smooth muscle cells of the uterus (1). While many fibroids remain asymptomatic, some present with clinical manifestations such as menorrhagia, pelvic pain, and infertility (2). The etiology of fibroids is believed to be multifactorial, involving hormonal, genetic, and environmental influences (3).

The most commonly utilized diagnostic modality is transvaginal ultrasonography. Additionally, magnetic resonance imaging (MRI) and hysteroscopy are valuable diagnostic tools for evaluating fibroids (4). Treatment decisions are tailored based on factors such as patient age, symptom severity, and reproductive aspirations, incorporating medical, surgical, or minimally invasive techniques as appropriate (5).

This section provides a comprehensive examination of the etiology, clinical presentation, diagnostic methodologies, and contemporary treatment strategies for uterine fibroids, drawing upon the latest insights from the scientific literature.

2. EPIDEMIOLOGY, ETIOLOGY, AND PATHOPHYSIOLOGY OF UTERINE MYOMA

Uterine fibroids represent the most frequently occurring benign tumors in women of reproductive age. Epidemiological data indicate that approximately 20–40% of women are affected, with prevalence increasing as age advances (1). In women over the age of 40, the occurrence rate may rise to as high as 70% (2). Notably, racial differences have been documented; African American women

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