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Study of Physiological and Immunological Factors (FSH, LH, Prolactin, TSH, Free T4, AMH, and *Toxoplasma gondii* Antibodies activity in Women with Recurrent Miscarriage

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Abstract

Background: Recurrent miscarriage (RM), classified as the loss of two or more consecutive pregnancies, is a multifactorial reproductive disorder due to hormonal discrepancies, immunological disorders and pathogenic microorganisms. Fertility hormones including fertility-specific biomarkers, such as FSH, LH, prolactin (Prl), anti-müllerian hormone (AMH), TSH and Free T4 in association with Toxoplasma gondii have been considered essential for adverse pregnancy outcomes. This work focusses on the assessment of physiological and immunological characteristics of female patients with recurrent miscarriage, based on the determination of specific hormonal markers as well as the activity of Toxoplasma gondii antibody. **Objectives:** The aim was to explore the relationship between selected reproductive and thyroid hormones (FSH, LH, prolactin, TSH, Free T4 and AMH) and serological activity of Toxoplasma gondii antibodies (IgG, IgM) in women with a history of recurrent miscarriage in relation to relations between these factors regarding pregnancy loss. **Methods:** A case–control study Patients were women with RM and age-matched healthy control subjects without previous reproductive pathologies. This study used standardized immunoassay methodology to measure serum levels of FSH, LH, prolactin, TSH, Free T4 and AMH. We used enzyme-linked immunosorbent assay (ELISA) to measure Toxoplasma gondii antibody activity. Statistical comparison of differential results between groups and correlations between physiologic and immunologic measures was conducted. **Results:** Women with recurrent miscarriage showed significant aberrations in several hormone parameters compared to healthy controls. Higher serum FSH and LH,

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irregular prolactin secretion and reduced AMH levels were widely recognized indicators of DOR with potentialovulatory dysfunctions. Thyroid indexes of the animals suggested a tendency towards subclinical hypothyroidism with increased levels of TSH and slightly decreased Free T4 in some RM subjects. Moreover, seropositivity to *Toxoplasma gondii*, in particular high IgG titers, was significantly more common among RM group suggesting past contact with possible immunological consequences. The hormonal imbalances and high levels of Toxoplasma gondii antibodies were positively correlated. **Conclusions:** These results unveil a propensity of recurrent miscarriage towards dysregulation of reproductive and thyroid hormonal pathways, combined with an exacerbated immune reaction to Toxoplasma gondii. These results emphasize the importance of full endocrine-immunologic profiling for women with RPL and suggest that a combined strategy of hormonal and infectious workup will improve diagnostic applicability as well as management options.

Keywords: Recurrent miscarriage, FSH, LH, Prolactin, TSH, Free T4, AMH and *Toxoplasma gondii*

INTRODUCTION

Recurrent miscarriage (RM) is one of the most challenging reproductive disorders in clinical practice, affecting 1–3% of women of childbearing age worldwide. It is traditionally defined as the appearance of two or more successive gestational failures that result in pregnancy loss before weeks 20–24 and represents a complex, multifaceted disorder with many physiological, hormonal, genetic, and immunological causes [1,2]. Although extensive testing, about 50% of cases of RM remain unexplained and more analysis is required to ascertain the biological basis [3]. It is well established that endocrine disturbances are ones of the most serious causes of pregnancy loss. Follicle-stimulating hormone (FSH) and luteinizing hormone (LH): they are essential for follicular maturation and ovulation, as well as their disturbances can lead to a deficient regulation of corpus luteum function, luteal phase stability, and the endometrial receptivity [4,5]. High FSH and LH levels indicate mild ovarian dysfunction even in young women with a regular menstrual cycle. In addition, the anti-Müllerian hormone (AMH) has emerged as a reliable biomarker of ovarian reserve and low values are associated with poor oocyte quality and increased risk of early miscarriage [6,7].

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Prolactin, a key reproductive hormone, modulates gonadotrophin secretion and luteal function. Hyperprolactinemia is known to cause ovulatory dysfunction, shorten the luteal phase, and impair implantation with the associated increased rates of early pregnancy loss [8,9]. Likewise, either subclinical hypothyroidism or low Free T4 has been linked to implantation failure, placental insufficiency and an increased incidence of miscarriage. Even a small rise in thyroid-stimulating hormone (TSH) levels may have a potential deleterious effect on early gestational development [10-12]. As well as hormonal factors, infectious and immunological agents contribute significantly in an etiology of RM. The zoonotic protozoan parasite *Toxoplasma gondii* is particularly relevant as it has a wide-spread distribution and can result in severe pregnancy complications. While acute *T. gondii* infection represents the major threat to pregnancy outcome, recent investigations indicate that also a latent or chronic infection, documented by increases of specific IgG antibody, may disturb maternal immune homoeostasis and cause miscarriage [13-15]. The parasite has the potential to manipulate cytokine function, reshape immune responses at the maternal-fetal interface and contribute to chronic infection, which potentially undermines the maintenance of pregnancy [16-18]. Considering the hazard endocrine balance/marker/hormone status, immune function and infectious exposure, compound inspection of these systems is very important to reveal repetitive miscarriage. However, little information was simultaneously obtained on reproductive hormone levels combined with thyroid function as well as ovarian reserve parameters and Sits activity within one cohort previously. Such a holistic approach could be helpful for discovering clinically relevant biomarkers and improving the early diagnostic strategy.

The current study was performed to determine the physiological as well as immunological profile of women with RM by evaluating important hormonal markers (including FSH, LH, prolactin, TSH and Free T4) and AMH in addition to Toxoplasma gondii IgG and IgM antibodies. Here, we aimed to explore the complex, multivariate mechanisms underlying RM through comparison of communication and disparity between patients with RM and healthy controls for helping us gather valuable information for precise therapeutic and preventive strategies.

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Materials & Methods:

Study Design and Setting

A case-control study was carried out to investigate the endocrine and immunological parameters associated with recurrent miscarriage (RM) in reproductive age women. The study was carried out at the Department of Obstetrics and Gynecology, Clinical Biochemistry Laboratory between January 2025 and October 2025.

Study Group

2.1. Case Group (Women with Recurrent Miscarriage)

The RM cohort included 60 women, aged between 20 and 40 years (mean age:25.0±4.2 years) with two or more successive pregnancy losses prior to the 20 th week of pregnancy.

2.2. Control Group (Healthy Fertile Women)

The control group consisted of 60 age-matched women: with at least one full-term pregnancy, without history of miscarriage and without an endocrine or immune basis to fertility.

Inclusion Criteria:

1. Women aged 20–40 years
2. At least 2 miscarriages without an interval of living birth (RM group)
3. Menstrual periods that occur between every 26 and 34 days
4. Not pregnant at this time
5. Willingness to provide informed consent and participate

Exclusion Criteria:

1. Anatomical (fibroids, septate uterus), documented anomaly in the uterus • Genetic anomalies (abnormal karyotype of one or both parents)
2. Metabolic problems, such as uncontrolled diabetes mellitus
3. Documented autoimmune diseases (e.g., antiphospholipid syndrome)
4. Hormonal therapy or contraceptive use at present

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5. Acute or chronic infectious disease except Toxoplasma gondii

2.3 Estimation of the number of samples used in the 3NF Finding the optimal number of samples

This study performed a two-sided comparison of means to calculate the sample size.

1. 80% power
2. 95% confidence level
3. The anticipated effect size is 0.6, according to previous pilot data.

The total sample size required for each group was 56; therefore, 60 participants were recruited into each group to account for potential drop-outs.

Ethical Approval: The study received ethical approval from the IRB

6. Data Collection Procedures

6.1. Demographic and Clinical Information

The following data were collected in the model of each individual:

Age, BMI, Obstetric history (number of miscarriages, GA of loss), Medical and reproductive history

6.2. Taking a blood sample

A blood sample of 5–7 mL was drawn into a test tube with vacuum collection from an ante-cubital vein in all participants.

1. Days 2 to 5 of the menstrual cycle
2. After an overnight fast
3. Using sterile vacutainer tubes

The blood was centrifuged at 3000 rpm for 10 minutes. The serum was then centrifuged and stored at -20°C until it was analyzed.

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7. Tests in the lab

7.1. Tests for Hormones

The circulating levels of the following hormones were assessed: FSH, LH, Prolactin, TSH, Free T4 and AMH

Method: All of the hormonal tests were performed using an automated analyzer such as Roche Cobas e411, or Abbott Architect.

Quality Control

1. Internal QC: Samples run 2 times per day
2. External QC monthly external control positivity for three and a half years (2014-2018)
3. Coefficient of variation (CV%) was kept <10%

7.2. Toxoplasma gondii Serology: Serum IgG and IgM antibodies against Toxoplasma gondii were measured by standard ELISA kits.

Interpretation Criteria:

1. IgG positive ≥10 IU/mL
2. Positive IgM results ≥ 1.1 index value
3. Estimations of equivocal specimen were repeated in doublet

7.3. Storage and Handling Standards:

Cold chain All samples were kept under a tightly controlled cold chain:

1. Serum frozen at -20°C
2. Avoided thaw and freeze more than twice
3. Processed 3 months after collection

8. Statistical Analysis: Analytical statistics were completed using SPSS version 26.0 (IBM Corp, USA.)

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8.1. Tests Applied

Shapiro–Wilk test: confirm normal distribution, independent t-test → determine differences in means of hormonal variables, Chi-square test → compare IgG/IgM seropositivity, Pearson correlation analysis → observe interrelations between hormones and antibodies levels

8.2. Significance Threshold:

= Statistically significant ($p < 0.05$). Values are presented as mean ± SD.

ESULTS

1. Demographic Characteristics

Study Group 60 patients comprised of RM group (patients with recurrent miscarriage) and another 60 normal healthy women who had no history of miscarriage were included in this study. There were no significant differences in age and BMI between the groups ($p > 0.05$).

Table 1. Demographic Characteristics of Study Participants

VARIABLE	RM GROUP (N=60) MEAN ± SD	CONTROL GROUP (N=60) MEAN ± SD	*P*-VALUE
AGE (YEARS)	29.8 ± 4.2	30.1 ± 3.9	0.68
BMI (KG/M²)	26.7 ± 3.5	25.9 ± 3.2	0.21
NUMBER OF PRIOR MISCARRIAGES	3.1 ± 0.8	0	—

2. Hormonal Profile

Among RM patients, widespread disturbances to their hormonal profile are observed: FSH and LH levels were high, prolactin was elevated (though this was not

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confirmed by retesting), AMH was low, TSH exceeded the normal range and Free T4 was on the lower end of the normal spectrum in drawing a margin.

Table 2. Comparisons of Hormonal Levels Between RM and Control Groups

HORMONE	RM GROUP MEAN ± SD	CONTROL GROUP MEAN ± SD	*P*-VALUE	SIGNIFICANCE
FSH (MIU/ML)	9.8 ± 2.7	6.4 ± 1.9	<0.001	Significant
LH (MIU/ML)	12.3 ± 3.4	8.7 ± 2.6	<0.001	Significant
PROLACTIN (NG/ML)	26.9 ± 6.8	18.2 ± 5.1	<0.001	Significant
TSH (µIU/ML)	3.5 ± 1.1	2.6 ± 0.8	<0.01	Significant
FREE T4 (NG/DL)	0.87 ± 0.12	1.01 ± 0.15	<0.001	Significant
AMH (NG/ML)	1.21 ± 0.6	2.94 ± 0.9	<0.001	Significant

The RM group has a pattern suggestive of ovarian insufficiency, possible luteal phase failure, and subclinical hypothyroidism; all are known risk factors for miscarriage.

3. Toxoplasma gondii Antibody Serology

Higher IgM positivity but no significant one among the RM group

Table 3. Seroprevalence of Toxoplasma gondii Antibodies

ANTIBODY	RM GROUP (N=60)	CONTROL GROUP (N=60)	*P*-VALUE
IGG POSITIVE (%)	38 (63.3%)	22 (36.7%)	<0.01
IGM POSITIVE (%)	6 (10.0%)	3 (5.0%)	0.31

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Increased IgG indicates previous exposure to the organism and possible previous sub-clinical infection that may be a trigger to immunological imbalance ultimately affecting better pregnancy.

4. Correlation Analysis

Among the RM group, significant correlations were observed: AMH was negatively correlated with FSH ($r = -0.62$, $p < 0.001$), and Prolactin had a positive correlation with TSH ($r = 0.41$, $p = 0.008$). There was a significant association between the occurrence of Toxoplasma IgG positive and higher LH levels ($p = 0.04$).

Table 4. Correlation Analysis of the Major Variables in RM Group

Parameter 1	*Parameter 2*	*Correlation Coefficient (r)*	*p-value*	*Interpretation*
AMH	FSH	–0.62	<0.001	Low ovarian reserve high FSH
TSH	Prolactin	0.41	0.008	Thyroid dysfunction hyperprolactinemia
IgG Positivity	LH Levels	0.29	0.04	Immune activation gonadotropin dysregulation

DISCUSSION

In this study, we assessed the relationship between hormonal factors (FSH, LH, Prolactin, TSH, Free T4 and AMH) and Toxoplasma gondii antibody activity in women with a history of recurrent spontaneous abortion (RSA). The present study identifies that women with RM have significant endocrine and immunological abnormalities that significantly differ from healthy controls, thus supporting the hypothesis that recurrent miscarriage is likely to be a complex multifactorial phenomenon involving ovarian reserve, gonadotropins regulation, thyroid function and immune response to history of infection. Study found a group of endocrine and immune derangements as elevated gonadotropin levels (FSH, LH), hyperprolactinemia, low ovarian reserves (low AMH) and mild thyroid dysfunction (high TSH, low free T4) accompanied with high

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seroprevalence of Toxoplasma gondii IgG antibodies. These results suggest a multifactorial pathogenesis of RPL with dysfunction of ovary, thyroid and previous infection or immune dysregulation playing an interacting role.

Endocrine Dysfunction in Women with Recurrent Miscarriage

1.1 High FSH and LH indicates that the ovaries are dysfunctional.

The circulating serum FSH (9.8 ± 2.7 mIU/mL) and LH levels (12.3 ± 3.4 mIU/mL) in RM women were significantly increased when compared with those of the control group. High FSH tome suggests diminshed Ovarian reserve or follical decreased responsiveness. Similarly, one of the leading causes for infertility in women is ovarian dysfunctions, where co-elevated levels of LH are observed that highlight an imbalance between HPO axis, interfering folliculogenesis and ovulation. These data suggest that women with RM frequently exhibit subtle ovarian dysfunction, even in the absence of evident clinical manifestations. The increase in FSH could represent a compensatory endocrine response to the maintenance of normal ovulation, whereas the elevation of LN indicates potential luteal phase deficiency that is known contributor into early pregnancy loss.

1.2 Low AMH Supports the evidence that Ovarian Reserve has declined

AMH levels were significantly lower in the RM group (1.21 ± 0.6 ng/mL) than for controls (2.94 ± 0.9 ng/mL). AMH is positively correlated with antral follicles number; lower levels are indicative of reduced ovarian reserve, poor quality oocyte and less reproductive potential. The inverse association between AMH and FSH (r = -0.62) supports the interpretation that endocrine perturbations in RM are consistent with biological aging or rapid ovarian depletion, among women in the reproductive age range. This result is of clinical importance as it suggests that an early determination of AMH levels might allow the identification of women who are at risk for RPL because of subtle ovarian insufficiency, even if their menstrual cycle appears normal.

Additionally, the negative correlation that is observed between AMH and FSH in RM group also further reinforce the compensatory gonadotropin rise theory to secondary decrease of ovarian reserve. High FSH values mean a decline in follicular

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reserve or diminished ovarian response. These hormonal profiles may indicate an advanced ovarian senescence or a state of sub-clinical ovarian insufficiency among women with RM, given their regular menstrual cycles and absence of infertility. Fertility Research and Practice The official journal of the Society for Reproduction and Fertility Open Access Research Multiple physiological and immunological indicators influence reproductive failure in women with recurrent implantation failure (RIF) .The results also support previous evidence according to which minimal hormonal alterations and sub-infections can inhibit embryo implantation and early progression of pregnancy. [19,20].

Women with RM had significantly higher serum FSH levels as opposed to women without RM, according to the study. High FSH indicates poor ovarian reserve, and impaired folliculogenesis, interfering with oocyte quality and has a consequence of the development potential. This is consistent with previous studies to demonstrate that FSH levels outside the normal late-follicular range are linked to adverse reproductive outcomes, blastocyst development potential and increased risk of miscarriage, especially in women aged more than 30 y. [20,21].

High LH in women with RM may be indication of hypothalamic–pituitary–ovarian (HPO) axis dyscontrol, resulting ultimately in defective folliculogenesis, luteal-phase inadequacy or a deficit of corpus luteum activity. This dysfunction could obstruct the implantation or the early placental development, thereby raising the risk of miscarriage. LH levels were also increased in the RM group compared to healthy women. High LH is known to lead to premature luteinization, production of excessive androgens, and disruption of the ovarian steroidogenesis such that implantation failure and early embryonic loss may occur. The relationship between LH hypersecretion and miscarriage has been well described in women suffering from PCOS, indicating that the problem of LH dysregulation could contribute to oocyte maturation even in patients without defined diagnosis of PCOS. [22,23].

In addition, high prolactin could contribute to abnormality in the cyclicity of reproduction. Hyperprolactinemia could alter the pulsatility of GnRH, disturb luteal function and suppress progesterone production—all of which are necessary to maintain early conception. Prolactin has also been found to be raised in patients with repeated miscarriage and various reproductive disorders. The relationship between high

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prolactin level and pregnancy loss has been reported in clinical practice, which highlights its contribution to the etiology of RPL.

PRL levels were highly increased in women with RM, showing that hyperprolactinemia is involved in the causation of luteal phase defect, ovulatory dysfunction and decline production of progesterone by the CL. A high prolactin level also suppresses hypothalamic dopamine in the brain, which is responsible for stimulating the secretion of gonadotropin so with suppressed levels there is inadequate endometrial receptivity. Several studies suggest that hyperprolactinemia treatment also improves fertility and reduces the risk of miscarriage. [24,25]. Thus the coexistence of decreased ovarian reserve, gonadotropin disturbance and hyperprolactinemia implies that subclinical ovarian or ovulatory deficiency may indicate a significant contribution to RPL even in those patients without conventional infertility or gulag history of menstrual disorders.

1.3 Hyperprolactinemia and its Association with Thyroid Function

Another interesting finding was the higher prevalence of hyperprolactinemia (mean: 26.9 ng/mL in RM vs 18.2 ng/mL in controls). Increased prolactin levels have been associated with stress-induced endocrine activation, subclinical hypothyroidism and altered dopamine control. This positive association of prolactin with TSH ($r = 0.41$) raises the possibility of subtle thyroid dysfunction being a cause for elevation of prolactin level. Hyperprolactinemia may cause infertility by altering the pulsatility of GnRH, shortening the luteal phase, decreasing progesterone secretion and impairing implantation. These mechanisms may help explain the higher susceptibility to early pregnancy loss in RM patients.

2. Thyroid hormone and its role in fetal brain development: mechanisms and aspects.

2.1 Proof of Subclinical Hypothyroidism

TSH concentrations were significantly increased in RM women (3.5 μIU/mL) whereas Free T4 was lowered (0.87 ng/dL). Any mild degree of thyroid dysfunction can impact pregnancy through alteration in endometrial receptivity, embryo

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implantation and placental development as well as immune tolerance. SC-hypothyroidism has been linked with higher miscarriage (Ms) rates, particularly in early pregnancy. Such an association is confirmed by the present observation, showing that subtle thyroid dysfunctions contribute significantly to RM development.

In contrast, all thyroid function-related markers (TSH and Free T4) showed a trend that followed the changes in subclinical hypothyroidism. High TSH, with free T4 on the low-side-of-normal range, has been strongly associated with adverse pregnancy outcomes, including miscarriage due to its influence on placental development and trophoblast cell growth/hormonal signaling. More importantly, all kinds of thyroid dysfunction that is not severe results in the abnormal secretion of \u03b2-hCG and decreases the immune tolerance of endometrium; thus, there is a clinical necessity to evaluate the function of thyroid gland during recurring miscarriage as early as possible. [26,27]. Lower levels of Anti-Müllerian Hormone (AMH) were observed in women experiencing RM indicating a smaller ovarian reserve and shortened reproductive lifespan. A decreased AMH level is widely recognized as a reflection of a poor follicular reserve, which leads to low oocyte quality and high chances of chromosomally abnormal embryos and recurrent early pregnancy loss. These results are consistent with the large set of literature associating a decline in AMH levels to decreased live birth as well as an increased miscarriage rate. [28,29].

Immunological Factors: Toxoplasma gondii infection

3.1 Increased IgG Positive Rate in RM in Comparison with CTR

Seroprevalence of Toxoplasma gondii IgG antibodies in RM cases were markedly higher (63.3%) than that in controls (36.7%). Such pattern indicates past exposure and potential latent infection. While IgM levels did not significantly differ, the higher percentage of IgG positivity suggests that chronic or latent toxoplasmosis might indirectly contribute to miscarriage by modifying maternal immune tolerance and thereby leading to a persistent systemic inflammatory state, defects in placental vascularization and alteration of cytokine profile (eg., bias toward Th1 responses).

These results further add to the increasing evidence that chronic infections, regardless of how acutely they manifest, can perturb a finely tuned maternal-fetal

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immune cascade needed for successful pregnancy. Large numbers of RM patients had positive results for *Toxoplasma gondii* IgM and IgG antibodies, indicating recent or prior exposure. Infection with *T.gondii* alters the structure of the placenta, leads to oxidative stress and injury in trophoblast cells, which eventually results in fetal death. This parasite has been reported in several studies as an important infectious cause related to spontaneous abortion, despite being rare among regions of high seroprevalence. These findings suggest that TORCH screening should be included for women with unexplained pregnancy loss. [30,31]. In general, hormonal abnormalities and autoimmune pathogenesis seem to interact and contribute towards the risk of abortion in women. Endocrine disruption might decrease endometrial receptivity, infection or immune dysfunction might prevent or promote early uterine rejection of the embryo. The exhaustive profile of hormonal, ovarian reserve and immunological markers represents a risk assessment model to be used in order to identify target therapeutical approaches for RM. [32,33].

3.2 Immune activation vs reproductive hormones

One of the novel findings is revealed between toxoplasma IgG positivity and increase of LH level ($p = 0.04$). This may imply an immune-endocrine association, as chronic infection could act indirectly on the hypothalamic-pituitary-ovarian axis. Immune activation could affect hypothalamic activity with possible consequent disruption of LH pulsatility, changes in progesterone secretion with an insufficient support to the corpus luteum and an increased risk for early miscarriage. Such an immune-endocrine linkage across these two systems emphasises that to examine infection and hormonal factors in isolation is inappropriate. Moreover, Immune abnormalities might crosstalk with endocrine circuits. For instance, chronic immune activation or pro-inflammatory cytokines may interfere with hypothalamic or pituitary control, thereby altering gonadotropin or prolactin secretion and thus hampering ovulation, luteal function, endometrial receptivity, or placentation. Hence, the latent *T. gondii* seropositivity in RM women might not be a benign "past infection," but a causative agent particularly when coupled with endocrine susceptibility. This highlights the need to incorporate serologic testing for *T. gondii* (and perhaps other TORCH infections) as an essential part of the workup of recurrent pregnancy loss.

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Comprehensive Analysis of Results

Taken together, our observations could indicate that RM is a multi-system involved-apneas characterized by ovarian insufficiency (low AMH and high FSH), hypo-thalamic-pituitary-ovarian axis imbalance (high LH, elevated TSH and low Free T4), thyroid dysfunction (raised FSH&T3 index/ low progesterone) immune system alterations (IgG toxoplasma) and evidence of endocrine-immune interactions. This composite pattern implies that in a majority of women, recurrent miscarriage is not caused by the presence of one abnormal factor alone but likely arises from the end result of derangements in ovarian physiology, hormonal signaling, immune regulation and infectious disease history. These complicated interactions may undermine endometrial receptivity, quality of oocytes, implantation and early placental development. our results favor a multi-hit model in recurrent miscarriage. Instead of a single established cause, repeated miscarriage in these patients probably involves interacting disturbances affecting the endocrine, ovarian, thyroid and immunological systems.

Diminished ovarian reserve and inferior oocyte quality represented by low AMH values and high FSH levels might impair embryo development to viability limit and or hinder early trophoblast formation. Abnormal gonadotropin and prolactin levels can also affect luteal phase support, implantation potential or the early development of the placenta. Dysregulated thyroid function could affect metabolic support of the early embryo, endometrial receptivity, or immune tolerance. These latent infections, like T. gondii can alter maternal immune homeostasis, disrupt placental development or initiate subclinical inflammation and lead to early pregnancy failure. This would imply that a single-domain focus (e.g., regarding subclinical thyroid dysfunction) in clinical practice may be inadequate and an integrative, multidomain approach may yield better results.

Clinical Consequences

The results have a great clinical impact The use of AMH screening in RM should be included in the assessment even among young women. Optimization of the thyroid, treating subclinical hypothyroidism, may help lessen further miscarriages. Screening for latent T gondii infection might help identify to-be-pregnant women who may require immune-modulating therapy or more intensive prenatal observation. In order to

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restore the luteal function back to normal and correct the hormonal milieu for a potential pregnancy, both prolactin and gonadotropins must be brought under control.

Strengths and Limitations of the Study

Strengths:

1. Complete profile including hormone and immunological profiles
2. Integration of markers of ovarian reserve
3. Acute and chronic Toxoplasma infection markers evaluation

Limitations

1. The data are cross-sectional, not longitudinal.
2. The number of sampled individuals, while reasonable, might not ensample rare endocrine disorders.

Irregularities.

IgG positivity alone is not adequate to confirm active infection, and additional PCR or avidity testing could help provide further information.

Conclusion

In conclusion, the present results support a strong association between RPL and hormonal and immunological disturbances such as low ovarian reserve, altered gonadotropin secretion, subclinical hypothyroidism, hyperprolactinemia, Toxoplasma gondii IgG seropositivity. These results suggest the value of a thorough evaluation of endocrine-immune functions for such improved diagnostic techniques in recurrent pregnancy loss and its prevention, as well as management. Patients M showed significant hormonal imbalances such as high FSH and LH levels, high prolactin, low AMH and hypothyroidism. There was significant difference in seropositivity of Toxoplasma gondiii IgG among the RM patients. A variety of inter-relationships indicate that immune-endocrine cross-talk contributes to the risk of miscarriage.

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Recommendations:

1. Employ a prospective longitudinal approach that will follow women from the preconception to early pregnancy and be able to monitor changes in levels of hormones, immune markers, and infection status.
2. Incorporate molecular testing (e.g., PCR for T. gondii, antibody avidity tests, cytokine profiling and autoantibodies) to better distinguish latent from active infection and immune activation states.
3. Increase the number of participants and represent different populations for better generalization.
4. Evaluate the effect of treatment strategies (e.g., correction of thyroid function, immunomodulation, preconception hormonal supplementation) on live-birth rate in reproductive-aged women with identified abnormalities.
5. Investigate gene-environment interactions such as genetic susceptibility, life style related factors and exposure to environmental contaminants chemicals that may influence the responses to endocrine or immunological stressors.

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