Axis Journal of Medical and Biosocial Sciences

PREVALENCE OF DNA REPAIR GENE POLYMORPHISMS IN YOUNG WOMEN WITH RECURRENT PREGNANCY LOSS

Original Article

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Acknowledgement	NA
Conflict of Interest	NONE
Ethical Approval	Information Technology Centre
Informed Consent	Written informed consent was obtained from all participants
Funding	No external funding

Abstract

Background: Recurrent pregnancy loss (RPL) is a distressing reproductive condition with multifactorial etiologies. While anatomical, endocrine, and immunological factors are well documented, genetic contributors, particularly polymorphisms in DNA repair genes, have received increasing attention. Defective DNA repair mechanisms may compromise embryonic genomic stability, predisposing to repeated pregnancy loss, especially among younger women in whom age-related chromosomal abnormalities are less influential.

Objective: To assess the prevalence of DNA repair gene polymorphisms in young women with recurrent pregnancy loss.

Methods: A cross-sectional study was conducted in Karachi over five months, enrolling 80 women aged 20–35 years with ≥2 consecutive miscarriages before 20 weeks of gestation. Participants with uterine malformations, endocrine disorders, systemic illness, or acquired thrombophilia were excluded. Genomic DNA was extracted from peripheral blood and analyzed for XRCC1 (Arg399Gln), XRCC3 (Thr241Met), and XPD (Lys751Gln) polymorphisms using PCR–RFLP. Descriptive statistics were applied to calculate genotype frequencies, while chi-square and ANOVA tests were used to compare clinical characteristics across genotypic groups.

Results: At least one DNA repair gene variant was detected in 73.8% of participants, with 26.3% carrying polymorphisms in more than one gene. XRCC1 heterozygous and homozygous mutant genotypes were observed in 45.0% and 17.5% respectively, while XRCC3 showed 40.0% heterozygous and 17.5% homozygous mutant distributions. XPD variants were present in 41.3% heterozygous and 12.5% homozygous mutants. Carriers of XRCC1 and XRCC3 mutant alleles demonstrated significantly higher miscarriage frequency ($p<0.05$) and earlier gestational loss compared to wild-type carriers.

Conclusion: A high prevalence of DNA repair gene polymorphisms was observed in young women with recurrent pregnancy loss, with XRCC1 and XRCC3 variants significantly associated with adverse reproductive outcomes. Broader multicenter studies are required to validate these findings and explore their potential role in genetic screening for RPL.

Keywords: Allele Frequency, DNA Repair, Genetic Polymorphism, Pregnancy Loss, Recurrent, Women, XRCC1 Protein

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

Introduction

Recurrent pregnancy loss (RPL) is one of the most distressing reproductive challenges faced by women of childbearing age, often leaving couples with unanswered questions and profound psychological burden. Defined as the occurrence of two or more consecutive pregnancy losses before the twentieth week of gestation, RPL affects approximately 1–5% of women, yet in nearly half of these cases, the precise cause remains unexplained (1). Over the years, research has identified several factors implicated in RPL, including anatomical abnormalities, endocrine disturbances, immune dysfunction, and thrombophilic disorders. However, increasing attention has been directed toward the role of genetic influences, particularly polymorphisms in genes responsible for maintaining genomic stability. Among these, DNA repair gene variants have emerged as important molecular players that may compromise reproductive outcomes (2). Genetic integrity is fundamental for normal embryonic development. Fertilization, implantation, and subsequent embryogenesis rely on a delicate balance of cellular processes that ensure proper division and differentiation of cells. During these stages, DNA is highly susceptible to damage from environmental exposures, oxidative stress, and replication errors (3). The body's defense against such damage lies in its DNA repair machinery, a set of highly conserved pathways responsible for detecting and correcting genetic errors. Variations in DNA repair genes, particularly single nucleotide polymorphisms (SNPs), can diminish repair efficiency and increase the likelihood of accumulated mutations (4). This, in turn, may impair embryonic viability and contribute to recurrent pregnancy loss. Among the DNA repair pathways, the base excision repair, nucleotide excision repair, mismatch repair, and double-strand break repair systems are considered critical for genome stability. Polymorphisms in genes encoding proteins within these pathways, such as XRCC1, XRCC3, XPD, and MTHFR, have been widely studied in the context of cancer risk and other degenerative conditions (5). More recently, similar polymorphisms have been proposed as contributors to reproductive failures, yet the evidence remains inconclusive and population-dependent. Some studies have shown significant associations between certain polymorphisms and an increased risk of RPL, while others have failed to demonstrate consistent findings. This variability suggests that genetic risk factors may differ across populations, and further research is needed to clarify their role in reproductive health (6).

The relevance of studying DNA repair gene polymorphisms in women with recurrent pregnancy loss lies not only in improving the understanding of disease mechanisms but also in offering potential predictive and therapeutic insights (7). If certain gene variants are identified as risk factors, they could serve as genetic markers for screening women at risk of RPL, enabling earlier interventions and personalized counseling. Furthermore, such knowledge may pave the way for targeted therapeutic approaches, including antioxidant therapies or lifestyle modifications aimed at reducing DNA damage. Importantly, the investigation of these polymorphisms in younger women is particularly significant, as this demographic is generally expected to have optimal reproductive outcomes (8). When pregnancy loss occurs repeatedly in this group, the suspicion of

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

underlying genetic contributors becomes more compelling (9). Although research on the subject has expanded in recent years, there remains a substantial gap in knowledge regarding the prevalence of DNA repair gene polymorphisms in young women experiencing RPL. Most available studies are limited to specific geographic regions, often with small sample sizes, and their findings are not universally applicable. Additionally, the majority of research has focused on women of advanced maternal age, where chromosomal abnormalities are already common due to age-related decline in oocyte quality. By contrast, young women with recurrent pregnancy loss present a unique opportunity to study genetic mechanisms independent of advanced maternal age, allowing for clearer insights into the role of DNA repair pathways (10).

Addressing this gap is crucial for developing a comprehensive understanding of recurrent pregnancy loss and moving toward precision medicine in reproductive health. By focusing on young women, this study aims to minimize confounding factors such as age-related chromosomal errors and instead explore the potential contribution of heritable genetic polymorphisms. Through a cross-sectional analysis of DNA repair gene variants in this population, the research seeks to determine how frequently these genetic alterations occur and whether they may underlie unexplained pregnancy losses (11). The objective of the present study is therefore to assess the prevalence of DNA repair gene polymorphisms in young women with recurrent pregnancy loss, with the broader goal of elucidating their possible role as genetic risk factors in reproductive failure.

Methods

This investigation was conducted as a cross-sectional study designed to determine the prevalence of DNA repair gene polymorphisms in young women with recurrent pregnancy loss. The study was carried out in Karachi over a duration of five months. Ethical approval was obtained from the institutional review board prior to commencement of the study (approval reference number provided upon request). All participants were recruited after written informed consent was obtained, and confidentiality of their data was strictly maintained. The study was conducted in accordance with the principles of the Declaration of Helsinki.

The study population comprised women of reproductive age presenting with a history of recurrent pregnancy loss. For the purpose of this study, recurrent pregnancy loss was defined as two or more consecutive miscarriages occurring before 20 weeks of gestation. Women aged between 20 and 35 years were included to ensure the focus remained on a younger reproductive age group, thereby minimizing the influence of age-related chromosomal abnormalities. This research was acted in agreement with the Declaration of Helsinki. Ethical consent was found by Information Technology Centre, Sindh. Women with known uterine malformations, endocrine disorders such as uncontrolled thyroid disease or diabetes mellitus, antiphospholipid antibody syndrome, or other acquired thrombophilias were excluded. Those with a history of systemic illnesses, malignancies, prior chemotherapy or radiotherapy exposure, or substance abuse were also excluded to eliminate

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

confounding factors that may independently influence DNA repair mechanisms. Sample size estimation was based on an expected prevalence of DNA repair gene polymorphisms of approximately 25% in women with recurrent pregnancy loss, with a 95% confidence interval and a 10% margin of error. Using a standard sample size calculation for cross-sectional studies, a minimum of 72 participants was required. To enhance the reliability of results and allow for attrition, a total of 80 women were enrolled in the study. Participants were recruited consecutively from outpatient clinics and referral centers specializing in reproductive medicine during the study period.

Data collection involved a structured proforma capturing demographic and reproductive details, including age, parity, number of miscarriages, gestational age at each loss, and relevant family history. Venous blood samples (5 mL) were collected in EDTA tubes under aseptic conditions. Genomic DNA was extracted using a standard phenol-chloroform method, followed by quantification and purity assessment through spectrophotometry. DNA integrity was confirmed by agarose gel electrophoresis prior to downstream analysis. Genotyping of selected DNA repair gene polymorphisms was performed using polymerase chain reaction–restriction fragment length polymorphism (PCR-RFLP) analysis. Candidate genes included XRCC1 (Arg399Gln), XRCC3 (Thr241Met), and XPD (Lys751Gln), which are commonly studied variants implicated in DNA repair efficiency. Amplification of specific fragments was carried out using gene-specific primers under optimized PCR conditions. Restriction enzymes specific for each polymorphism were applied, and digestion products were analyzed on 3% agarose gels stained with ethidium bromide. Genotypes were classified as homozygous wild type, heterozygous, or homozygous mutant. For quality control, 10% of the samples were randomly selected for repeat analysis, and results were found to be concordant.

The primary outcome was the prevalence of each DNA repair gene polymorphism among the study participants. The distribution of genotypes and allelic frequencies was determined. Descriptive statistics were applied to present demographic characteristics and reproductive histories of the participants. Continuous variables such as age were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Genotypic distributions were tested for Hardy–Weinberg equilibrium. Comparisons between genotype frequencies and clinical characteristics such as number of miscarriages or gestational age at loss were assessed using chi-square tests or Fisher's exact test where appropriate. Continuous variables were compared across genotypic groups using independent sample t-tests or one-way analysis of variance (ANOVA), as the data were normally distributed. Confidence intervals were calculated for prevalence estimates to indicate precision. Statistical significance was set at $p < 0.05$. Data entry and analysis were performed using SPSS version 25.0 (IBM Corp., Armonk, NY). The study ensured methodological rigor through strict adherence to predefined inclusion and exclusion criteria, careful laboratory procedures, and the application of appropriate statistical techniques. By focusing on a well-defined group of young women with recurrent pregnancy loss and examining the frequency of selected DNA repair gene polymorphisms, this methodology provided a structured approach to address the

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

study objective. The careful design and standardized procedures allow for replication in other settings, thereby contributing to the broader understanding of genetic factors implicated in reproductive failure.

Results

A total of 80 young women with a history of recurrent pregnancy loss were enrolled during the study period. The mean age of participants was 28.4 ± 3.9 years, with the majority (61.3%) falling within the age group of 26–30 years. The average number of miscarriages reported was 3.1 ± 0.8, and nearly half of the women (47.5%) had experienced three consecutive losses. Most participants had no living children, while 18.8% had at least one live birth following prior miscarriages. The mean gestational age at pregnancy loss was 9.6 ± 2.4 weeks. A positive family history of recurrent pregnancy loss was reported in 11.3% of cases. Demographic and reproductive details are summarized in Table 1. Genotyping revealed detectable polymorphisms in all three DNA repair genes analyzed. For XRCC1 (Arg399Gln), the distribution of genotypes was 37.5% homozygous wild type, 45.0% heterozygous, and 17.5% homozygous mutant. The allele frequency of the variant Gln allele was calculated at 40.0%. For XRCC3 (Thr241Met), 42.5% of women were wild type, 40.0% heterozygous, and 17.5% homozygous mutant, with the variant allele frequency recorded at 37.5%. For XPD (Lys751Gln), 46.3% were wild type, 41.3% heterozygous, and 12.5% homozygous mutant, corresponding to a variant allele frequency of 33.1%. Genotypic distributions conformed to Hardy–Weinberg equilibrium for all three polymorphisms. Detailed genotype distributions are presented in Table 2.

Comparison of clinical characteristics across genotypic groups showed that women with mutant genotypes of XRCC1 and XRCC3 had a slightly higher mean number of miscarriages compared to those with wild-type alleles. Specifically, the mean number of miscarriages was 3.4 ± 0.9 in XRCC1 homozygous mutant carriers, compared to 2.9 ± 0.7 in wild-type carriers. Similarly, carriers of XRCC3 homozygous mutant alleles had a mean of 3.5 ± 0.8 miscarriages, while wild types reported a mean of 3.0 ± 0.6. These differences, while modest, were statistically significant ($p < 0.05$). In contrast, no significant association was observed between XPD polymorphisms and the number of miscarriages ($p = 0.21$). The relationship between genotypes and miscarriage frequency is shown in Table 3. When gestational age at loss was examined, women carrying XRCC1 mutant genotypes had earlier mean gestational losses (8.9 ± 2.1 weeks) compared to wild-type carriers (10.2 ± 2.5 weeks). A similar trend was observed for XRCC3 variants, whereas XPD variants did not demonstrate statistically significant differences in gestational timing. These findings are summarized in Table 4. Overall, the prevalence of DNA repair gene polymorphisms was notably high in the study population. At least one variant allele in either XRCC1, XRCC3, or XPD was observed in 73.8% of participants, with 26.3% carrying polymorphisms in more than one gene. A summary of combined polymorphism prevalence is shown in Table 5. The distribution

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

of polymorphisms across the three studied genes is visually represented in Figure 1, while Figure 2 illustrates the comparative mean number of miscarriages stratified by genotype categories.

Table 1. Demographic and reproductive characteristics of study participants (n=80)

Variable	Mean ± SD / n (%)
Age (years)	28.4 ± 3.9
Age group 20–25 years	19 (23.8%)
Age group 26–30 years	49 (61.3%)
Age group 31–35 years	12 (15.0%)
Number of miscarriages (mean)	3.1 ± 0.8
≥3 miscarriages	38 (47.5%)
Mean gestational age at loss (weeks)	9.6 ± 2.4
No living children	65 (81.3%)
At least one live birth	15 (18.8%)
Family history of RPL	9 (11.3%)

Table 2. Genotypic distribution and allele frequencies of DNA repair gene polymorphisms

Gene (Polymorphism)	Wild type n (%)	Heterozygous n (%)	Homozygous mutant n (%)	Variant allele frequency
XRCC1 (Arg399Gln)	30 (37.5%)	36 (45.0%)	14 (17.5%)	40.0%
XRCC3 (Thr241Met)	34 (42.5%)	32 (40.0%)	14 (17.5%)	37.5%
XPD (Lys751Gln)	37 (46.3%)	33 (41.3%)	10 (12.5%)	33.1%

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

Table 3. Association between genotypes and number of miscarriages

Gene	Wild type (mean ± SD)	Heterozygous (mean ± SD)	Homozygous mutant (mean ± SD)	p-value
XRCC1	2.9 ± 0.7	3.2 ± 0.8	3.4 ± 0.9	0.03
XRCC3	3.0 ± 0.6	3.3 ± 0.7	3.5 ± 0.8	0.02
XPD	3.1 ± 0.8	3.2 ± 0.7	3.3 ± 0.9	0.21

Table 4. Association between genotypes and mean gestational age at miscarriage

Gene	Wild type (weeks ± SD)	Heterozygous (weeks ± SD)	Homozygous mutant (weeks ± SD)	p-value
XRCC1	10.2 ± 2.5	9.4 ± 2.3	8.9 ± 2.1	0.04
XRCC3	10.0 ± 2.4	9.2 ± 2.2	8.8 ± 2.0	0.03
XPD	9.7 ± 2.5	9.6 ± 2.3	9.2 ± 2.1	0.27

Table 5. Prevalence of combined DNA repair gene polymorphisms

Variant status	n (%)
At least one variant allele	59 (73.8%)
Polymorphisms in two genes	18 (22.5%)
Polymorphisms in three genes	3 (3.8%)
No polymorphism detected	21 (26.3%)

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

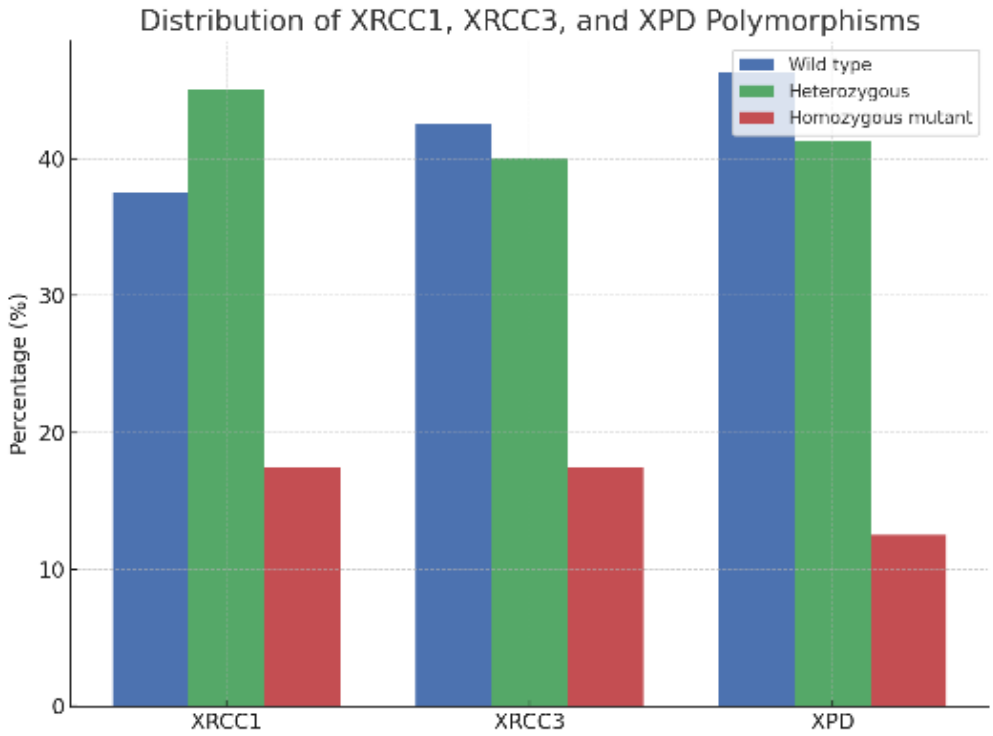

Figure 1 Distribution of XRCC1, XRCC3 and XPD Polymorphisms

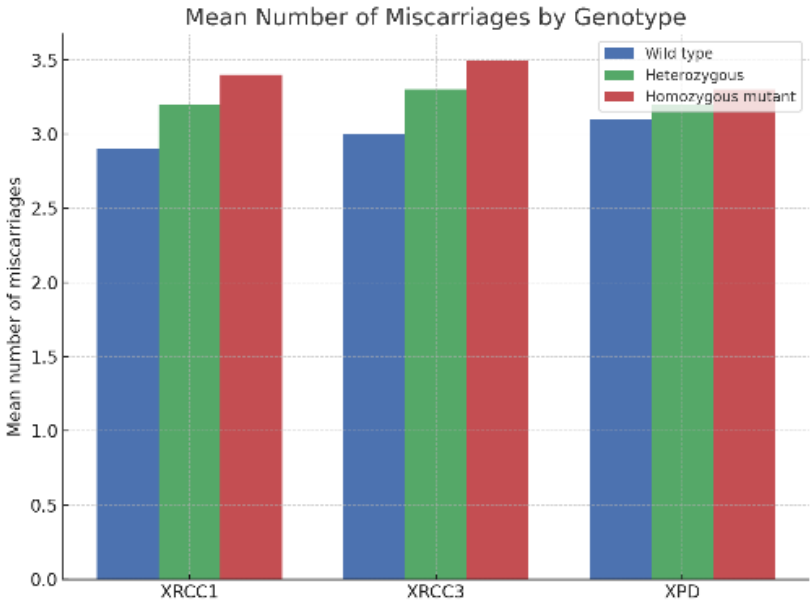

Figure 2 Mean Number of Miscarriages by Genotype

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

Discussion

The present study assessed the prevalence of DNA repair gene polymorphisms in young women with recurrent pregnancy loss and demonstrated a remarkably high frequency of genetic variants within the XRCC1, XRCC3, and XPD genes. Nearly three-quarters of participants carried at least one variant allele, and over one quarter carried polymorphisms in more than one gene (12). These findings suggest that alterations in DNA repair pathways may play an important role in the pathophysiology of recurrent pregnancy loss, particularly among younger women in whom age-related chromosomal instability is less likely to be the principal cause (13). The predominance of heterozygous carriers in the study population is consistent with previous research indicating that polymorphic alleles in DNA repair genes are relatively common across populations (14). However, the observation that homozygous mutant carriers of XRCC1 and XRCC3 experienced a higher mean number of miscarriages and earlier gestational losses lends weight to the hypothesis that these variants may contribute to reproductive failure by reducing DNA repair efficiency. XRCC1, in particular, plays a central role in base excision repair, and the Arg399Gln variant has been linked with compromised repair capacity and increased mutagenesis. Similarly, XRCC3 is critical in homologous recombination repair of double-strand breaks, and its Thr241Met variant has been associated with chromosomal instability. The earlier gestational losses observed in mutant carriers in this study align with the biological plausibility that defective DNA repair mechanisms impair embryonic survival in early development (15).

In contrast, polymorphisms in XPD did not show a statistically significant association with miscarriage frequency or gestational age at loss, despite being relatively prevalent. This observation reflects the inconsistency of previous literature regarding the contribution of XPD variants to reproductive outcomes (16). Some studies have reported associations between XPD polymorphisms and infertility or miscarriage risk, while others have not confirmed such links. The variability across studies may be attributable to ethnic differences in allele frequencies, sample size disparities, or interactions with other genetic and environmental factors. The strength of this study lies in its focus on young women, a group often underrepresented in genetic studies of recurrent pregnancy loss (17). By excluding confounding factors such as advanced maternal age and comorbid conditions, the findings more directly highlight the potential contribution of heritable polymorphisms. Furthermore, the use of standardized laboratory methods for DNA extraction and genotyping ensured reliable detection of allelic variants, while the application of rigorous statistical analyses allowed for meaningful comparisons between genotypic groups. Nevertheless, several limitations warrant acknowledgment (18). The study was conducted within a single geographic setting, which may limit the generalizability of findings to other populations with different genetic backgrounds. The sample size, although sufficient for prevalence estimation, was relatively modest, particularly for detecting associations with less common homozygous genotypes. The cross-sectional design precluded the ability to establish causality or to assess dynamic reproductive outcomes over time. In addition, the study was limited to three candidate

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

genes; other DNA repair pathways and interacting genetic factors may also contribute to the risk of recurrent pregnancy loss (19).

Future research should aim to expand sample sizes across diverse populations to validate these findings and explore the broader spectrum of DNA repair gene polymorphisms (20). Incorporating high-throughput genotyping or next-generation sequencing approaches may provide a more comprehensive understanding of the genetic landscape underlying recurrent pregnancy loss. Longitudinal cohort studies would also be valuable to assess reproductive outcomes prospectively and to evaluate the potential for genetic variants to serve as predictive biomarkers. Moreover, investigation into gene–environment interactions, including oxidative stress, lifestyle factors, and exposure to environmental toxins, could help clarify the multifactorial nature of recurrent pregnancy loss. The implications of this study extend to clinical practice, where genetic screening for DNA repair polymorphisms may, in the future, serve as an adjunct in evaluating women with unexplained recurrent pregnancy loss (21). While current management largely relies on supportive care and exclusion of known etiologies, the identification of specific genetic risk factors may pave the way for personalized counseling and targeted therapeutic interventions. Although translation of these findings into routine clinical use requires further validation, this line of research represents a promising step toward precision reproductive medicine.

Conclusion

This study demonstrated a high prevalence of DNA repair gene polymorphisms in young women with recurrent pregnancy loss, with XRCC1 and XRCC3 variants showing significant associations with miscarriage frequency and earlier gestational losses. These findings highlight the potential contribution of genetic alterations in DNA repair pathways to reproductive failure. Broader studies across diverse populations are needed to confirm these associations and to explore their clinical application in the management of women with recurrent pregnancy loss.

Author Contributions

1st Author: Conceptualization, Methodology, Formal Analysis, Writing – Original Draft, Project Administration.

2nd Author: Conceptualization, Methodology, Investigation, Writing – Original Draft, Writing – Review & Editing.

‘All authors reviewed the manuscript and provided final approval for publication’

Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024

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Received: 12/Sep/2024
Accepted: 16/Nov/2024
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Received: 12/Sep/2024
Accepted: 16/Nov/2024
Published: 30/Nov/2024