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HLA-G rs1632947 AA Genotype as a Predictive Biomarker for IVF Failure: A Case-Control Study in Iraqi Women

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ABSTRACT

Background: Human leukocyte antigen-G (HLA-G) plays a critical role in immune tolerance during pregnancy, and its genetic polymorphisms have been linked to reproductive disorders. **This study aimed** to investigate the association between HLA-G polymorphisms (rs1632947) and in vitro fertilization (IVF) outcomes in Iraqi women.

Methods: A case-control study was conducted involving 30 women with recurrent implantation failure (RIF) or recurrent pregnancy loss (RPL) and 30 fertile controls. Genotyping was performed using PCR-RFLP, HLA-G mRNA expression was quantified via qRT-PCR, and plasma soluble HLA-G (sHLA-G) levels were measured by ELISA. Statistical analyses included chi-square tests, Student's t-tests, and odds ratios (ORs).

Results: The rs1632947 AA genotype was significantly more frequent in infertile women (41%) compared to controls (10%) (OR = 13.75, p = 0.0178), with the A allele associated with increased risk (OR = 3.967, p = 0.0115). HLA-G expression was upregulated in infertile women (1.95-fold, p = 0.0442), particularly in GA heterozygotes (2.68-fold). Lower sHLA-G levels were observed in infertility cases, suggesting impaired immune tolerance.

Conclusion: The HLA-G rs1632947 AA genotype is a potential genetic marker for IVF failure, likely mediated through immune dysregulation. These findings support integrating HLA-G genotyping into IVF risk stratification and highlight avenues for immunotherapy-based interventions.

Keyword: *HLA-G, rs1632947, IVF failure, genetic polymorphism*

1. INTRODUCTION

Infertility can result from male and female causes alike. Thirty percent of cases have no known explanation for infertility, whereas thirty-five percent involve both male and female variables alone. In twenty percent of instances, infertility is caused by a mix of factors. Environmental and occupational variables, the effects of tobacco usage toxins, severe exercise, extremely high or extremely low weight, and young age of couples are other factors that raise the risk of infertility[1], [2].

When it comes to assisted reproductive technology (ART), in vitro fertilization (IVF) is one of the most widely used techniques to help the population of infertile people achieve childbirth. Over time, many aspects of IVF treatments have changed. A lot of research has been done to improve IVF results by taking into account its influencing factors. Despite this, there is still a lack of knowledge regarding the predictors of IVF outcomes, and overall pregnancy rates have only reached about 30% [3], [4].

IVF-assisted effective fertility (i.e., successful embryo implantation) remains rare; cohort studies found an average likelihood of 32.1 % and a chance of pregnancy ranging from 18.9% to 41.8%. [5], [6]. The human leukocyte antigen (HLA), sometimes referred to as the major histocompatibility complex (MHC) in humans, is a highly polymorphic gene complex that codes for cell surface molecules with the specific ability to display and identify both self and non-self-peptides. Over 200 loci have been found as part of the HLA complex, which is situated on the short arm of chromosome 6 along a 3 Mbp span. Numerous thousands of allelic variations of HLA molecules have been found through population surveys. These variations mostly impact the structure and makeup of their peptide-binding groove, which controls the peptide repertoire shown on the cell membrane[7], [8].

Three decades ago, extravillous trophoblasts (EVT) were revealed to contain a unique protein called HLA-G, which is

similar to the human leukocyte antigen (HLA). As of right now, HLA-G is known to be a crucial molecule that contributes

significantly to the development of fetal-induced maternal immunological tolerance. On human chromosome 6, HLA-G was initially identified in 1982 as an unusual HLA class I gene using Southern blot hybridization study [9], [10].

Not long after, the gene encoding HLA-G was originally designated HLA-6.0 when it was extracted from a genomic DNA library made from the B-LCL 721.11 cell line. DNA sequencing showed that HLA-G had 86% overall protein homology and a comparable exon/intron structure with the classical class I genes HLA-A, -B, and -C, but had a unique 5' regulatory element in its 5' flanking region[9], [11]. Functional investigations showed that HLA-G prevented the killing of decidual natural killer (dNK) cells in 1994. Two years later, it was discovered that HLA-G also suppressed the cytotoxic activity of peripheral natural killer (pNK) cells[12], [13]. A "universal" HLA-G receptor on NK cells for all KIR haplotypes was identified in 1999 for the killer immunoglobulin-like receptor 2DL4 (KIR2DL4) [14]. The groundwork for further investigations into the function of HLA-G in developing maternal-fetal immunological tolerance in the early stages of pregnancy was laid by these pioneering investigations[15].

Because of alternative splicing of its transcript, the HLA-G gene produces either soluble or membrane-bound proteins: HLA-G5 to HLA-G7 are soluble proteins, whereas HLA-G1 to HLA-G4 are membrane-bound. Human embryonic stem cells, human oocytes, amniotic fluid, preimplantation embryos, trophoblasts, and maternal-fetal circulation were found to contain soluble isoforms. Additionally, HLA-G expression varied as the blastocyst developed [16], [17].

Amniotic fluid, cord blood, and maternal blood all contain soluble HLA-G (sHLA-G). Recurrent spontaneous abortion (RSA) and preeclampsia (PE) are two unfavorable pregnancy outcomes that are linked to aberrant expression and polymorphisms of HLA-G. Here, we provide the most recent research on the three primary functions of HLA-G during pregnancy, which are the stimulation of spiral artery remodeling, immunological tolerance, and fetal development, all of which are brought about by the protein's interaction with immune cells[18].

The HLA-G gene has eight exons and seven introns. It is found on chromosome 6p21.3. The HLA-G gene has limited tissue expression and modest allelic polymorphism in contrast to the highly variable traditional HLA Ia genes (HLA-A, B, and C).

Rather than only single polymorphisms in the 3'-UTR, 5'URR, or coding areas, the combined HLA-G genetic influence is more likely to be responsible for the link between HLA-G and RIF. The 14 bp insert, together with the +3187A/A and +3142G/G SNP, is important for the regulation of HLA-G mRNA in human endometrial stromal cells (ESCs) [19]

2. MATERIALS AND METHODS

The specimens samples collection and the practical work of this study extended through twelve months from Jan. 2023 until April 2024 This study was approved by the Ethics Committee of the Department of Biotechnology, Monastir University – Faculte de Medecine Ibn El Jazzar de Sousse, Tunisia, as did the Iraqi Ministry of Health .Kamal Al-Samarrai & Al-Alamy Hospital , IVF Lab, before they were included in the study tests. A signed written consent was obtained from each individual participating in the study. The study was intended to be a prospective one. This investigation comprised Abortion and non-Abortion females of various ages and regions of Iraq. The patient group consists of three groups (G1 , G2 & G3) and control group . They were picked from the Al-Alamy Private Laboratory in Baghdad, Iraq. G1 for patients its consist of Triozol (20) femals, G2 contain TE solution (20) females , G3 it’s without any addition also 20 females, as well as 40 healthy . All of the patients and the control group are between the ages of 20 and 51.

Inclusion criteria

These patients were considered as cases with recurrent spontaneous miscarriage if they had the following criteria:

Three or more recurrent miscarriage before 12 weeks conception.

Exclusion criteria

1- Chromosomal abnormalities in either partner.

2- Uterine anomalies, endocrine disorders (e.g., PCOS, thyroid dysfunction), or infections.

3- Patients with thyroid dysfunction abnormal low or high T3, T4 and /or TSH).

4- Patients with uterine surgery before recurrent pregnancy losses (Cesarean section or previous myomectomy ……etc).

5- Diabetes mellitus (Known from the history)

Group 1: It includes 20 females with Triozol . **Group 2**: It includes 20 females with TE solution . **Group 3**: It includes 20 females without any addition. **Group 4**: It contains 40 control-healthy females. They were chosen from among the healthy females with at least one healthy child and no history of infertility; their ages were similar to those in groups 1, 2, and 3. It is referred to as a control group.

Sample Collection and Processing: RNA Extraction: Total RNA was isolated from follicular fluid using TransZol Up

Plus RNA Kit, followed by chloroform phase separation and ethanol precipitation.

cDNA Synthesis: Reverse transcription was performed using the EasyScript® One-Step gDNA Removal and cDNA Synthesis SuperMix Kit. **DNA Extraction:** Genomic DNA was extracted using the EasyPure® Genomic DNA Kit.

Molecular Techniques: qRT-PCR: HLA-G and GAPDH (reference gene) expression were quantified using TransStart® Top Green qPCR Super Mix on a QIAGEN Rotor-Gene Q system. Fold changes were calculated via the $2^{-\Delta\Delta Ct}$ method.

Genotyping: SNPs rs1632947 was analyzed by PCR-RFLP and Sanger sequencing (ABI3730XL).

ELISA: Soluble HLA-G (sHLA-G) levels were measured using a biotinylated antibody-based ELISA.

Statistical Analysis

Data were analyzed using SPSS v22. Hardy-Weinberg equilibrium, chi-square tests, and odds ratios (ORs) assessed genotype/allele frequencies. Student's t-tests compared gene expression and sHLA-G levels ($p < 0.05$ significant).

Primers: Designed via Primer 3 plus and synthesized by Alpha DNA (Canada).

3. RESULTS AND DISCUSSION

HLA-G Genetic Variation

One intergenic variants (SNPs: rs1632947 G/A) of *HLA-G* gene were investigated.

rs1632947 G/A: The analysis of Hardy-Weinberg equilibrium (HWE) indicated no significant differences between the observed and the expected genotype frequencies in patients and control, demonstrated a strong agreement with equilibrium conditions (Table 1). The heterogenous genotype GA showed no significant variation between patients and control (44% vs. 40%, $p = 0.1266$), however the rate of minor homogenous AA genotype was significantly ($p = 0.0178$) higher in the patient compared to control, 41% and 10%, respectively (Table 2). The corresponding odd ratio of genotype variations were 3.8 (95% CI: 0.6588 to 21.88) and 13.75 (95% CI: 1.474 to 173.0) for GA and AA, respectively.

The allele frequency of *HLA-G* SNP rs1632947 exhibited a significant variation in allele frequency between patients and control with respect to G/A alleles and with *p* value of 0.0115. The frequency of the wild G allele was 0.4 in patients *vs.* 0.7 in control, conversely the frequency rate of the recessive allele A was highly increased in the patient group (0.6 vs. 0.3) compared to control.

Table 1 : Hardy-Weinberg equilibrium (HWE) analysis of *HLA-G* gene SNP rs1632947 among patients and controls.

		Control		*p*-value	Patients		*p*-value
		Observed	Expected		Observed	Expected	
rs1632947	**GG**	5 (50%)	5 (50%)	>0.9999 NS	4 (15%)	3 (11%)	0.9126 NS
	GA	4 (40%)	4 (40%)		12 (44%)	13 (48%)	
	AA	1 (10%)	1 (10%)		11 (41%)	11 (41%)	

Table 2 : Allele and genotype frequencies of *HLA-G* gene SNP rs1632947 among patients and controls.

Genotype Frequency (%) HLA-G *rs1632947*						
Genotype	**Control n=10**	**Patients n=27**	**P-value**	**Chi-square**	**Odds Ratio**	**95% Cl**
GG	5 (50%)	4 (15%)	----	----	1.0	----
GA	4 (40%)	12 (44%)	0.1266 NS	2.334	3.8	0.6588 to 21.88
AA	1 (10%)	11 (41%)	0.0178 *	5.619	13.75	1.474 to 173.0
Chi-square	0.0227 NS	0.05979 NS				
***p* Value**	0.9887	0.9705				

Allele frequency (%)						
Allele	**n=20**	**n=54**	**P-value**	**Chi-square**	**Odds Ratio**	**95% Cl**
G	0.7 (14)	0.4 (20)	0.0115 *	6.385	3.967	1.247 to 12.51
A	0.3 (6)	0.6 (34)				

NS = Non-significant, * significant at *p* value < 0.05.

***HLA-G* Gene Expression**

The expression of *HLA-G* gene in infertility patients was quantitatively investigated using reverse transcriptase polymerase chain reaction (RT-qPCR) compared with control. The level of HLA-G mRNA was calculated by determining the fold of change in patient group. Results in Fig. (1) indicated that the expression of HLA-G was significantly (p = 0.0442) upregulated with fold of change of 1.95±0.78.

HLA principally acts as immunosuppressive molecule that protects semi-allogenic fetal tissue against maternal immunological rejection. HLA suppress natural killer cells, T-lymphocytes and Ag-presenting cells by contact with inhibitory receptors including ILT2, ILT4, and KIR2DL4. Under typical physiological conditions, HLA-G expressions adhere to strictly controlled temporal and spatial patterns, exhibited peak expression in extravillous trophoblast during early pregnancy, followed by modulation throughout gestation.

The overexpression of *HLA-G* found in infertile patients prompt significant inquiries on immunological dysregulation in reproductive failure. Several mechanisms may be attributed to the upregulation of HLA-G in infertility patients. Epigenetic modification of HLA-G promoter has been seen in endometrial tissues of females experiencing infertility and recurrent implantation failure. These modifications (hypomethylations) may result in transcriptional upregulation and enhanced protein synthesis. Most importantly, polymorphisms in the HLA-G promoter region and untranslated region (3'UTR), highly affect mRNA stability and translation efficiency, potentially leading to abnormal expression levels.

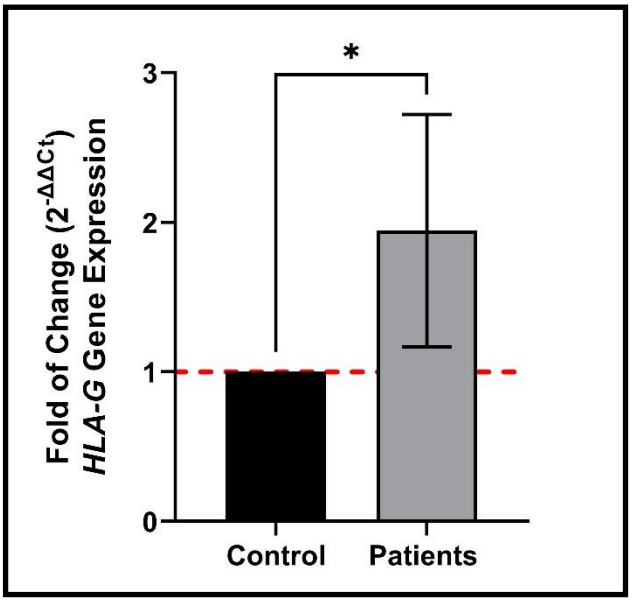

Figure 1 : Mean (±SD) fold of change for *HLA-G* gene expression quantitatively determined by RT-qPCR. * *p* < 0.05.

Although HLA-G generally facilitates immunological tolerance at the maternal fetal interface, the excessive expression of HLA-G affects the regulated inflammatory response needed for endometrial receptivity, disrupting the immunological balance needed for successful implantation. By dysregulating VEGF and MMP synthesis, elevated HLA-G levels can hinder trophoblast invasion and spiral artery remodeling. This hinders placental vasculature development, which is vital to embryo survival. HLA-G overexpression may also affect decidual immune cell recruitment and function which promote implantation. Immune cell dysfunction can impair blastocyte adhesion and invasion by reducing growth factor and cytokine release.

Genotype Impact on *HLA-G* Expression

The impact of *HLA-G* genotype variation in rs1632947 SNPs on *HLA-G* gene expression was investigated. Results in Fig. (2) and Table (3), show *HLA-G* gene expression across the three different genotypes (GG, AG and AA) of rs1632947.

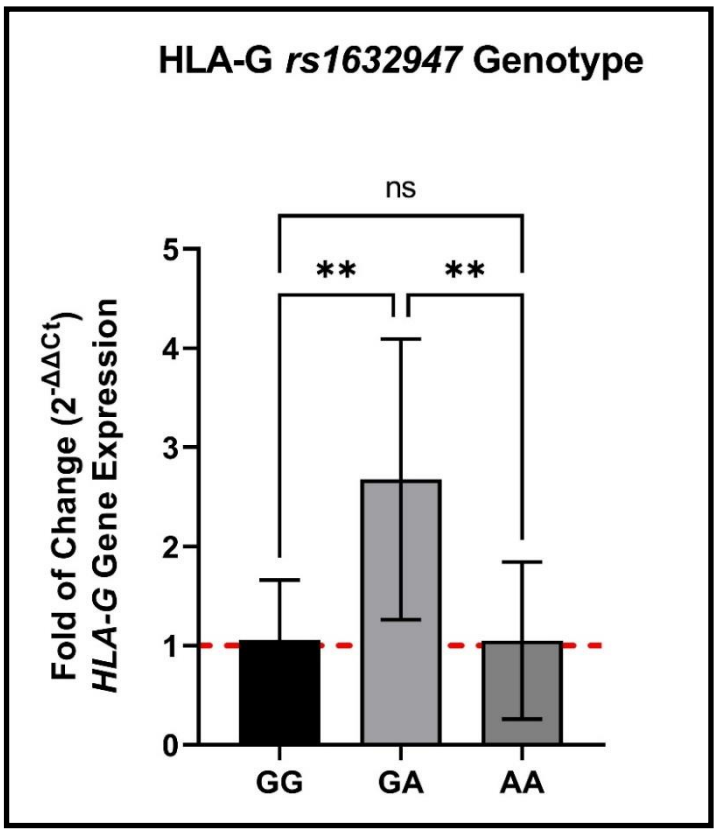

Figure 2 : Mean (±SD) fold of change for *HLA-G* gene expression quantitatively determined by RT-qPCR and distributed according to genotype of rs1632947 SNP. ** $p < 0.01$, NS: Non-significant.

Table (3): Mean ± SD fold of change for HLA-G gene expression based on genotype distribution in rs1632947 and rs1707 SNPs.

***HLA-G* Mean ± SD Fold of Change (rs1632947)**			**Sig.**	***p* Value**
GG	**GA**	**AA**		
1.06 ± 0.59a	2.68 ± 1.42b	1.05 ± 0.79a	**	0.0003

**** $p < 0.01$.**

4. DISCUSSION

The HLA-G gene, located at the short are of chromosome 6 within major histocompatibility complex (MHC) and encodes for human leukocyte antigen-G protein which essential for immunological tolerance at maternal – fetal interface [20]. HLA-G, a non-classical HLA class 1 molecule, demonstrated less polymorphism relative to conventional HLA molecule, yet variations, such as the intergenic SNP rs1632947 A/G have attracted interest for possible correlation with reproductive outcomes. The results of genotype and allele frequency show that the investigation of different loci in the HLA-G gene as a risk marker for infertility did not yield the anticipated findings. Only the AA genotype in the promoter rs1632947 was associated with protection against infertility across patient and control groups.

Nowak et al, were reported that haplotypes and diplotypes of different SNPs including rs1632947 G/A were associated with infertility. The study included 389 female patients suffered from different forms of infertility. Compared to control, patients exhibited reduced frequency of genotype AA in infertility patients (p = 0.025) compared to control [21] . In contrast, a study from Iran included 90 patients with recurrent spontaneous abortions showed that the frequency of minor genotype AA was highly increased in patient (12.23%) compared to control (3.33%) [22] . Along with results obtained, Berger et al., investigated the correlation between rs1632947 G/A and recurrent spontaneous abortion in Caucasian women. A positive correlation was confirmed between the minor A allele and recurrent spontaneous abortion which highly

corroborates the results obtained [23] . The odd ratio (5.619) for the recessive AA genotype in this study suggests a strong association with infertility.

The rs1632947 SNP in the promoter region of HLA-G, around 964 pb upstream of the transcription start point [24] . This site is important because promotor polymorphisms can influence gene expression levels which may impact the immunological tolerance processes critical for successful implantation and the maintenance of pregnancy [25] . The rs1632947 genotype may affect HLA-G mRNA expression levels and soluble HLA-G protein concentrations in peripheral blood and reproductive organs. Based on previous studies, it was indicated that the A allele is associated with a heightened risk of recurrent spontaneous abortion and implantation failure in IVFs treatments [26] .

SNPs in HLA-G gene can modulate the expression of HLA-G gene and may influence fertility. Several case-control and meta-analysis studies across various ethnic and population groups have investigated the association of HLA-G gene SNPs with susceptibility to infertility; however, some findings remain unreplicated. Regarding rs1632947 SNP genetic association analysis suggests that GA genotype influence the expression of HLA-G gene by upregulating the gene and might have susceptibility role in infertility. Variation in HLA-G expression has been shown to correlate with different assisted reproductive techniques, indicating that procedural choices may modulate the effects of SNP rs1632947 on implantation success [21] . Additionally, emerging evidence suggests that maternal immune tolerance mechanisms, mediated by HLA-G expression, could be significantly affected by both genetic polymorphisms and external stimuli such as hormonal treatments during IVF cycles [27] . Results of Aljumaili et al., demonstrated a significant variation in the frequency of G allele for SNP rs1632947 correlated with the level of HLA-G [24] . Moreover, specific HLA-G alleles or polymorphisms were linked to HLA-G in IVF couples. HLA-G polymorphisms in various ethnicities may linked to recurrent implantation failure [26] . In contrast, polymorphisms in rs1707 SNP, mainly the CA and AA alleles, affect the expression of HLA-G by downregulating the mRNA production of HLA-G which in infertility patients. A local study by Merdas et al., investigating the impact of different SNPs in HLA-G gene on the serum level of HLA-G protein, which they confirmed that serum HLA-G levels in patients showing HLA-G polymorphism and experiencing threatened abortion are significantly lower compared to those in normal pregnant women [25]

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