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Original article

Association between Interleukin-32 Gene Polymorphism (rs4786370) and the Susceptibility to Preeclampsia in Egyptian Pregnant Women

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Abstract:

Background: Preeclampsia (PE) is recognized as a major global health concern, with a disproportionately higher impact in developing countries. It remains a significant preventable source of maternal and neonatal morbidity and mortality. In Egypt, the prevalence of PE is estimated at 6%–8% of pregnancies, reaching up to 15% in university-affiliated referral hospitals. **This study aimed to** investigate the potential correlation between IL-32 rs4786370 gene polymorphism and the risk of developing PE in Egyptian pregnant women. **Methods:** The present study enrolled 70 cases with PE from Benha University Hospitals, Egypt. In addition to 30 apparently healthy pregnant women served as control, IL-32 gene (rs4786370) genetic variant was investigated by PCR followed by restriction fragment length polymorphism (RFLP) with restriction enzyme PagI. Amplified purified PCR Product was confirmed by Sanger Sequencing. **Results**: A significant association of CT genotype and TT genotype with the risk to develop PE and T allele was also linked to an elevated risk for PE in comparison to C allele. **Conclusion:** IL-32 rs4786370 gene polymorphism can be regarded as a potential risk factor for PE in Egyptian pregnant women.

Keywords: IL-32, Egyptian, Preeclampsia, rs4786370, Restriction Fragment Length Polymorphism.

Introduction

Preeclampsia (PE) is regarded as one of the principal medical conditions within this disease spectrum and has been associated with numerous documented gestational complications [1]. Per International Society for the Study of Hypertension in Pregnancy (ISSHP) guidelines, PE is diagnosed when new-onset hypertension (HTN) appears after 20 weeks of gestation in association with proteinuria or evidence of impairment in other organ systems. Eclampsia is manifestation of grand seizures in a woman affected by PE [2].

Such conditions are identified after twenty weeks of gestation when accompanied by proteinuria or generalized edema. These may also involve hematologic alterations such as thrombocytopenia. End organ complications which may be present are renal or liver dysfunction, pulmonary edema, and disturbances of the CNS i.e. cerebral or visual [2]. A range of maternal and fetal gene alleles and mutations have been involved in pre-eclampsia [3]. Several of these genes are related to thrombophilic factors [4], angiogenic factors [5], or pathways involved in immune responses [6].

IL-32 was first characterized in 1992 as a 27-kDa human protein expressed predominantly in activated natural killer (NK) and T lymphocytes. At the time of its discovery, it was referred to as NK4 because of its selective expression in IL-2–stimulated NK cells [7]. Since its identification, IL-32 has emerged as a potent pro-inflammatory cytokine exhibiting a wide array of biological activities that influence immune regulation and host defense [7].

Over the past decades, IL-32 has been intensively investigated in the context of numerous pathological conditions, including infectious, autoimmune, neoplastic, and chronic inflammatory diseases [8]. The human IL-32 gene is located on chromosome 16p13.3 and gives rise to multiple alternative mRNA splice variants, of which IL-32α, IL-32β, IL-32γ, and IL-32δ represent the major isoforms [9]. Depending on the cell type and the nature of the stimulating signal, IL-32 can either be released passively during necrotic cell death or actively secreted through vesicle-associated pathways, particularly via exosomes [10].

Functionally, IL-32 acts as a strong inducer of pro-inflammatory mediators, stimulating the production of cytokines such as TNF-α, IL-6, MIP-2, IL-8, and IL-1β, underscoring its pivotal role in the orchestration of inflammatory cascades [11]. In turn, IL-32 expression can be upregulated by several other cytokines, notably IL-1β, IL-18, IL-12, and TNF-α [12]. Marked elevations in IL-32 levels have been documented in various cell populations—such as adipocytes, primary human keratinocytes, CD4+ T lymphocytes, fibroblast-like synoviocytes, and monocyte-derived dendritic cells—when exposed to TNF-α stimulation [13]. Genetic variability further contributes to IL-32 regulation. Among the most studied single nucleotide polymorphisms (SNPs) within the IL-32 gene are rs4786370 and rs9927163. The rs4786370 variant, positioned within the promoter region, has been linked to enhanced transcriptional activity and elevated IL-32 gene expression [14]. The objective of current investigation was to detect any possible correlation between IL-32 rs4786370 gene polymorphism and PE among Egyptian pregnant women, and to analyze how this polymorphism relates to clinical symptoms and laboratory measurements.

Patient and Methods

Patients:

From September 2022 through September 2023, this case-control study was undertaken in Clinical and Chemical Pathology Department at Benha University Hospitals, Egypt. The preeclamptic cases were attending antenatal care clinic at Benha University Hospitals or admitted to emergency department for suspected PE or referred from other hospitals for further evaluation at Benha University Hospitals in Egypt. Ethical clearance was granted by

local Ethics Committee of Benha University, Egypt (**approval number MD 15-7-2022**). Informed consent was taken.

Seventy pregnant women with PE were recruited in accordance with ACOG guideline [15], which requires ≥0.3 g protein in a 24-hour urine specimen and two separate BP readings >140/90 mmHg. Severe PE was defined by DBP ≥110 mmHg, SBP ≥160 mmHg, or proteinuria ≥5 g/24 h. The control group comprised 30 apparently healthy pregnant women matched to cases by age and gestational age. All participants underwent clinical assessment, and their medical history was documented.

Sample size

Sample size estimation was conducted using the Quanto software (v1.2.4) designed for sample size and power analysis in genetic association studies. Parameters applied included a minor allele frequency of 0.194, an OR of 2.142 as described by Mazlum et al. [16], and a PE prevalence of 0.03% according to Getaneh et al.[17]. With 80% power and $\alpha = 0.05$, the minimal calculated sample size required for this case–control design was 74 participants. To ensure greater statistical robustness, the cohort was expanded to 100 subjects, comprising 70 PE cases and 30 healthy controls.

Inclusion criteria:

Eligible participants were pregnant women between 32 and 35 weeks of gestation who satisfied the diagnostic thresholds for PE based on the 2018 ACOG criteria. PE was defined by the occurrence of new-onset HTN (SBP ≥ 140 mmHg or DBP ≥ 90 mmHg) after 20 weeks in previously normotensive women accompanied by proteinuria (≥ 0.3 g/24 h). In the absence of proteinuria, the diagnosis was established when HTN was associated with one or more severe manifestations, including thrombocytopenia (platelets < 100 × 10^9/L), hepatic dysfunction with transaminases ≥ 2× the upper normal limit, or renal impairment defined by serum creatinine > 1.1 mg/dL or a twofold increase from baseline not attributable to other renal causes.

Exclusion criteria:

Participants were excluded if they had a history of HTN, a confirmed diagnosis of cancer, pre-existing hepatic or renal disorders, immunological disorders, or other medical comorbidities, including heart diseases.

Methods:

Sample collection

Blood sample

For each participant, a sterile blood collection was performed from peripheral veins. The collected blood was then separated into three parts. Whole blood was taken in an EDTA tube then divided into 2 aliquots one was used for: CBC and other was stored at -80ºC for later analysis by RFLP- PCR to detect IL-32 (rs4786370) polymorphism. To perform coagulation tests, a sample was mixed with 3.2 mg/ml of sodium citrate in a tube. Blood was allowed to coagulate via leaving sample for 30 min in a plain tube at room temperature and then sample spun at a high speed in a centrifuge to extract serum. Renal function tests and liver function tests were performed on obtained samples.

Urine sample

A 24-hour urine sample was collected for protein estimation, with container stored in a refrigerator throughout collection period. Additionally, a random midstream urine sample, preferably a morning specimen, was collected in a sterile container to measure urine protein and creatinine concentrations. UPCR was calculated by taking ratio of urine protein concentration (mg/L) to urine creatinine concentration (mg/L), multiplying ratio by 10, and expressing result as mg/mg without units.

Lab tests

The complete blood counting was conducted using an automatic cell counter by using (sysmex XN-550, Japan). The coagulation profile—comprising PT, INR, and Activated partial thromboplastin

time—was processed using CS Sysmex system (Japan). Measurement of liver function tests and renal function tests was carried out on Dialab auto-analyzer (Austria).

Molecular determination of IL-32 (rs4786370) SNP by RFLP-PCR method.

To obtain high-quality DNA from a sample of blood GF-1 Whole Blood Genomic DNA Extraction Kit was used to extract DNA from EDTA anticoagulated blood (Cat. No GF-BD-100, Lot No .40308-23). PCR amplification was carried out on a Thermal Cycling Block (Applied Biosystems; serial number 271003648). The following primers were used for amplification reactions. Forward primer: 5′TTGCATTGCCTGTAAATTGC3′ and reverse
primer :5′AACCGCCTTTCCACATACA G3′.

For PCR amplification, a total reaction mixture of 50 µl was prepared, containing 3.0 µl of genomic DNA template and 2.5 µl each of forward and reverse primers. The amplification protocol commenced with an initial denaturation step at 95 °C for 2 min, followed by 35 consecutive cycles consisting of denaturation at 92 °C for 15 s and annealing/extension at 57 °C for 20 s. The amplified fragments were subsequently separated by electrophoresis on a 2% agarose gel. Genotyping of the IL-32 (rs4786370) variant was achieved using the PCR–RFLP technique, employing the restriction enzyme PagI (BspHI) (Thermo Scientific). The digested PCR products were resolved on a 2% agarose gel stained with ethidium bromide (**Figure 1)**.

Visualization was performed under UV illumination using an Alpha Innotech Corporation trans-illuminator to detect DNA fragments corresponding to 64, 117, and 172 bp. Genotypes were determined according to the banding patterns observed: the homozygous wild-type (CC) genotype displayed two distinct fragments at 64 bp and 117 bp (lanes 2, 8, 9); the heterozygous (CT) genotype showed three fragments at 64, 117, and 172 bp (lanes 1, 4, 5, 7, 10); and the mutant homozygous (TT) genotype presented a single band at 172 bp (lanes 3, 6). The leftmost lane contained the PCR molecular weight marker (50–1000 bp ladder).

Sanger sequencing

Detection of polymorphisms and confirmation of genotyping accuracy were achieved by Sanger sequencing of purified DNA. Sequencing of PCR products was carried out on an ABI (Applied Biosystems) 3500 Genetic Analyzer using Big Dye Terminator Reaction Kit version 3.1 as per manufacturer's protocol. Sequence reads were analyzed using BLAST (www.ncbi.nlm.nih.gov) and CLC-BIO Workbench 7.7 software (www.clcbio.com). Variant interpretation included database comparison with NCBI dbSNP and HGMD, and variant nomenclature was reported following Human Genome Variation Society (HGVS) criteria.

Statistical Analysis:

All analyses were carried out using SPSS software, version 25.0 (IBM Corp., Armonk, NY, USA). The relationship between the IL-32 (rs4786370) polymorphism and susceptibility to PE was evaluated through calculation of ORs and their corresponding 95% CIs. Continuous variables were summarized as mean ± SD and range. Comparisons of means between two independent groups were performed using the Student's *t*-test, whereas non-normally distributed data were analyzed with the Mann–Whitney *U*-test. Categorical variables were assessed using the Chi-square or Fisher's exact test when appropriate. A two-tailed P value < 0.05 was considered statistically significant within the 95% CI framework.

Results

This study enrolled 90 cases who attended Among 70 pregnant women with PE, majority were multigravida (51.4%). The mean age was 27.37 ± 3.32 years, and mean gestational age was 34.37 ± 1.81 weeks.

Cases with PE demonstrated a substantially higher BMI (28.22 ± 2.09 kg/m²) than controls (21.03 ± 4.26 kg/m²) ($p < 0.001$). SBP was also elevated in cases group, averaging 157.7 ± 16.75 mmHg versus 112.8 ± 6.01 mmHg in controls ($p < 0.001$). Likewise, DBP was markedly higher in cases (104.4 ± 14.59 mmHg) compared with controls (72.13 ± 3.47 mmHg) ($p < 0.001$). **Table 1**

The rs4786370 CT genotype was notably more common in cases (60.0%) than controls (50.0%) with P<0.044.Additionally, TT genotype increased possibility of developing PE.Both groups demonstrated a markedly different allelic distribution P<0.008.A higher PE risk was common with T allele than C allele (P<0.008). **Table 2**

The IL-32 (rs4786370) genotype frequencies differed substantially between the two groups. The CT and TT genotypes were more common in severe PE group than in the mild group (CT: 67.9% vs. 54.8%; TT: 25.0% vs. 14.3%). The CT, TT, CT+TT genotypes and the T allele were markedly correlated with an elevated risk of severe PE ($p < 0.05$). **Table 3**

After Sanger sequencing, polymorphism was detected according to the primer design as demonstrated in **Figure 2** at rs in rs 4786370.

IL-32 Gene: Location of (rs4786370):

Table 1: Demographic and clinical features of pre-eclamptic and healthy women.

	PE n = 70		**Control n = 30**		**Test**	**p**
Age (years)						
Mean ± SD.	27.37 ± 3.32		26.27 ± 3.49		t=1.500	0.137
Min. – Max.	21.0 – 35.0		20.0 – 32.0			
Gravidity	No.	%	No.	%		
Primigravida	34	48.6	12	40.0	X2=0.621	0.431
Multigravida	36	51.4	18	60.0		
Gestational age (week)						
Mean ± SD.	34.37 ± 1.81		35.07 ± 2.74		t=1.275	0.210
Min. – Max.	32.0 – 38.0		31.0 – 39.0			
BMI (kg/m2)						
Mean ± SD.	28.22 ± 2.17		21.03 ± 4.26		t=11.189*	**<0.001***
Min. – Max.	23.92 – 31.40		1.06 – 24.80			
SBP (mm/Hg)						
Mean ± SD.	157.7 ± 16.75		112.8 ± 6.01		t=19.685*	**<0.001***
Min. – Max.	135.0 – 193.0		105.0 – 129.0			
DBP (mm/Hg)						
Mean ± SD.	104.4 ± 14.59		72.13 ± 3.47		t=17.366*	**<0.001***
Min. – Max.	81.0 – 129.0		66.0 – 78.0			

SD.: Standard deviation, Min.: Minimum, Max.: Maximum. X^2: Chi Square, t: Student t test, p: Comparing cases and control. *: P value Significant <0.05.

Table 2: Genotypic and allelic frequencies for IL-32 (rs4786370) gene among preeclampsic cases and control group.

IL-32 (rs4786370) gene polymorphism		PE n = 70		Control n = 30		P value	OR (95 % CI)
		No.	%	No.	%		
Genotypes	CC	15	21.4	14	46.7		Reference
	CT	42	60.0	15	50.0	**0.044***	1.805(1.015–3.208)
	TT	13	18.6	1	3.3	**0.011***	4.145(1.395–12.323)
Dominant model	CC	15	21.4	14	46.7		Reference
	CT + TT	55	78.6	16	53.3	**0.014***	2.014(1.150–3.528)
Recessive model	CC + CT	57	81.4	29	96.7		Reference
	TT	13	18.6	1	3.3	**0.044***	2.875(1.030-8.026)
Alleles	C	72	51.4	43	71.7		Reference
	T	68	48.6	17	28.3	**0.008***	1.682(1.147–2.467)

*: P value Significant <0.05. C, Cytosine; T, Thymine; OR>1 is considered risky; OR<1 is considered protective

Table 3: Genotype & allele distribution for rs 4786370 in mild and sever form of the disease.

IL-32 (rs4786370) gene polymorphism		Degree of preeclampsia Mild n = 42		Severe n = 28		P value	OR (95 % CI)
		No.	%	No.	%		
Genotypes	CC	13	31.0	2	7.1		Reference
	CT	23	54.8	19	67.9	**0.024***	2.770(1.141–6.724)
	TT	6	14.3	7	25.0	**0.024***	3.345(1.169–9.566)
Dominant model	CC	13	31.0	2	7.1		Reference
	CT + TT	29	69.0	26	92.9	**0.016***	2.899(1.219–6.894)
Recessive model	CC + CT	36	85.7	21	75.0		Reference
	TT	6	14.3	7	25.0	0.284	1.515(0.708–3.239)
Alleles	C	49	58.33	23	41.07		Reference
	T	35	41.67	33	58.93	**0.046***	1.541(1.009–2.354)

*: P value Significant <0.05.

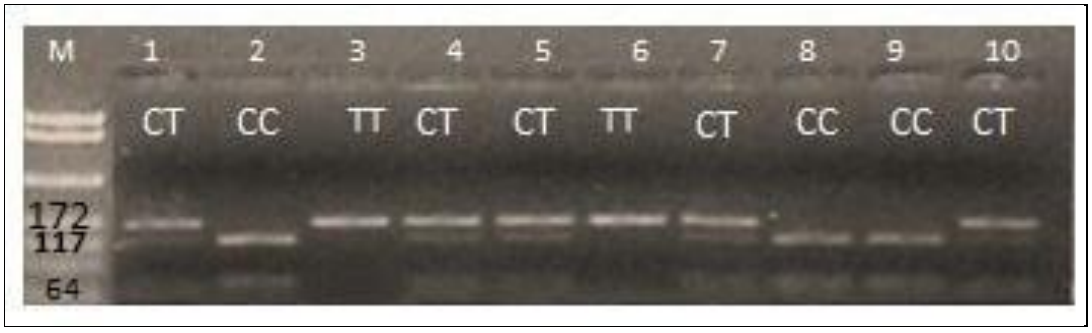

Figure 1: Agarose Gel Electrophoresis after digestion with PagI (BspHI) restriction enzyme for IL-32 rs 4786370.

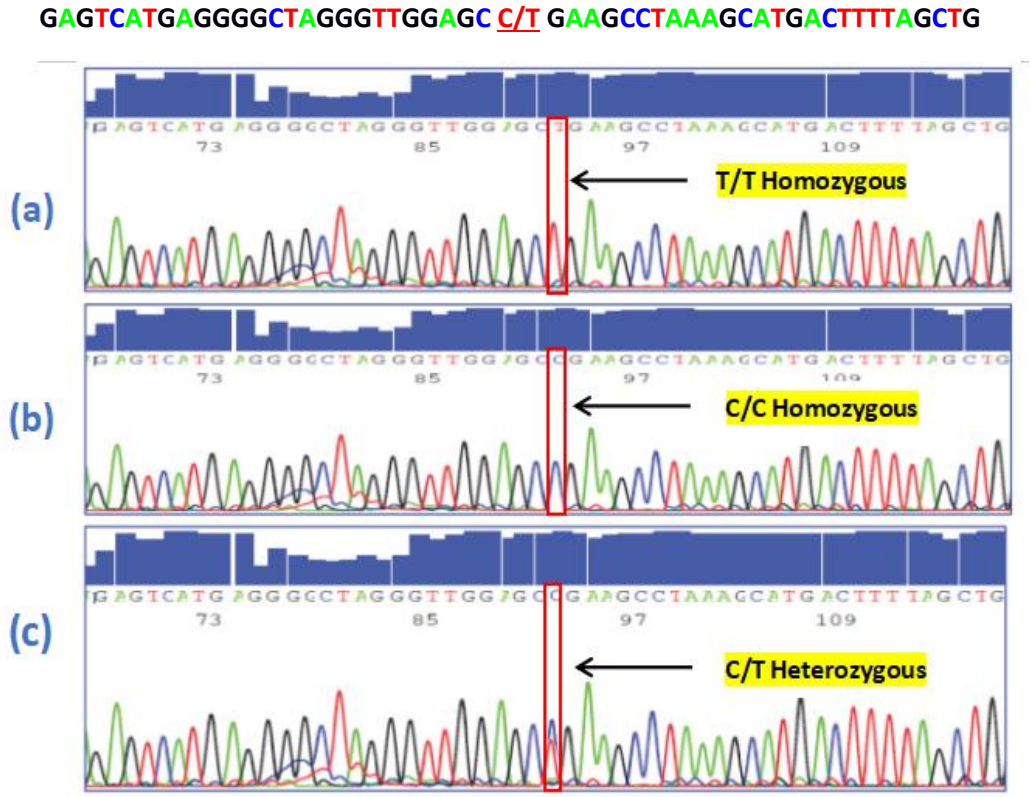

Figure 2: Chromatogram analysis by Sanger sequencing using the Forward Primer showing genotyping of the IL-32 rs4786370 SNP. The target SNP site is marked with a black arrow and highlighted in red. (a) Homozygous T is identified by a red peak; (b) Homozygous C by a blue peak; (c) Heterozygous C/T by overlapping red and blue peaks at the same nucleotide position, indicating the presence of both alleles.

Discussion

Evidence from prior studies suggests that cytokine-mediated immune imbalance plays a role in pathogenesis of PE. Women with PE typically exhibit heightened Th1-type immunity and reduced Th2 response profiles, particularly in second and third trimesters [18]. Moreover, IL-32 has been recognized as a key mediator in diseases marked by abnormal inflammatory activity, such as IBD, COPD, and RA [19].

To elucidate genetic involvement in PE, extensive research has evaluated SNPs in cytokine-encoding genes and their receptors as potential candidate markers. Cytokines originate from various cell types and serve as critical modulators of cell and tissue growth, maturation, differentiation, and immune regulation.

The involvement of IL-32 has been documented in numerous autoimmune and inflammatory disease processes, including PCOS [20].

Beyond immune system functions, genetic variations are critical contributors to disease susceptibility. Therefore, current study focused on exploring correlation between pre-eclampsia and IL-32 gene polymorphism.

Evaluation of IL-32 (rs4786370) gene polymorphism among study cohort showed a significant correlation of CT genotype, TT genotype, and T allele with PE. These findings suggest that polymorphism may contribute to development of disorder.

IL-32 gene polymorphism rs4786370 has been documented as having significant associations with multiple immunological disorders [21].

Concordant with our results, Mazlum et al. [16] found that IL-32 rs4786370 C/T SNP displayed significant genotype and allele frequency differences between PE cases and healthy controls. Their study reported that CT and TT genotypes, as well as T allele, were more prevalent in individuals with PE.

A substantial correlation was detected between IL-32 CT and TT genotypes and presence of clinical complications. Among individuals with CC genotype, 80.0% showed no complications. Conversely, complications were more frequent in cases carrying CT genotype (66.7%) and TT genotype (53.8%).

Mazlum et al. [16] investigated influence of IL-32 rs4786370 polymorphism on PE and observed significant variations in allele and genotype frequencies between cases and controls. CC genotype was more prevalent in control cohort, suggesting a protective role against PE, whereas CT and TT genotypes were more frequently detected in affected women, indicating increased susceptibility. They also reported a notably higher frequency of allele C (66.2%) in healthy controls, supporting its protective effect, while allele T appeared to confer a higher risk of developing PE.

The Sanger dideoxy chain termination reaction is considered established standard for DNA sequencing. Despite progressive enhancements throughout nearly thirty years of implementation, its use is still challenged by expense of sequencing platforms and labor-intensive nature of sample preparation [22].

Contemporary Sanger sequencing incorporates fluorescently labeled dideoxynucleotides that are visualized by laser detection following capillary electrophoresis, resulting in a chromatogram with fluorescent peaks that correspond to ddATP, ddCTP, ddGTP, and ddTTP. The method continues to serve as a standard technique for detecting recurrent single nucleotide mutations and small insertions/deletions in genes [23].

Conclusions

The IL-32 rs4786370 gene polymorphism may serve as a potential marker for predicting PE risk in Egyptian pregnant women. The TT and CT genotypes were more frequent in severe PE compared with CC genotype. These findings may help identify women with increased susceptibility to PE and contribute to future preventive and control strategies.

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