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ORIGINAL ARTICLE

Serum Anti-PLA2R Antibodies and *PLA2R1* Gene Polymorphisms in a Sample of Iraqi Patients With Membranous Nephropathy

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Abstract

Background/Aim: Membranous nephropathy (MN) is a leading cause of nephrotic syndrome in adults. Anti-phospholipase A2 receptor (anti-PLA2R) antibodies and *PLA2R1* variants are associated with primary MN, but data from Iraq are limited. This study compared biochemical parameters, serum anti-PLA2R levels and *PLA2R1* variants in Iraqi patients with MN and healthy controls.

Methods: Laboratory measurements were reported for 50 patients with MN undergoing maintenance haemodialysis and 50 apparently healthy controls. Patient samples were collected before dialysis at Al-Kindy Teaching Hospital, Baghdad, Iraq. Complete blood count and biochemical parameters were measured using automated analysers and serum anti-PLA2R levels were measured by sandwich enzyme-linked immunosorbent assay. DNA from 25 patients and 25 controls was analysed by polymerase chain reaction and sequencing.

Results: Patients had lower serum albumin, calcium, iron and haemoglobin and higher creatinine, urea, alkaline phosphatase, phosphate, ferritin, bilirubin and anti-PLA2R levels (all $p < 0.05$). Anti-PLA2R levels differed between groups ($p < 0.001$), with an area under the receiver operating characteristic curve of 1.000, but were not significantly correlated with creatinine ($r = -0.12$, $p = 0.411$). The 33,988 (G>A) variant had a reported odds ratio (OR) of 6.78 ($p = 0.041$), while 34,046 (G>A) had an OR of 0.07 ($p < 0.001$). No significant association was found at 33,861 or 34,254. In the multivariable model, anti-PLA2R level had the largest reported OR (17.6; $p < 0.001$).

Conclusion: Serum anti-PLA2R levels showed strong discrimination between patients and healthy controls in this dataset. Associations with selected *PLA2R1* variants warrant further investigation before clinical or causal conclusions are drawn.

Key words: Glomerulonephritis, membranous; Receptors, phospholipase A2; Autoantibodies; Polymorphism, single nucleotide; Renal dialysis..

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Introduction

Membranous nephropathy (MN) is a common cause of nephrotic syndrome in adults. It is characterised histopathologically by diffuse thickening of the glomerular basement membrane due to immune complex accumulation.[1] A higher frequency of associated cancer has been described in older patients with MN and diabetes.[2] Autoantibodies are an important immunological feature of primary MN. Phospholipase A2 receptor (PLA2R) is a transmembrane glycoprotein expressed predominantly on glomerular podocytes and autoimmune targeting of PLA2R has been identified in approximately 70–80 % of patients with primary MN.[3, 4] Circulating anti-PLA2R antibodies are used as diagnostic and monitoring markers and their titres have been associated with treatment response and relapse.[5]

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Genetic predisposition has also been implicated in the pathogenesis of MN in addition to serological markers. Genome-wide association studies (GWAS) have identified certain single nucleotide polymorphisms (SNPs) in the *PLA2R1* gene that are strongly associated with development of primary MN.[6] Despite increasing global data, little is known about the prevalence of anti-PLA2R antibodies and the polymorphism of the *PLA2R1* gene in Middle Eastern populations, particularly those in Iraq. Exploration of potential geographic and ethnic variation of these markers in Iraqi patients may provide insights into regional expression and pathophysiology of the disease.[7]

This study compared serum PLA2R antibody levels and the frequency of *PLA2R1* variants in Iraqi patients with MN and apparently healthy controls. The findings may inform further investigation of these markers in this population.

Methods

Sample collection

Blood samples were collected from patients undergoing maintenance haemodialysis at Al-Kindy Teaching Hospital, Baghdad, Iraq. All patient samples were obtained prior to the dialysis session under aseptic conditions. Blood specimens were processed to obtain serum, which was analysed immediately or stored according to standard laboratory protocols. Control samples were collected from apparently healthy individuals using the same procedures.

Eligibility criteria

Patients aged ≥ 18 years with biopsy-confirmed MN who were receiving maintenance haemodialysis at Al-Kindy Teaching Hospital were eligible. Controls were apparently healthy individuals without a history of renal or autoimmune disease. Individuals with acute infection, malignancy, liver disease, other autoimmune disorders, recent blood transfusion or incomplete clinical or laboratory data were excluded.

Haematological testing

Complete blood count was performed for all samples using the Mindray BC-20 automated haematology analyser. White blood cell (WBC) counts and haemoglobin (Hb) levels were determined.

Biochemical testing

Albumin, creatinine, urea, alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), potassium, total protein, total serum bilirubin, iron and calcium were measured using the Cobas c311 analyser (*Roche*). Serum ferritin was measured using the Cobas e411 immunoassay analyser (*Roche*).

Serum PLA2R measurement

Serum anti-PLA2R levels were measured using a commercial sandwich enzyme-linked immunosorbent assay (ELISA) kit (*MyBioSource*, USA) according to the manufacturer's protocol. Microplates were pre-coated with anti-human PLA2R antibodies and a standard curve was prepared by serial dilution of the reference standard (80–1.25 ng/mL). Standards and samples were added in duplicate and incubated at 37 °C. Sequential incubations with a biotinylated detection antibody and a horseradish peroxidase conjugate were separated by washing steps. Substrate solution was added, followed by stop reagent. Optical density was measured at 450 nm and PLA2R concentrations were calculated from the standard curve.

DNA extraction

DNA amplification and sequencing

Forward and reverse primers were supplied in lyophilised form by Macrogen and are listed in Table 1. The primers were reconstituted in distilled water to prepare a stock solution of 100 pmol. A working solution of 10 pmol/µL was prepared by adding 10 µL of stock solution to 90 µL of deionised distilled water. Polymerase chain reaction (PCR) was performed in a total volume of 25 µL, using the components in Table 2 and the programme in Table 3. Amplification products were examined by electrophoresis on a 2 % agarose gel with a 1000 bp DNA ladder (Promega, USA), followed by DNA sequencing. Sequences were compared with a *PLA2R1* reference sequence to identify base substitutions.

Table 1: Primer sequences

Primer	Sequence (5'–3')	Tm
PLA2R-F	AGGAGAACAAGAAAGATGTGT	55.5
PLA2R-R	TCAACAACACCTAAGAACAAC	54.7

F: forward; R: reverse; Tm: melting temperature;

Table 2: Reaction mixture for polymerase chain reaction (PCR) amplification

Component	Volume (µL)	Final concentration
Master mix: Taq DNA polymerase, dNTPs, MgCl2 and reaction buffer	12.5	1x
Forward primer	1	0.2 p/mol
Reverse primer	1	0.2 p/mol
DNA template	3	< 250 ng/reaction
Distilled water	7.5	
Final volume	25	

dNTPs: deoxynucleoside triphosphates; PCR: polymerase chain reaction;

Table 3: Polymerase chain reaction (PCR) programme[†]

Step	Temperature (°C)	Duration	Cycles
Initial denaturation	94	5 min	1
Denaturation	94	30 s	30
Annealing	55	30 s	30
Extension	72	1 min	30
Final extension	72	10 min	

PCR: polymerase chain reaction.

Statistical analysis

Age was compared using the Mann–Whitney U test and sex distribution using Fisher's exact test. Allelic odds ratios (ORs) and 95 % confidence intervals (CIs) were calculated, with Fisher's exact test used to assess genetic associations and $p < 0.05$ considered significant. Spearman correlation, receiver operating characteristic (ROC) analysis and multivariable logistic regression were used for the reported serological and genetic analyses.

Results

Demographic characteristics

The demographic characteristics of the study groups are presented in Table 4. Median age was 48.00 years (interquartile range [IQR]: 42.25–56.00) in patients and 55.00 years (IQR: 45.00–60.75) in controls, with no significant difference between groups (Mann–Whitney U test, $p = 0.0827$).

Each group comprised 24 women (48.0 %) and 26 men (52.0 %). Sex distribution did not differ significantly between patients and controls (Fisher's exact test, $p = 1.0$).

Laboratory findings

The laboratory findings are summarised in Table 5. Patients had significantly lower serum albumin, calcium, iron and haemoglobin than controls (all $p < 0.05$). Creatinine, urea, ALP, phosphate, total serum bilirubin, ferritin and PLA2R antibody levels were significantly higher in patients (all $p < 0.001$). AST and ALT activities were also lower in patients, with significant differences for both parameters ($p < 0.01$). WBC counts were modestly reduced ($p = 0.0156$). Total protein did not differ significantly between groups ($p = 0.4924$).

Table 4: Demographic characteristics of patients and controls

Characteristic	Patients	Controls	p-value
Age (years)	48.00 (42.25–56.00)	55.00 (45.00–60.75)	0.083
Sex, n (%)	F: 24 (48.0 %), M: 26 (52.0 %)	F: 24 (48.0 %), M: 26 (52.0 %)	1.000

Age is shown as median (IQR). Sex is shown as n (%); F: female; M: male; IQR: interquartile range.

Table 5: Biochemical, haematological and serum PLA2R antibody measurements in patients and controls

Marker	n	Patients		n	Controls		p-value
		Median (IQR)	Mean ± SD		Median (IQR)	Mean ± SD	
ALB (g/dL)	50	3.70 (3.50–3.90)	3.64 ± 0.34	50	4.50 (4.20–4.90)	4.51 ± 0.49	< 0.001
CR (mg/dL)	50	11.60 (8.60–13.00)	11.07 ± 3.32	50	0.89 (0.78–1.08)	0.92 ± 0.19	< 0.001
AST (U/L)	50	15.00 (12.00–18.00)	15.79 ± 7.25	50	22.00 (19.00–29.00)	23.32 ± 6.94	< 0.001
ALT (U/L)	50	18.00 (11.50–29.75)	20.78 ± 11.29	50	26.50 (19.50–38.50)	27.54 ± 11.25	0.006

ALP (U/L)	50	311.50 (265.85–547.00)	402.57 ± 196.52	50	93.50 (65.50–129.00)	96.16 ± 33.05	< 0.001
Urea (mg/dL)	50	146.15 (135.00–180.00)	154.66 ± 33.00	50	30.50 (23.00–40.00)	30.96 ± 9.80	< 0.001
PO4 (mg/dL)	50	6.20 (5.10–6.70)	5.95 ± 1.14	50	3.55 (2.82–4.30)	3.55 ± 0.69	< 0.001
TP (g/dL)	50	5.50 (4.30–9.17)	6.71 ± 2.48	50	7.50 (6.60–7.90)	7.26 ± 0.71	0.492
TSB (mg/dL)	50	1.40 (0.83–1.70)	1.31 ± 0.63	50	0.72 (0.49–0.82)	0.69 ± 0.26	< 0.001
Iron (µg/dL)	50	41.70 (37.12–58.02)	49.02 ± 23.83	50	124.00 (91.00–161.00)	122.98 ± 36.84	< 0.001
Ca (mg/dL)	50	7.90 (7.08–8.50)	7.84 ± 1.05	50	9.40 (9.00–9.80)	9.40 ± 0.48	< 0.001
FERR (ng/mL)	50	316.50 (83.25–540.75)	347.21 ± 303.98	50	102.05 (58.00–142.57)	112.81 ± 75.86	< 0.001
WBC (/L)	50	6.30 (4.80–7.20)	6.14 ± 1.90	50	7.30 (5.70–8.00)	7.06 ± 1.81	0.015
Hb (g/dL)	50	8.90 (7.30–10.38)	9.04 ± 1.96	50	14.95 (13.90–16.03)	14.91 ± 1.37	< 0.001
PLA2R Ab (ng/mL)	50	1.53 (1.31–1.65)	1.48 ± 0.28	50	0.34 (0.24–0.45)	0.34 ± 0.13	< 0.001

IQR: interquartile range; SD: standard deviation; ALB: albumin; CR: creatinine; AST: aspartate aminotransferase; ALT: alanine aminotransferase; ALP: alkaline phosphatase; PO4: phosphate; TP: total protein; TSB: total serum bilirubin; Ca: calcium; FERR: ferritin; WBC: white blood cells; Hb: haemoglobin; PLA2R Ab: antibody to phospholipase A2 receptor.

Serum anti-PLA2R antibody levels were higher in patients than in controls, with separation between the groups in the individual-value plots (Figure 1). No significant correlation with serum creatinine was found within the patient group (Spearman $r = -0.12$, $p = 0.411$). Receiver operating characteristic (ROC) analysis yielded an area under the curve (AUC) of 1.000 in this dataset.

Genotype and allele distributions

Genotype distributions and allele frequencies at the reported *PLA2R1* positions were compared between patients and controls (Table 6). ORs and 95 % confidence intervals (CIs) were calculated using allelic models. Significance was assessed using Fisher's exact test, with $p < 0.05$ considered significant.

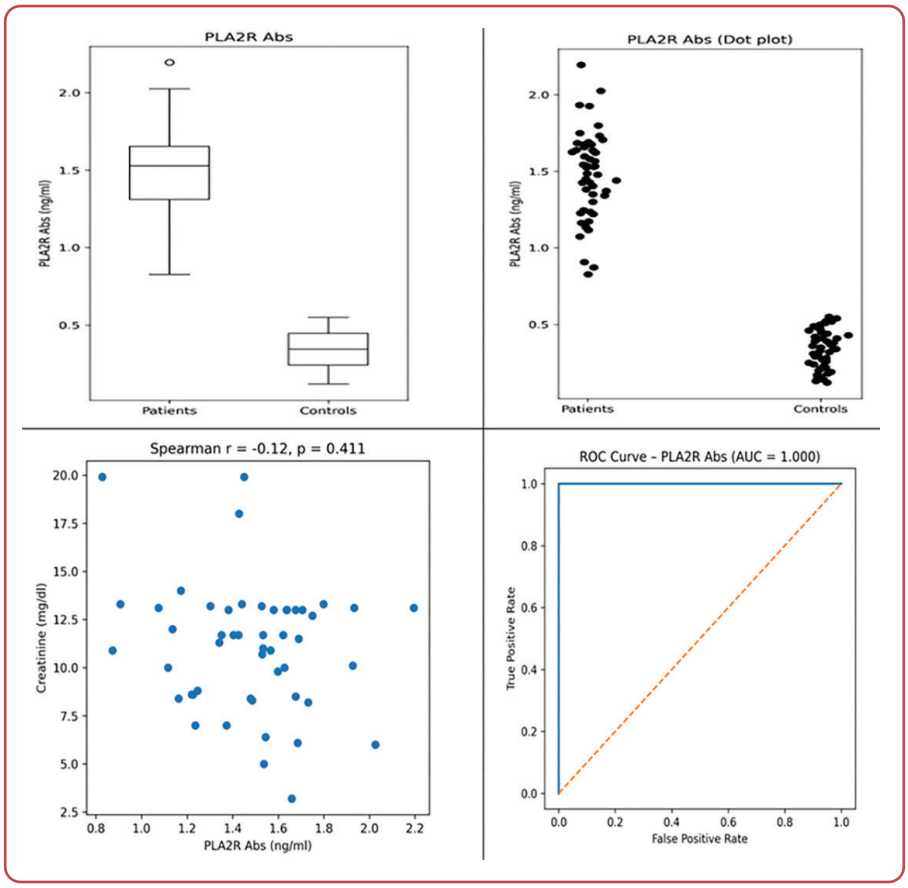


Figure 1: Distribution, correlation with renal function and discrimination of serum anti-PLA2R antibody levels

Upper left: box plot comparing patients and controls. Upper right: individual-value plot. Lower left: correlation with serum creatinine (Spearman $r = -0.12$, $p = 0.411$). Lower right: ROC curve (AUC = 1.000). PLA2R: phospholipase A2 receptor; ROC: receiver operating characteristic; AUC: area under the curve.

Table 6: Genotype and allele distributions at the reported PLA2R1 positions

PLA2R1 position	Genotype in patients, n (%)	Genotype in controls, n (%)	A-allele frequency: patients	A-allele frequency: controls	OR (95 % CI)	p-value	Interpretation
33,861 (G>A)	GG: 20 (80 %) GA:5 (20 %) AA: 0	GG: 20 (80 %) GA: 5 (20 %) AA: 0	0.10	0.10	1.00 (0.24–4.12)	1.000	No association
33,988 (G>A)	GG: 20 (80 %) GA: 5 (20 %) AA: 0	GG: 25 (100 %) GA: 0 AA: 0	0.10	0.00	6.78 (0.78–59.1)	0.041	Possible risk
34,046 (G>A)	GG: 25 (100 %) GA: 0 AA: 0	GG: 15 (60 %) GA: 10 (40 %) AA: 0	0.10	0.20	0.07 (0.01–0.42)	< 0.001	Protective polymorphism
34,254 (G>A)	GG: 20 (80 %) GA: 5 (20 %) AA: 0	GG: 20 (80 %) GA: 5 (20 %) AA: 0	0.10	0.10	1.00 (0.24–4.12)	1.000	No association

OR: odds ratio; CI: confidence interval. Genotyping was reported for 25 patients and 25 controls. Variants are identified using the nucleotide positions supplied in the manuscript.

Table 7: Reported multivariable logistic regression model

Variable	Comparison / Model	β coefficient	Odds ratio (OR)	95 % Confidence interval	p-value	Interpretation
PLA2R Ab (ng/mL)	Continuous	2.87	17.60	6.2 – 49.10	< 0.001	Strong independent predictor
Nucleotide position 33,988 (G>A)	GA vs GG	1.34	3.81	1.02 – 14.10	0.046	Possible risk factor
Nucleotide position 34,046 (G>A)	GA vs GG	-2.21	0.11	0.03 – 0.39	< 0.001	Protective polymorphism
Nucleotide position 33,861 (G>A)	GA vs GG	0.02	1.02	0.32 – 3.21	0.970	Not significant
Nucleotide position 34,254 (G>A)	GA vs GG	0.05	1.05	0.33 – 3.35	0.940	Not significant

OR: odds ratio; CI: confidence interval; GA vs GG: genotype comparison. The serum PLA2R variable is continuous, whereas the genetic variables are genotype contrasts.

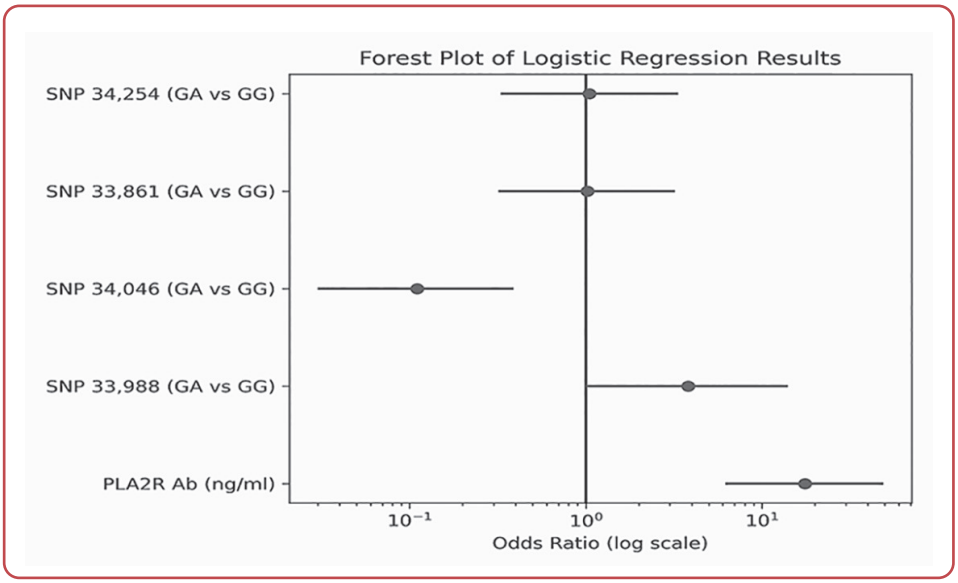


Figure 2: Forest plot of the reported multivariable logistic regression results

Point estimates and 95 % CIs are shown on a logarithmic OR scale. PLA2R Ab: antibody to phospholipase A2 receptor; CI: confidence interval; OR: odds ratio.

Multivariable analysis

The reported multivariable logistic regression model is shown in Table 7. Serum PLA2R antibody level had the largest reported OR (17.6; $p < 0.001$). The variant at 33,988 was associated with higher odds of disease (OR = 3.81, $p = 0.046$), while 34,046 was associated with lower odds (OR = 0.11, $p < 0.001$). No significant associations were found at 33,861 or 34,254.

The forest plot summarises the reported associations between serum PLA2R antibody levels, selected *PLA2R1* variants and disease status (Figure 2). The OR for serum antibody level was above one, with a CI that did not include one.

The reported OR for the 33,988 (G>A) variant was above one, suggesting an association with higher disease odds. This finding requires further investigation in larger cohorts.

The reported OR for 34,046 (G>A) was below one, with a CI entirely below one. This indicates an inverse association with disease status in the reported model, rather than demonstrating a causal protective effect.
The reported ORs for 33,861 and 34,254 were close to one, with CIs that included one. No significant associations were detected in this cohort; this does not establish biological neutrality.

In the reported model, serum PLA2R antibody level had a larger OR than the genetic variables. Genetic associations may contribute to understanding disease susceptibility, but effect estimates for differently scaled variables do not by themselves establish the relative importance of their pathogenic mechanisms.

Discussion

The biochemical and haematological differences observed between patients and controls were highly significant. These differences reflect adverse metabolic, renal, hepatic and immunological effects associated with chronic kidney disease under haemodialysis and with immune-mediated glomerular injury that are not specific to membranous nephropathy.[8] Serum albumin was significantly lower in patients than in controls ($p < 0.001$). Hypoalbuminaemia may reflect urinary protein loss, malnutrition, inflammation or altered hepatic synthesis in renal disease.[9–11]

Creatinine and blood urea were markedly higher in patients ($p < 0.001$). These findings are consistent with advanced renal impairment and reduced clearance of nitrogenous waste products in patients receiving haemodialysis.[12]

The AST and ALT levels in patients were significantly lower than those in controls but were within the lower end of the normal range, as revealed by the liver enzyme profiles. This pattern has often been observed in the dialysis population and may reflect haemodilution, pyridoxine (vitamin B6) deficiency and/or decreased hepatic enzyme synthesis rather than true recovery. By contrast, alkaline phosphatase was observed to increase markedly ($p < 0.001$), the likely cause being secondary hyperparathyroidism and increased bone turnover in chronic renal disease.[13]

Serum phosphate was higher in patients, while calcium was lower ($p < 0.001$).[14, 15] These findings are consistent with chronic kidney disease–mineral and bone disorder (CKD-MBD), which is associated with vascular calcification, fracture and cardiovascular risk.
Total protein did not differ significantly between groups ($p = 0.49$). Increased globulin concentrations due to chronic immune activation could potentially contribute to this finding despite hypoalbuminaemia, although globulin concentrations were not reported.

Total serum bilirubin was higher in patients ($p < 0.001$). Reduced hepatic clearance, dialysis-related haemolysis and subclinical cholestasis are possible explanations, but these mechanisms were not established by the reported measurements.[16]

Serum iron was lower and ferritin was higher in patients ($p < 0.001$). In chronic kidney disease, low iron with normal or elevated ferritin may reflect functional iron deficiency and inflammation, with ferritin acting as an acute-phase reactant and iron being sequestered in reticuloendothelial stores.[17]

Haemoglobin was significantly lower in patients ($p < 0.001$), consistent with anaemia in chronic kidney disease. Erythropoietin deficiency and altered iron handling may contribute, but the morphological classification of anaemia cannot be established from haemoglobin alone. WBC counts were also modestly lower ($p = 0.0156$), which may reflect several processes rather than demonstrating immune suppression.[12]

Serum PLA2R antibody levels were higher in patients than in controls ($p < 0.001$). This finding is consistent with the recognised involvement of anti-PLA2R antibodies in primary MN, but the present comparison does not establish the mechanism or extent of glomerular injury.

Within the patient group, serum PLA2R antibody levels were weakly negatively correlated with creatinine ($r = -0.12$), but the association was not significant ($p = 0.411$; Figure 1). Thus, higher antibody levels were not associated with greater creatinine concentrations in this cohort. Antibody levels may reflect immunological activity, whereas creatinine is influenced by renal function and dialysis-related factors.

ROC analysis yielded an AUC of 1.000 for distinguishing patients from healthy controls. The reported 100 % sensitivity and specificity describe performance in this dataset and do not establish performance in an independent clinical population. This finding is consistent with the significant between-group difference in antibody levels ($p < 0.001$), but its clinical applicability requires further evaluation.

Serum anti-PLA2R antibody level had the largest reported OR in the multivariable model. The established roles of anti-PLA2R antibodies in immune complex formation, complement activation and podocyte injury support their use in diagnosis and disease monitoring.[18, 19] The 2021 KDIGO guideline includes anti-PLA2R antibodies in diagnostic and risk-stratification assessment for MN.[18] Associations with the 33,988 (G>A) and 34,046 (G>A) variants were smaller in numerical OR magnitude, but comparison of a continuous antibody measure with genotype contrasts does not establish which mechanism is dominant. Published studies also support the non-invasive diagnostic assessment of primary MN using anti-PLA2R antibodies.[20]

Conclusion

Serum PLA2R antibody levels and selected *PLA2R1* variants differed between Iraqi patients with MN receiving haemodialysis and healthy controls. Antibody levels showed strong discrimination in the present dataset, while the reported genetic associations warrant further investigation. The biochemical and haematological findings were consistent with advanced renal impairment and dialysis-related disturbances. These findings should be interpreted within the selected dialysis cohort and do not establish general diagnostic performance or causal genetic effects.

Ethics

This study was approved by the Ethics Committee of the Higher Institute of Forensic Sciences, Al-Nahrain University (decision No: RFC-B-003, dated on 17 May 2026). The authors stated that informed consent and institutional permissions had been obtained where applicable.

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None.

Conflicts of interest

The authors declare that there is no conflict of interest.

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Data access

The data that support the findings of this study are available from the corresponding author upon reasonable individual request.

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