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Assessment of anti-severe acute respiratory syndrome coronavirus 2 immunoglobulin G antibody levels in coronavirus disease 2019 patients with varying stages of chronic kidney disease

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Abstract

Background: Immune impairment, capable of undermining humoral immunity to severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, occurs in chronic kidney disease (CKD). This study evaluated anti-SARS-CoV-2 immunoglobulin G (IgG) levels, their predictors, and temporal patterns in coronavirus disease 2019 (COVID-19) recovered patients with CKD stages 3 and 4.

Methods: This cross-sectional study was conducted at Dhaka Medical College from January to December 2021, including 80 COVID-19 – recovered patients with CKD stages 3 and 4. Serum anti-SARS-CoV-2 IgG antibodies were quantified by chemiluminescent microparticle immunoassay, and values ≥50 AU/mL were considered positive. Demographic, clinical, and biochemical data were recorded, and patients with dialysis, malignancy, or immunosuppressive therapy were excluded. Data were analyzed using the Statistical Package for the Social Sciences v26.

Results: Gross IgG positivity was 92.5%, Stage 3 had 95.0% positivity compared to 90.0% in Stage 4. Median IgG titers were significantly higher in Stage 3 (5120 AU/mL, interquartile range [IQR] 2730–8920) compared to Stage 4 (980 AU/mL, IQR = 520–2890; $P < 0.001$). Low IgG response was noted in 45.0% of patients with Stage 4 as compared to 20.0% in Stage 3 ($P = 0.019$). Multivariable logistic regression identified Stage 4 CKD (adjusted odds ratios [OR] = 3.06, 95% confidence interval [CI] = 1.18–7.92, $P = 0.021$) and shorter time since infection (OR = 0.83/week, 95% CI = 0.72–0.96, $P = 0.012$) as independent predictors of poor antibody response. IgG titers were highly correlated with both estimated glomerular filtration rate ($r = 0.68$) and weeks since infection ($r = 0.72$).

Conclusion: Advanced CKD significantly impairs humoral immune responses against SARS-CoV-2, with Stage 4 patients having much lower titers of antibodies and higher proportions of inappropriate serological response than Stage 3. These findings emphasize the need for more monitoring and personalized strategies to vaccination among this at-risk population.

Keywords: Chronic kidney disease, estimated glomerular filtration rate, humoral immunity, severe acute respiratory syndrome coronavirus 2

Introduction

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2)-induced coronavirus disease 2019 (COVID-19) pandemic posed unprecedented challenges to the healthcare system of the world, with the worst effects on patients with pre-existing chronic illnesses.[1] Chronic kidney disease (CKD) has emerged as a significant risk factor for unfavorable outcomes from COVID-19, including increased mortality, critical deterioration of the disease, and prolonged hospitalization.[2] The bidirectional interaction between CKD and COVID-19 is multifaceted, as SARS-CoV-2 infection exacerbates pre-existing renal dysfunction and immunomodulatory alterations in CKD compromise host antiviral defense.[3] CKD is an immune dysregulatory disease that exists as a paradoxical inflammatory state of chronicity with dysfunctional adaptive immunity.[4] This condition, also commonly known as "immune paralysis," is caused by the accumulation of uremic toxins, disordered cytokine profiles, and functional deficits of cellular and humoral immune compartments.[5] The reduced capacity of CKD patients to develop protective antibody responses following immunization has been widely documented in a number of pathogens, including hepatitis B virus and influenza, with antibody titers being inversely correlated with declining glomerular filtration rate.[6] These results raise serious concerns regarding the adequacy of humoral immunity following natural SARS-CoV-2 infection in subjects with advanced kidney disease. Humoral immunity, dominated by neutralizing antibodies that are secreted by B cells, is crucial for viral elimination and protection against reinfection.[7] Following SARS-CoV-2 infection, immunoglobulin G (IgG) antibodies against the viral spike protein are evident in 1–3 weeks and persist for many months, a serological marker of prior infection and potential immunity.[8] However, current evidence has shown that the extent and duration of antibody responses may be blunted in immunocompromised hosts, including CKD patients.[9] Epidemiological studies in humans have shown divergent findings for post-infection immunity in individuals with kidney disease, from maintained antibody responses in certain cases to spectacular defects, particularly with increasing severity of CKD.[10] The stage-specificity of the effect of CKD on SARS-CoV-2 antibody responses is largely unknown. Although severe renal impairment has been associated with suboptimal vaccine-induced immunity, the interaction of estimated glomerular filtration rate (eGFR) and naturally occurring antibody titers post-COVID-19 infection has not yet been fully investigated.[11] Understanding of the interaction will be essential to risk stratification, guiding vaccination policy, and predicting susceptibility to reinfection in this vulnerable population. This study aimed to assess and compare anti-SARS-CoV-2 IgG antibody titers in COVID-19-recovered patients with CKD stages 3 and 4, identify clinical predictors of unsatisfactory antibody responses, and analyze the temporal patterns of antibody generation in different levels of renal impairment.

Methods

This cross-sectional analytical study was conducted in the Department of Biochemistry, Dhaka Medical College, Dhaka, over 1 year from January to December 2021. A total of 80 COVID-19-recovered patients with pre-existing CKD were enrolled through purposive sampling after obtaining informed consent. The participants were stratified into two groups based on the Kidney Disease Improving Global Outcomes criteria:[11] Stage 3 CKD (eGFR 30–59 mL/min/1.73 m²) and Stage 4 CKD (eGFR 15–29 mL/min/1.73 m²), comprising 40 patients in each group. All patients had confirmed SARS-CoV-2 infection documented by reverse transcription–polymerase chain reaction and were evaluated 4–12 weeks following recovery to ensure adequate convalescence. Individuals with end-stage renal disease on dialysis, autoimmune disorders, active malignancy, or those receiving immunosuppressive or steroid therapy were excluded. Demographic information, clinical history, and comorbidities such as diabetes mellitus and hypertension were recorded using a structured data collection sheet. Blood samples were collected aseptically for biochemical assessment and anti-SARS-CoV-2 IgG antibody measurement. Serum IgG antibody levels against the SARS-CoV-2 spike protein were quantified using a chemiluminescent microparticle immunoassay method and expressed in arbitrary units per milliliter (AU/mL). A value ≥ 50 AU/mL was considered seropositive according to the manufacturer's cutoff guidelines.

All data were analyzed using the Statistical Package for the Social Sciences version 26.0. Continuous variables were expressed as mean ± standard deviation (SD) or median with interquartile range (IQR), depending on data distribution assessed by the Shapiro–Wilk test. Categorical variables were presented as frequency and percentage. Differences in proportions were evaluated by the Chi-square test, whereas continuous variables between the two CKD stages were compared using an independent sample t-test or Mann–Whitney U-test as appropriate. Correlation between antibody titers and clinical variables such as eGFR, age, body mass index (BMI), and weeks since infection was examined using Pearson's correlation coefficients. A multivariable logistic regression model was applied to identify independent predictors of low IgG response (<1000 AU/mL), and results were reported as adjusted odds ratios (OR) with 95% confidence interval (CI). $P < 0.05$ was considered statistically significant.

Results

Table 1 represents the baseline characteristics of the 80 participants by CKD stage. The population consisted of 40 patients in each Stage 3 (eGFR 30–59 mL/min/1.73 m²) and Stage 4 (eGFR = 15–29 mL/min/1.73 m²). The majority of participants (47.5%) were between 60 and 69 years, and there was no significant age difference among groups ($P = 0.402$). There were 62.5% males, with a similar gender distribution across stages ($P = 0.652$). Overweight (BMI = 25–29.9 kg/m²) was the most frequent category (47.5%), with similar BMI profiles between groups ($P = 0.869$). Prevalence of comorbid conditions was significant, with diabetes mellitus in 47.5% and hypertension in 66.3% among the study participants, with no statistically significant difference by CKD stages. The bulk of the study participants (56.2%) were assessed 8–12 weeks post-infection, and there were similar temporal distribution patterns between both CKD stages ($P = 0.821$), to enable comparability for serological comparison.

Table 2 unveils the primary outcome measures of seropositivity rates for IgG and antibody titers by CKD stage. Overall, seroprevalence was 92.5%, a little greater in Stage 3 (95.0%) than in Stage 4 (90.0%). The median IgG titer in Stage 3 patients was markedly elevated at 5120 AU/mL (IQR = 2730–8920), reflecting robust humoral responses. In contrast, patients in Stage 4 had significantly lower antibody titers with a median titer of 980 AU/mL (IQR = 520–2890), approximately an 80% reduction compared to Stage 3 ($P < 0.001$). This broad disparity indicates deteriorating humoral immunity with advancing renal failure. The stage-dependent disparity underlying this cohort median of 2520 AU/mL (IQR = 740–6420) is masked by this, highlighting the need for stage-stratified immunologic assessment in patients with CKD and COVID-19 recovery.

Table 3 reveals the prevalence of suboptimal antibody response (IgG <1000 AU/mL) by stage and comorbidity status of CKD. A far higher proportion of patients in Stage 4 had low IgG response (45.0%) than the patients in Stage 3 (20.0%), which gave a statistically significant finding (p=0.019). This gives strong evidence for stage-related immunocompromise in CKD. In diabetic mellitus patients, 37.5% had poor responses in comparison to 27.5% in the non-diabetic patients, although the difference was not statistically significant ($P = 0.309$). On this same vein, there was no impact of hypertension on decreased antibody production (35.4% in hypertensive compared to 28.1% in normotensive patients, $P = 0.498$).

Table 4 demonstrates the multivariable adjusted logistic model for predictors of low IgG after

Table 1: Baseline characteristics of the study population (*n*=80)

Variable	Category	Stage 3 (*n*=40), *n* (%)	Stage 4 (*n*=40), *n* (%)	*P*-value
Age (years)	40–49	6 (15.0)	3 (7.5)	
	50–59	10 (25.0)	7 (17.5)	
	60–69	18 (45.0)	20 (50.0)	
	≥70	6 (15.0)	10 (25.0)	0.402
Sex	Male	24 (60.0)	26 (65.0)	0.652
	Female	16 (40.0)	14 (35.0)	
BMI (kg/m²)	<25 (normal)	10 (25.0)	8 (20.0)	
	25–29.9 (overweight)	18 (45.0)	20 (50.0)	
	≥30 (obese)	12 (30.0)	12 (30.0)	0.869
Diabetes mellitus	Yes	16 (40.0)	22 (55.0)	0.176
	No	24 (60.0)	18 (45.0)	
Hypertension	Yes	24 (60.0)	29 (72.5)	0.243
	No	16 (40.0)	11 (27.5)	
eGFR (mL/min/1.73 m²)	30–44	25 (62.5)	0 (0.0)	
	45–59	15 (37.5)	0 (0.0)	
	15–29	0 (0.0)	40 (100.0)	<0.001
Weeks since infection	4–7	18 (45.0)	17 (42.5)	
	8–12	22 (55.0)	23 (57.5)	0.821

BMI: Body mass index, eGFR: estimated glomerular filtration rate

Table 2: Anti-SARS-CoV-2 IgG positivity and titers by CKD stage (*n*=80)

CKD stage	IgG positive (≥ 50 AU/mL), *n* (%)	IgG negative (< 50 AU/mL), *n* (%)	Median IgG (AU/mL, IQR)	*P*-value
Stage 3	38 (95.0)	2 (5.0)	5120 (2730–8920)	-
Stage 4	36 (90.0)	4 (10.0)	980 (520–2890)	<0.001
Overall	74 (92.5)	6 (7.5)	2520 (740–6420)	-

SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2, IQR: Interquartile range, CKD: Chronic kidney disease, IgG: Immunoglobulin G

Table 3: Distribution of low IgG response (<1000 AU/mL) by stage and comorbidities

Variable	Category	Low IgG, *n* (%)	Adequate IgG (≥1000 AU/mL), *n* (%)	*P*-value
CKD stage	Stage 3	8 (20.0)	32 (80.0)	
	Stage 4	18 (45.0)	22 (55.0)	0.019
Diabetes mellitus	Yes	15 (37.5)	25 (62.5)	
	No	11 (27.5)	29 (72.5)	0.309
Hypertension	Yes	17 (35.4)	31 (64.6)	
	No	9 (28.1)	23 (71.9)	0.498

CKD: Chronic kidney disease, IgG: Immunoglobulin G

adjustment for confounders. After adjustment for potential confounders, Stage 4 CKD was a predictor of low IgG with patients having 3 times greater odds (adjusted OR = 3.06, 95% CI = 1.18–7.92, P = 0.021) compared to Stage 3 patients. Time since infection had a protective effect, with more weeks reducing the odds of poor antibody response by 17% (OR = 0.83, 95% CI = 0.72–0.96, $P = 0.012$), showing maturation of the antibodies over time as the infection heals. Interestingly, age, BMI, diabetes mellitus, and hypertension did not independently predict antibody levels even after adjustment, suggesting that their effect may be confounded through or overruled by renal function status.

Figure 1 illustrates the pattern and magnitude of correlation between serum IgG antibody levels and key clinical parameters among COVID-19–recovered CKD patients. A strong positive association is observed between weeks since infection and IgG titers, indicating progressive antibody maturation over time. Similarly, eGFR shows a robust positive correlation, reflecting better humoral immune responses with preserved renal function. In contrast, age demonstrates a mild negative correlation, suggesting a tendency for reduced antibody generation in older individuals, while BMI displays a weak inverse trend without clinical significance.

Table 4: Multivariable logistic regression predicting low IgG (<1000 AU/mL)

Predictor	Adjusted OR (95% CI)	*P*-value
Stage 4 (vs. Stage 3)	3.06 (1.18–7.92)	0.021
Age (per year)	1.03 (0.98–1.08)	0.164
BMI (per kg/m²)	1.01 (0.89–1.16)	0.802
Diabetes (Yes vs. No)	1.45 (0.56–3.76)	0.447
Hypertension (Yes vs. No)	1.29 (0.48–3.49)	0.592
Weeks since infection (per week)	0.83 (0.72–0.96)	0.012

IgG: Immunoglobulin G, BMI: Body mass index, OR: Odds ratio, CI: Confidence interval

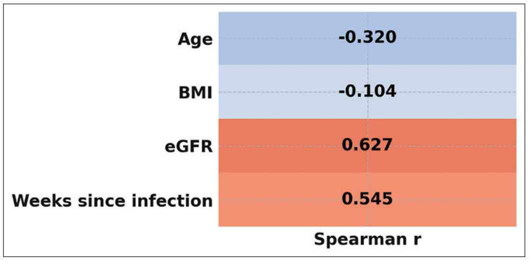

Figure 1: Correlation between anti-severe acute respiratory syndrome coronavirus 2 immunoglobulin G antibody titers and clinical variables in chronic kidney disease patients

Table 5 summarizes a comprehensive subgroup analysis of IgG titers by demographic and clinical strata. Stage-stratified comparison confirms the broad disparity between Stage 3 (median 5120 AU/mL) and Stage 4 (median 980 AU/mL) groups ($P < 0.001$). Age stratification revealed significantly higher titers in younger patients (<60 years: 4200 AU/mL) compared to older adults (≥60 years: 1820 AU/mL, $P = 0.014$), which suggests immune senescence due to age contributes to CKD-associated immunodeficiency. Diabetic patients presented with lower median titers (1450 AU/mL) compared to non-diabetic patients ($P = 0.028$), indicating diabetes as a modifying factor for immune responses. Blood pressure did not influence it significantly ($P = 0.112$). Temporal analysis proved continuous maturation of antibodies, where titers increased from 1260 AU/mL during weeks 4–7 to 4970 AU/mL during weeks 8–12 after infection ($P = 0.002$), reiterating the dynamic nature of immunity in convalescence and the importance of timing in serological assessment.

Figure 2a and b comprises two complementary panels demonstrating the serological pattern of anti-SARS-CoV-2 IgG antibodies among patients with CKD. Figure 2a shows a boxplot comparison revealing substantially lower IgG titers in Stage 4 compared with Stage 3 CKD, indicating impaired humoral response with worsening renal function. Figure 2b depicts a line graph of mean IgG levels over 4–12 weeks following infection, showing a progressive rise and stabilization of antibody titers over time, with consistently higher levels in Stage 3. Together, these panels emphasize that both disease severity and convalescent duration significantly influence antibody magnitude, illustrating the dual effect of renal function and time since infection on post-COVID-19 immune response.

Table 5: Anti-SARS-CoV-2 IgG response across CKD stages and clinical subgroups

Variable	Sub-category	*n* (%)	Median IgG (AU/mL, IQR)	*P*-value
CKD Stage	Stage 3 (eGFR 30–59)	40 (50.0)	5120 (2730–8920)	
	Stage 4 (SARS-CoV-2, IQR)	40 (50.0)	980 (520–2890)	<0.001
Age group (years)	<60	30 (37.5)	4200 (1880–7360)	
	≥60	50 (62.5)	1820 (720–4250)	0.014
Diabetes mellitus	Yes	38 (47.5)	1450 (650–4100)	0.028
Hypertension	Yes	53 (66.3)	1680 (740–4650)	0.112
Weeks since infection	4–7	35 (43.8)	1260 (580–2900)	
	8–12	45 (56.2)	4970 (2750–8730)	0.002

CKD: Chronic kidney disease, IgG: Immunoglobulin G, SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2, IQR: Interquartile range, eGFR: estimated glomerular filtration rate

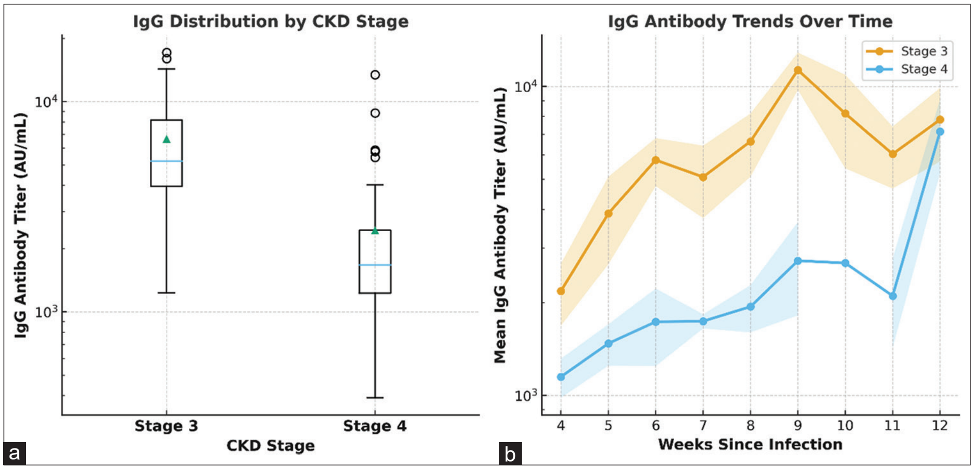

Figure 2: (a) Immunoglobulin G antibody distribution by chronic kidney disease stage and (b) temporal trends following severe acute respiratory syndrome coronavirus 2 infection

Discussion

This study provides robust evidence that CKD significantly compromises humoral immune response to natural SARS-CoV-2 infection, with Stage 4 CKD patients having very low anti-SARS-CoV-2 IgG antibody titers compared to their Stage 3 counterparts. The reduction in median antibody levels is fivefold between Stage 3 and Stage 4 patients and is clinically relevant and may have important implications for protection against reinfection as well as disease severity upon subsequent exposure. These findings are consistent with the general paradigm of immune dysregulation in CKD but provide additional data by quantifying stage-specific antibody response to natural infection, not vaccine. The pathophysiology of compromised antibody production in CKD is multifactorial and clearly defined by Vaziri *et al*.[5] Uremia-induced abnormalities of B-cell differentiation, impaired antigen presentation by non-viable dendritic cells, and lymphocyte exhaustion mediated by chronic inflammation are important processes that collectively contribute to compromised humoral immunity.[12] Clinical trials examining vaccine response in patients with CKD have all consistently demonstrated lower titers of antibody and accelerated waning of protective immunity, with more pronounced deficits at more advanced disease states.[13] Our findings validate these observations in the setting of natural infection and establish that immune compromise in CKD affects both vaccine-induced and infection-acquired immunity. The three-fold increased risk of compromised antibody response in Stage 4 patients, after adjustment for comorbidities and age, places emphasis on renal function as a major determinant of humoral competence. The patterns of antibody responses over time in our study are of particular interest. The progressive increase of IgG titers between 4–7 weeks and 8–12 weeks post-infection, with each of the extra weeks having 17% reduced odds of poor antibody response, shows that adequate time for the maturation of the antibodies is crucial for optimal humoral immunity.[14] This is consistent with widely documented kinetics of antibody class switch and affinity maturation following viral infections.[14] However, it is currently unclear if CKD patients ever achieve similar peak antibody levels to non-CKD patients on long follow-up or whether they simply plateau at lower peak levels. The significant positive correlation of IgG titers with eGFR ($r = 0.68$) described herein in our correlation analysis objectively defines a dose-response relation between antibody production and renal function. This correlation may be the cumulative burden of uremic toxins, proven directly to damage lymphocytes using model systems.[15] Protein-bound uremic toxins such as indoxyl sulfate and p-cresyl sulfate have specifically been implicated to inhibit B-cell growth and antibody production.[16] A threshold phenomenon observed between Stage 3 and Stage 4, where antibody function is also observed to decrease precipitously, hypothesizes that critical immune impairments could occur when eGFR dips below 30 mL/min/1.73m², a finding of immense clinical significance in the context of risk stratification. Interestingly, while diabetes mellitus had previously been associated with decreased titers of antibodies in univariable analysis, it did not appear as an independent predictor in the model that was adjusted. This result would suggest that the observed effect of diabetes could be mediated by quicker progression of CKD or shared pathophysiologic mechanisms and not as an independent immunologic defect.[17] Similarly, the inability to demonstrate independent association with hypertension indicates that risk factors for cardiovascular disease are weaker predictors of post-infection antibody responses than renal functional status. The poor negative correlation with age ($r = -0.35$), while not independently significant in multivariable analysis, likely represents evidence for established immunosenescence with advancing age,[18] which, in turn, can worsen CKD-induced immune impairment in older patients. The clinical implication of our findings is substantial. CKD patients, particularly Stage 4 disease, may also be at a higher risk of breakthrough infection or reinfection in the context of prior SARS-CoV-2 exposure due to lower antibody levels in them. Such vulnerability necessitates stricter surveillance for such patients, priority for vaccination and booster doses, constant emphasis on non-pharmacological precautionary measures, and prophylactic antibody therapy for high-risk conditions.[19] In addition, the utilization of antibody titers as surrogate markers for immunity in CKD patients should be used with caution because the boundary of protective immunity could be different from the general population.

Limitations of the study

A cross-sectional design prevents assessment of antibody persistence and temporal changes more than 12 weeks following infection. The study did not measure neutralizing antibody function or cellular immunity that would act as a compensatory protection in patients with impaired humoral responses.

Conclusion

This study demonstrates that CKD has a substantial negative impact on anti-SARS-CoV-2 IgG antibody titers following natural infection, with Stage 4 CKD individuals having significantly lower titers compared to Stage 3 patients. CKD stage and post-infection duration were independent predictors of antibody concentration, but traditional comorbidities did not have significant independent effects. These findings identify patients with severe renal impairment as being vulnerable to impaired humoral immunity and emphasize the necessity for individualized immunological surveillance, heightened vaccination, and potentially other protective interventions in this vulnerable group to ensure their maximum protection from COVID-19.

Future Recommendation

Future studies have to examine antibody stability and waning kinetics with long-term follow-up (6–12 months) to inform the optimal timing to give booster vaccines for CKD patients. Clinical outcome research defining the correlation among antibody titers and reinfection rates, and disease severity, is necessary to define stage-specific protective immunity thresholds for CKD patients.

References

1. Chen Y, Klein SL, Garibaldi BT, Li H, Wu C, Osevala NM, *et al*. Aging in COVID-19: Vulnerability, immunity and intervention. Ageing Res Rev 2021;65:101205.
2. Jdiaa SS, Mansour R, El Alayli A, Gautam A, Thomas P, Mustafa RA. COVID-19 and chronic kidney disease: An updated overview of reviews. J Nephrol 2022;35:69-85.
3. Hilbrands LB, Duivenvoorden R, Vart P, Franssen CF, Hemmelder MH, Jager KJ, *et al*. COVID-19-related mortality in kidney transplant and dialysis patients: Results of the ERACODA collaboration. Nephrol Dial Transplant 2020;35:1973-83.
4. Ferdous J, Rahman MM, Alam F. Level of knowledge about Covid 19 during pregnancy. SSB Glob J Med Sci 2021;2:5-10.
5. Vaziri ND, Pahl MV, Crum A, Norris K. Effect of uremia on structure and function of immune system. J Renal Nutr 2012;22:149-56.
6. Syed-Ahmed M, Narayanan M. Immune dysfunction and risk of infection in chronic kidney disease. Adv Chron Kidney Dis 2019;26:8-15.
7. Begum S. Baseline characteristics of Covid-19 infected spontaneous abortion cases. SSB Glob J Med Sci 2023;4:13-6.
8. Fox T, Geppert J, Dinnes J, Scandrett K, Bigio J, Sulis G, *et al*. Antibody tests for identification of current and past infection with SARS-CoV-2. Cochrane Database Syst Rev 2022;11:CD013652.
9. Carr EJ, Kronbichler A, Graham-Brown M, Abra G, Argyropoulos C, Harper L, *et al*. Review of early immune response to SARS-CoV-2 vaccination among patients with CKD. Kidney Int Rep 2021;6:2292-304.
10. Shovon MH, Imtiaz M, Biswas P, Tareq MM, Zilani MN, Hasan MN. A pan-genomic analysis based multi-epitope vaccine development by targeting Stenotrophomonas maltophilia using reverse vaccinology method: An *in-silico* approach. *In Silico* Pharmacol 2024;12:93.
11. Levin A, Stevens PE, Bilous RW, Coresh J, De Francisco AL, De Jong PE, *et al*. KDIGO 2012 clinical practice guideline for the evaluation and management of chronic kidney disease. Kidney Int Suppl 2013;3:1-50.
12. Betjes MG. Immune cell dysfunction and inflammation in end-stage renal disease. Nat Rev Nephrol 2013;9:255-65.
13. Krueger KM, Ison MG, Ghossein C. Practical guide to vaccination in all stages of CKD, including patients treated by dialysis or kidney transplantation. Am J Kidney Dis 2020;75:417-25.
14. Gaebler C, Wang Z, Lorenzi JC, Muecksch F, Finkin S, Tokuyama M, *et al*. Evolution of antibody immunity to SARS-CoV-2. Nature 2021;591:639-44.
15. Vanholder R, Schepers E, Pletinck A, Nagler EV, Glorieux G. The uremic toxicity of indoxyl sulfate and p-cresyl sulfate: A systematic review. J Am Soc Nephrol 2014;25:1897-907.
16. Lano G, Burtey S, Sallée M. Indoxyl sulfate, a uremic endotheliotoxin. Toxins (Basel) 2020;12:229.
17. Critchley JA, Carey IM, Harris T, DeWilde S, Hosking FJ, Cook DG. Glycemic control and risk of infections among people with type 1 or type 2 diabetes in a large primary care cohort study. Diabetes Care 2018;41:2127-35.
18. Fulop T, Larbi A, Pawelec G, Khalil A, Cohen AA, Hirokawa K, *et al*. Immunology of aging: the birth of inflammaging. Clin Rev Allergy Immunol 2023;64:109-22.
19. Stevens KI, Frangou E, Shin JI, Anders HJ, Bruchfeld A, Schönermarck U, *et al*. Perspective on COVID-19 vaccination in patients with immune-mediated kidney diseases: Consensus statements from the ERA-IWG and EUVAS. Nephrol Dial Transplant 2022;37:1400-10.