Clinical and Experimental Nephrology (2023) 27:236–242
https://doi.org/10.1007/s10157-022-02299-6

ORIGINAL ARTICLE

Association between COVID-19 vaccination and relapse of glomerulonephritis

Yuki Ota[1] · Ryoma Kuroki[1] · Mayu Iwata[1] · Hiroshi Taira[2] · Sayumi Matsuo[3] · Masafumi Kamijo[1] · Kumiko Muta[3] · Tomoya Nishino[3]

Received: 12 August 2022 / Accepted: 11 November 2022 / Published online: 23 November 2022

Abstract

Background Vaccines for coronavirus disease 2019 (COVID-19) have been developed and are recommended for patients with chronic kidney disease; however, it has been reported that glomerulonephritis worsens after vaccination. We aimed to elucidate the incidence and association between COVID-19 vaccination and glomerulonephritis relapse.

Methods We investigated the onset of renal events and adverse reactions after COVID-19 vaccination in 111 patients diagnosed with glomerulonephritis. Renal events were defined as worsening hematuria, increased proteinuria, and an increased creatine level over 1.5-fold from baseline.

Results Patients were 57 ± 18 years old (55.9% female) and had an estimated glomerular filtration rate of 57.0 ± 25.0 ml/min/1.73 m^2. A pathological diagnosis of IgA nephropathy was confirmed in 55.0%, minimal change disease in 22.5%, and membranous nephropathy in 10.8% of the patients. The BNT162b2 (Pfizer) and mRNA-1273 (Moderna) vaccines were administered in 88.2% and 11.7% of the cases, respectively. Renal events were observed in 22.5% of patients, 10.8% had increased proteinuria, 12.6% had worsening hematuria, and 1.8% received additional immunosuppressive treatment. Only 0.9% required temporary hemodialysis from exacerbation of renal dysfunction. Renal events were higher in younger patients ($P = 0.02$), being highest in those with IgA nephropathy, but there was no difference in the incidence between pathological diagnoses. There was a significantly higher incidence of renal events in patients with fever ($P = 0.02$).

Conclusions COVID-19 vaccination and glomerulonephritis relapse may be related, but further research is needed.

Keywords COVID-19 vaccination · Glomerulonephritis · Relapse

Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection has had a major impact on the world, causing numerous infections and deaths. Vaccination is underway to prevent the spread of the infection, and it has been reported that coronavirus disease 2019 (COVID-19) vaccines may prevent the onset and aggravation of SARS-CoV-2 infection [1, 2]. Patients with chronic kidney disease can become more severely ill, and vaccination has been suggested [1]. However, there have been many reports of new onset or relapse of glomerulonephritis and nephrotic syndrome after COVID-19 vaccination [2], although its incidence and relevance remain unclear. This study aimed to verify the incidence of nephritis relapse by following the clinical course of patients with glomerulonephritis after COVID-19 vaccination.

Materials and methods

Patients who visited the Sasebo City General Hospital from April 1, 2021, to December 31, 2021, and who were previously diagnosed with glomerulonephritis by renal biopsy and received COVID-19 vaccines twice were included in this study. Patients under the age of 18 years, who had been

✉ Yuki Ota
yukiota@hospital.sasebo.nagasaki.jp

[1] Department of Nephrology, Sasebo City General Hospital, 9-3 Hirase-cho, Sasebo, Nagasaki 857-8511, Japan

[2] Department of Nephrology, Saiseikai Nagasaki Hospital, 2-5-1 Katafuchi, Nagasaki, Nagasaki 850-0003, Japan

[3] Department of Nephrology, Nagasaki University Hospital, 1-7-1 Sakamoto, Nagasaki, Nagasaki 852-8501, Japan

vaccinated only once, or whose pathological diagnosis was unknown were excluded.

We collected clinical data, such as age, sex, height, weight, pathological diagnosis on renal biopsy, comorbidities, blood pressure, oral medication, blood test results, and urinalysis results, from the medical records. Data were obtained before and within 3 months of COVID-19 vaccination, and compared for renal events. Renal events were defined as a combination of worsening hematuria, increased proteinuria, and a 1.5-fold increase in creatine (Cr) levels. Increased proteinuria was defined as the extent of proteinuria before vaccination using the urinary protein-to-creatinine ratio (UPCR). Based on Japan Nephrology Society's guidelines [3], the following UPCR categories were used: A1, UPCR < 0.15 g/gCr; A2, 0.15 ≤ UPCR < 0.50 g/gCr; and A3, UPCR ≥ 0.50 g/gCr. Using the UPCR, previous values were interpreted as follows: < 0.3 g/gCr, progression to overt proteinuria (≥ 0.3 g/gCr); < 0.3 g-1.0 g/gCr, progression to ≥ 1 g/gCr; 1.0–3.5 g/gCr, difference increased by ≥ 2 g/gCr; and ≥ 3.5 g/gCr, increase of two-fold or higher. To investigate the relationship between pathological damage on previous renal biopsy and relapse after COVID-19 vaccination, we analyzed the pathological findings and incidence of renal events in patients with IgA nephropathy using the Japanese guidelines for IgA nephropathy [4] and the Oxford Classification [5]. In addition, adverse events were confirmed by face-to-face interviews with the patients.

Categorical variables are expressed as number (%) and continuous variables as mean ± standard deviation. Non-normally distributed data were presented as median values with interquartile ranges. JMP 15 software (SAS Institute Inc., Cary, NC, USA) was used to perform statistical analysis. Categorical variables were assessed using the chi-square test, and continuous variables were compared using the Mann–Whitney *U*-test. A subgroup analysis was performed to determine the incidence of renal events. Age, estimated glomerular filtration rate (eGFR), body mass index, and UPCR were distinguished by the median. We also selected clinically important factors and performed a multivariate logistic regression analysis. Statistical significance was set at $P < 0.05$. To calculate the eGFR from serum creatinine, age, and sex, the following estimation equations for Japanese patients with chronic kidney disease were used: for men, eGFR (mL/min/1.73 m^2) = 194 × age-0.287 × serum creatinine-1.094; for women, eGFR (mL/min/1.73 m^2) = 194 × age − 0.287 × serum creatinine-1.094 × 0.739 [6].

This study was approved by the ethics committee of Sasebo City General Hospital (Nagasaki, Japan) (2021-A022) and performed in accordance with the 1964 Declaration of Helsinki and its later amendments. Written informed consent was obtained from all patients.

Results

Of the 116 patients, a pathological diagnosis on renal biopsy could not be confirmed in three patients, and one did not provide consent before the study. The study was initiated in 112 patients, but one withdrew consent; hence, 111 patients were included in the final analysis (Fig. 1). The average age was 57 ± 18 years, 55.9% were female, and the average eGFR was 57.0 ± 25.0 ml/min/1.73 m^2. The UPCR classification was as follows: 35.1%, A1; 32.4%, A2; and 32.4%, A3. Based on renal biopsy pathological diagnosis, 55.0% of patients had IgA nephropathy; 22.5%, minimal change disease; and 10.8%, membranous nephropathy. Less than 10% had antineutrophil cytoplasmic autoantibody (ANCA)-associated nephritis, non-IgA-type mesangial proliferative

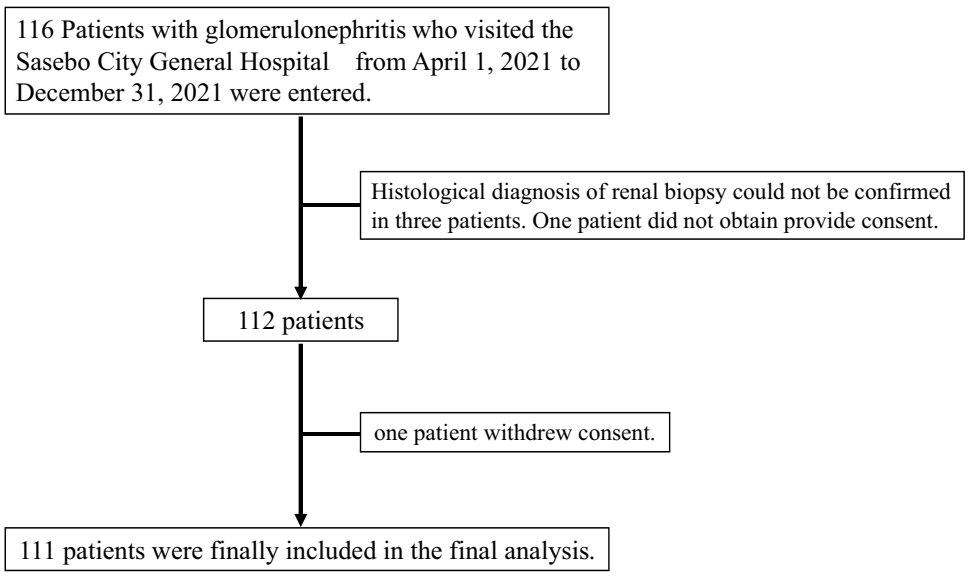

Fig. 1 Flow chart of patient selection

glomerulonephritis, membranous proliferative glomerulonephritis, lupus nephritis, and focal segmental glomerulosclerosis. Comorbidities were diabetes, hypertension, and heart disease in 20.7%, 77.4%, and 8.1% of patients, respectively. Oral medication prescription rates were as follows: 52.2%, steroids; 20.7%, immunosuppressive drugs; and 68.4%, renin-angiotensin-aldosterone system inhibitors. The COVID-19 vaccines BNT162b2 (Pfizer) and mRNA-1273 (Moderna) were administered in 88.2% and 11.7% of cases, respectively (Table 1). Renal events were observed in 22.5% of patients, 10.8% had increased proteinuria, 12.6% had worsening hematuria, and 1.8% received additional immunosuppressive treatment. Only 0.9% of the patients required temporary hemodialysis due to exacerbation of renal dysfunction (Fig. 2a). A renal biopsy was performed in one patient after a renal event of increased proteinuria and a 1.5-fold increase in Cr levels. Pathological findings did not reveal any association with COVID-19 vaccination. There were no cases of cardiovascular events or death. The incidence of renal events tended to be higher in younger patients ($P = 0.02$). There was no difference in the incidence of renal events with or without the use of oral steroids or immunosuppressive drugs. Pathological diagnosis-based renal event incidence was highest with IgA nephropathy; however, no difference in incidence rates between pathological diagnoses was noted (Fig. 2b, Table 2). Worsening hematuria was more common in IgA nephropathy, and increased proteinuria, in MCD and MN (Table 2). Gross hematuria after COVID-19 vaccination was observed in three of 61 patients with IgA nephropathy (Table 2).

In patients with IgA nephropathy, acute lesions (classified according to the clinical guidelines for IgA nephropathy in

Table 1 Baseline characteristics

Characteristic	Mean ± SD, median (IQR), or *n* (%)
Age, years	57.0 ± 17.9
Sex, *n* (%)	Male: 49 (44.1); Female: 62 (55.9)
BMI, kg/m^2	24.1 ± 4.6
Renal pathological diagnosis, *n* (%)	IgA nephropathy: 61 (55.0)
	Minimal change disease: 25 (22.5)
	Membranous nephropathy: 12 (10.8)
	ANCA-associated nephritis: 7 (6.3)
	Non-IgA-type mesangial proliferative glomerulonephritis: 2 (1.8)
	Membranous proliferative glomerulonephritis: 2 (1.8)
	Lupus nephritis: 1 (0.9)
	Focal segmental glomerulosclerosis: 1 (0.9)
DM, *n* (%)	23 (20.7)
Hypertension, *n* (%)	86 (77.4)
Heart disease, *n* (%)	9 (8.1)
BUN (mg/dL)	19.6 ± 10.0
Cr (mg/dL)	1.2 ± 0.9
eGFR (ml/min/1.73 m^2)	57.0 ± 25.0
UPCR (g/gCr)	0.3 (0.1–0.7)
UPCR ˃ 0.3 g/gCr, *n* (%)	57 (51.4)
Proteinuria categories, *n* (%)	A1: 39 (35.1), A2: 36 (32.4), A3: 36 (32.4)
Hematuria (˃ 5/HPF), *n* (%)	17 (15.3)
Steroids, *n* (%)	58 (52.2)
Immunosuppressive drugs, *n* (%)	23 (20.7)
RAAS inhibitor, *n* (%)	76 (68.4)
Anti-platelet drugs, *n* (%)	11 (9.9)
Diabetes drugs, *n* (%)	21 (18.9)
Vaccine type, *n* (%)	BNT162b2, 98 (88.2), mRNA-1273, 13 (11.7)
Systolic / Diastolic blood pressure (mmHg)	134 ± 17
Diastolic blood pressure (mmHg)	80 ± 11

BMI, body mass index; *DM*, diabetes mellitus; *BUN*, blood urea nitrogen; *Cr*, creatine; *eGFR*, estimated glomerular filtration rate; *UPCR*, urinary protein-to-creatinine ratio; *RAAS* inhibitor, renin-angiotensin-aldosterone system inhibitor; *ANCA*, antineutrophil cytoplasmic autoantibody

Proteinuria categories A1: UPCR < 0.15 g/gCr, A2: 0.15 ≤ UPCR < 0.50 g/gCr, A3: UPCR ≥ 0.50

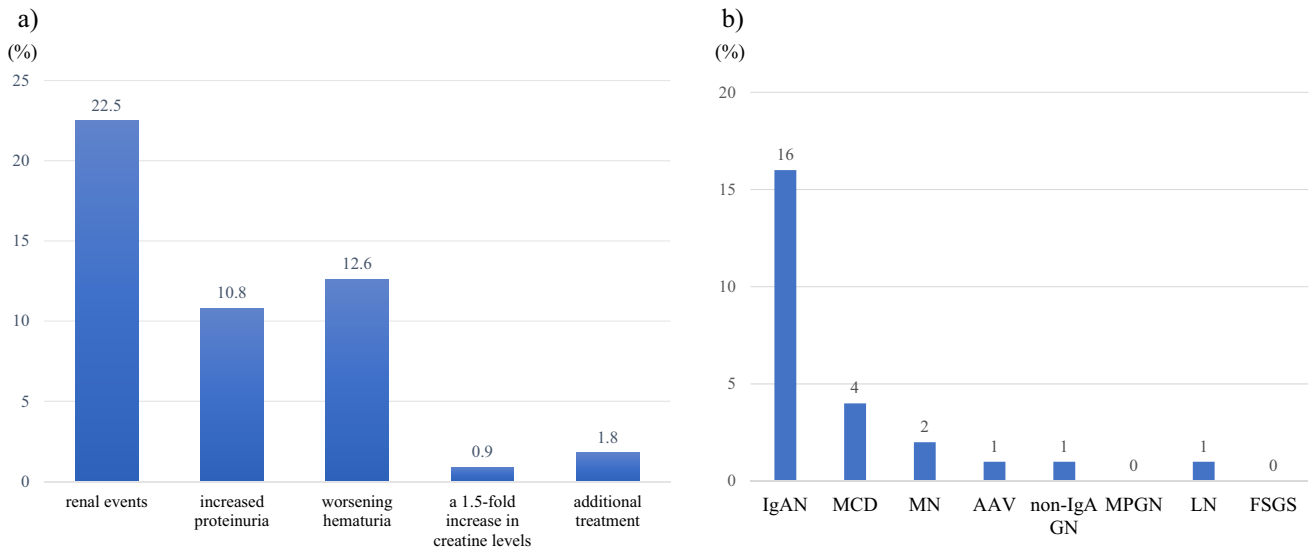

Fig. 2 Renal events. **a** Incidence of events after vaccination. **b** Number of renal events by pathological diagnosis. *IgAN*, IgA nephropathy; *MCD*, minimal change disease; *MN*, membranous nephropathy; *AAV*, ANCA-associated vasculitis; *non-IgAGN*, non-IgA-type mesangial proliferative glomerulonephritis; *MPGN*, membranous proliferative glomerulonephritis; *LN*, lupus nephritis; *FSGS*, focal segmental glomerulosclerosis

Table 2 Renal events by pathological diagnosis

	IgAN	MCD	MN	AAV	Non-IgAN	MPGN	LN	FSGS	*P* value
Renal event, *n* (%)	16 (26.2)	4 (16.0)	2 (16.7)	1 (14.3)	1 (50.0)	0 (0)	1 (100)	0 (0)	0.45
Increased proteinuria, *n* (%)	5 (8.2)	3 (12.0)	2 (16.7)	0 (0)	1 (50.0)	0 (0)	1 (100)	0 (0)	0.06
Worsening hematuria, *n* (%)	11 (18.0)	1 (4.0)	1 (8.3)	1 (14.3)	0 (0)	0 (0)	0 (0)	0 (0)	0.73
Gross hematuria, *n* (%)	3 (4.9)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0 (0)	0.93

IgAN, IgA nephropathy; *MCD*, minimal change disease; *MN*, membranous nephropathy; *AAV*, ANCA-associated vasculitis; *non-IgAGN*, non-IgA-type mesangial proliferative glomerulonephritis; *MPGN*, membranous proliferative glomerulonephritis; *LN*, lupus nephritis; *FSGS*, focal segmental glomerulosclerosis

Japan [4]) were associated with renal events, and E lesions (according to the Oxford Classification [5]) were associated with worsening hematuria (Supplementary Table S1). Significant gender-based differences in renal events by renal pathological diagnosis were not observed (Supplementary Table S2).

In the vaccine group, there was no difference in the onset of renal events between BNT162b2 and mRNA-1273 vaccines. Subgroup analysis showed that renal event incidence was significantly lower in patients aged ≥ 60 years and with an eGFR of < 55 ml/min/1.73 m^2, and that it was significantly higher in patients with fever ($P = 0.02$) (Tables 3, 4). Post-vaccination fever was observed in 42.3% of the patients (Supplementary Table S3). Younger patients had a higher incidence of fever than older patients ($P < 0.001$), and patients who took steroids had a significantly lower rate of fever than those who did not (available oral steroids: 32.2%, no oral steroids: 54.7%, $P = 0.02$). The rate of fever in patients vaccinated with mRNA-1273 was higher than that in those vaccinated with BNT162b2 (BNT162b2: 36.4%, mRNA-1273: 92.1%, $P < 0.001$).

Discussion

Several cases of new onset or relapse of glomerulonephritis after vaccination have been reported; however, its incidence has not been evaluated. This is the first report to investigate the clinical course of patients with nephritis in a single center after COVID-19 vaccination and to evaluate the incidence of COVID-19 vaccination and renal events. It is unclear whether renal events have a direct causal relationship with the vaccines, but we consider this study to be clinically useful because assessing the effects of vaccination in patients with nephritis may contribute to the safe vaccination of patients with glomerulonephritis.

Table 3 Subgroup analysis

	HR	*P* value
Age ≥ 60 years	0.34 (0.13–0.89)	0.04
Male	0.82 (0.33–2.00)	0.82
eGFR < 55 ml/min/1.73 m^2	0.25 (0.09–0.69)	0.006
BMI ˃ 22 kg/m^2	0.65 (0.26–1.60)	0.35
UPCR ˃ 0.3 g/gCr	1.03 (0.42–2.52)	1.00
Hematuria	2.96 (0.99–8.82)	0.06
IgA nephropathy	1.62 (0.65–4.06)	0.36
Diabetes mellitus	0.67 (0.21–2.20)	0.59
Hypertension	0.90 (0.31–2.57)	0.79
Heart disease	0.99 (0.19–5.11)	1.00
Steroids	0.97 (0.40–2.35)	1.00
Immunosuppressive drugs	1.30 (0.45–3.75)	0.59
RAAS inhibitor	0.96 (0.37–2.49)	1.00
BNT162b2 (Pfizer/BioNTech)	0.96 (0.24–3.81)	1.00
Fever	3.97 (1.53–10.3)	0.005

eGFR, estimated glomerular filtration rate; *BMI*, body mass index; *UPCR*, urinary protein-to-creatinine ratio; *RAAS inhibitor*, renin-angiotensin-aldosterone system inhibitor

Renal events were observed in 22.5% of patients, 10.8% had increased proteinuria, 12.6% had worsening hematuria, and 0.9% had Cr values greater than 1.5-fold. Of the patients, 1.8% received additional immunosuppressive treatment and 0.9% required temporary hemodialysis due to exacerbation of renal dysfunction. However, no one died, developed cardiovascular events, or required maintenance dialysis. In addition, none of the patients were infected with COVID-19 during the observation period.

In one review, among newly developed glomerulonephritis cases, minimal change disease was the most common outcome after COVID-19 vaccination, followed by IgA nephropathy and vasculitis [7]. However, data on which pathological diagnosis most often relapses in glomerulonephritis that also relapses after COVID-19 vaccination are lacking. Previous reports have shown that patients who relapsed after receiving a COVID-19 vaccine were at a higher risk of IgA nephropathy and typical nephrotic syndrome, but not of anti-glomerular basement membrane (GBM) disease or ANCA-related vasculitis [7]. IgA nephropathy with gross hematuria after vaccination has improved with almost no treatment [2]. In this study, renal events were more common with IgA nephropathy by pathological diagnosis; however, IgA nephropathy treatment was not required in any of the cases, while immunosuppressive treatment was required in two membranous nephropathy cases.

A study has explored the association between non-COVID-19 vaccines and nephritis. In this study, minimal change disease, membranous nephropathy, and vasculitis have been shown to develop after influenza vaccination [8]. Additionally, the onset of the nephrotic syndrome has also been reported after administering inactivated vaccines (e.g., hepatitis B, pneumococcal, and pertussis), live vaccines for measles, and Bacille Calmette-Guérin [8]. However, it is difficult to prove the causal relationship between these vaccines and glomerulonephritis, and it is not clear whether there are specific findings in renal histology.

The vaccines used in our study were all mRNA vaccines. COVID-19 mRNA vaccines are highly immunogenic and cross-reactive, which may exacerbate the autoimmune process and cause the onset or recurrence of glomerulonephritis

Table 4 Incidence of renal events

	Model 1			Model 2			Model 3		
	HR	95% CI	*P* value	HR	95% CI	*P* value	HR	95% CI	*P* value
Age	0.97	0.95–0.99	0.03	0.98	0.95–1.01	0.16	0.99	0.96–1.04	0.90
Sex (female)	0.83	0.33–2.06	0.68	0.85	0.34–2.13	0.73	0.65	0.24–1.78	0.46
BMI				0.99	0.90–1.10	0.95	0.99	0.89–1.10	0.82
eGFR				1.01	0.99–1.03	0.51	1.01	0.99–1.03	0.45
Diabetes mellitus							1.07	0.28–4.05	0.92
Steroids or immunosuppressive drugs							1.31	0.41–4.22	0.65
IgA nephropathy							1.68	0.50–5.64	0.39
Fever							3.76	1.23–11.5	0.02

Model 1: age, sex

Model 2: age, sex, BMI, eGFR

Model 3: age, sex, BMI, eGFR, diabetes mellitus, steroids or immunosuppressive drugs, IgA nephropathy, fever

In each model, multivariate analysis was performed with clinically important factors

BMI, body mass index; *eGFR*, estimated glomerular filtration rate

[2]. The mechanisms through which nephritis relapses may differ for each pathological diagnosis. IgA nephropathy has a relatively early onset after vaccination and may be associated with rapid immune mechanisms, such as memory recall response and recruitment of cells secreting galactose-deficient IgA1 antibodies. In contrast, the progression of minimal change disease takes a certain amount of time, suggesting the role of cell-mediated immunity [9]. The mechanism underlying podocyte damage after COVID-19 vaccination is hypothesized to involve the expression of permeability factors, such as cytokines and autoantibodies, by stimulating antigen-presenting cells, B cells, and activating T cells, which leads to loss of foot processes and disruption of the glomerular permeation barrier [10]. In addition, several COVID-19 infection-related nephritis cases have been reported, and COVID-19 infection is believed to directly cause podocyte damage [11]. COVID-19 vaccine-induced glomerulonephritis and COVID-19 infection may have similar mechanisms [2]. In this study, renal biopsy of a relapsed case after vaccination showed broad podocyte effacement, which is usually seen in nephrotic syndrome; however, no other characteristic findings were observed.

Our results showed that patients with fever and young people had more renal events, and multivariate analysis showed that fever was associated with the onset of renal events. The occurrence of a fever forms the basis for a high immune response. Renal events may occur in patients with a strong immune response due to immunological dysregulation in patients with glomerulonephritis and nephrotic syndrome. The two cases requiring additional treatment in this study were both long-term treatment-dependent membranous nephropathy cases and were treated with low-dose steroids. Oral steroids or immunosuppressive drugs were administered to 55.4% of the patients, and cyclosporine and mizoribine were used as immunosuppressive drugs. Although it has been reported that the COVID-19 vaccine may be less effective in patients receiving rituximab [12], no patient was treated with rituximab in this study. It is unclear how effective vaccination was in the target patients; however, no patients were infected with COVID-19 during the study period and no deaths were associated with vaccination. COVID-19 infection in patients with glomerulonephritis has been reported to result in higher mortality and an increased risk of acute kidney injury compared to controls [13]. It has been reported that many cases of new onset or relapse of glomerulonephritis caused by COVID-19 vaccines were in spontaneous remission or had a good therapeutic response [8]. Considering the aggravation of COVID-19 infection and the exacerbation of glomerulonephritis due to the infection itself, it is suggested that the benefits of COVID-19 vaccination outweigh the risks of developing nephritis or relapsing [8]. However, since it is unclear which set of patient characteristics has the highest risk of vaccine-related disadvantages, COVID-19 vaccination should be carried out with careful consideration of the patient background.

This study has several limitations. First, the number of cases was small, making it difficult to generalize results. Second, renal events may not have been collected or may have been underestimated in spontaneously relieved cases. Conversely, the exacerbation of nephritis itself, which is not related to the vaccine, may have been overestimated. Third, data could not be collected after the first vaccination, and the data were only compared after the second vaccination; therefore, the effect after the first vaccination could not be evaluated.

Conclusion

There were cases in which proteinuria, hematuria, and serum creatinine levels increased after COVID-19 vaccination, and the incidence was significantly higher in patients with fever and young people. Further research on the association between COVID-19 vaccination and glomerulonephritis is needed for the safe vaccination of patients with glomerulonephritis.

Supplementary Information The online version contains supplementary material available at https://doi.org/10.1007/s10157-022-02299-6.

Acknowledgements We are grateful to Mineaki Kitamura for his guidance on this research.

Author contributions All authors contributed to the study conception and design. Material preparation, data collection, and analysis were performed by YO, RK, MI, HT, SM, and MK. The first draft of the manuscript was written by YO, and all authors commented on previous versions of the manuscript. All authors read and approved the final manuscript.

Funding This research received no external funding.

Data availability The datasets generated during and/or analysed during the current study are available from the corresponding author on reasonable request.

Declarations

Conflict of interest The authors have declared that no conflict of interest exists.

Research involving human participants This study was approved by the ethics committee of the Sasebo City General Hospital (Nagasaki, Japan) (2021-A022) and performed in accordance with the 1964 Declaration of Helsinki and its later amendments.

Informed consent Informed consent was obtained from all individual participants included in the study.

References

1. Hou YC, Lu KC, Kuo KL. The efficacy of COVID-19 vaccines in chronic kidney disease and kidney transplantation patients: a narrative review. Vaccin (Basel). 2021;9:885.
2. Klomjit N, Alexander MP, Fervenza FC, Zoghby Z, Garg A, Hogan MC, et al. COVID-19 vaccination and glomerulonephritis. Kidney Int Rep. 2021;6:2969–78.
3. Japan Nephrology Society. Special issue: clinical practice guidebook for diagnosis and treatment of chronic kidney disease 2012. Nihon Jinzo Gakkai Shi. 2012;54:1034–191.
4. Matsuo S, Kawamura T, Joh K, Utsunomiya Y, Okonogi H, Miyazaki Y, Koike K, Yokoo T, Matsushima M, Yoshimura M, Ogura M. Clinical guides for immunoglobulin A (IgA) nephropathy in Japan, third version. Nihon Jinzo Gakkai Shi. 2011;53:123–35.
5. Trimarchi H, Barratt J, Cattran DC, Cook HT, Coppo R, Haas M, et al. Oxford classification of IgA nephropathy 2016: an update from the IgA Nephropathy Classification Working Group. Kidney Int. 2017;91:1014–21.
6. Matsuo S, Imai E, Horio M, Yasuda Y, Tomita K, Nitta K, et al. Revised equations for estimated GFR from serum creatinine in Japan. Am J Kidney Dis. 2009;53:982–92.
7. Wu HHL, Kalra PA, Chinnadurai R. New-onset and relapsed kidney histopathology following COVID-19 vaccination: a systematic review. Vaccin (Basel). 2021;9:1252.
8. Patel C, Shah HH. Vaccine-associated kidney diseases: a narrative review of the literature. Saudi J Kidney Dis Transpl. 2019;30:1002–9.
9. Li NL, Coates PT, Rovin BH. COVID-19 vaccination followed by activation of glomerular diseases: does association equal causation? Kidney Int. 2021;100:959–65.
10. Li Y, Rao M, Xu G. New-onset acute kidney disease post COVID-19 vaccination. Vaccin (Basel). 2022;10:742.
11. Shetty AA, Tawhari I, Safar-Boueri L, Seif N, Alahmadi A, Gargiulo R, et al. COVID-19-associated glomerular disease. J Am Soc Nephrol. 2021;32:33–40.
12. Houot R, Levy R, Cartron G, Armand P. Could anti-CD20 therapy jeopardise the efficacy of a SARS-CoV-2 vaccine? Eur J Cancer. 2020;136:4–6.
13. Waldman M, Soler MJ, García-Carro C, Lightstone L, Turner-Stokes T, Griffith M, et al. Results from the IRoc-GN international registry of patients with COVID-19 and glomerular disease suggest close monitoring. Kidney Int. 2021;99:227–37.