



Kidney complications following COVID-19 vaccination; a review of the literature

Shakiba Hassanzadeh¹, Arjang Djamali², Leila Mostafavi³, Aiyoub Pezeshgi^{4*}

¹Division of Pathology, Nickan Research Institute, Isfahan, Iran

²Division of Nephrology, Department of Medicine, School of Medicine and Public Health, University of Wisconsin, Madison, Wisconsin, USA

³Department of Radiology, Massachusetts General Hospital, Harvard Medical School, Boston, MA, USA

⁴Department of Internal Medicine and Zanjan Metabolic Disease Research Center, School of Medicine, Zanjan University of Medical Sciences, Zanjan, Iran

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ABSTRACT

Objective: To review the reported cases of kidney injury following vaccination for coronavirus disease 2019 (COVID-19) with a focus on renal pathology.

Methods: We searched for case reports of kidney complications after COVID-19 vaccine in PubMed.

Results: A total of 36 articles including 49 case reports were reported. These included minimal change disease (n=17), IgA nephropathy (IgAN) (n=15), IgA nephritis/vasculitis (n=5), ANCA glomerulonephritis/vasculitis (n=5), anti-glomerular basement membrane (GBM) nephritis (n=2), and 1 case of each granulomatous vasculitis, acute tubulointerstitial nephritis, scleroderma renal crisis, IgG4-related disease nephritis, and primary membranous nephropathy (MN).

Conclusion: We give an overview of the reported cases of post-COVID-19 renal complications. Further investigations of the underlying pathogenesis of post-COVID-19 vaccination renal adverse events are required, as prompt workup, diagnosis, and treatment of patients with renal complications may lead to complete remission, prevent kidney failure, and long-term complications such as end-stage renal disease (ESRD). However, these complications are overall extremely rare and the benefit of vaccination outweighs the potential risks.

Implication for health policy/practice/research/medical education:

Immediate consideration, diagnosis, and treatment of renal complications in the post-COVID-19 vaccination period may prevent long-term renal complications such as end-stage renal disease. In addition, the pharmacovigilance of COVID-19 vaccines may help determine the incidence of side effects.

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Introduction

The coronavirus disease 2019 (COVID-19) is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and began in Wuhan, China in December 2019 and a few months later in March 2020 the World Health Organization (WHO) declared it as a pandemic (1). The COVID-19 infection affects the lung and causes diffuse alveolar damage resulting in an acute respiratory distress syndrome (2). However, it may involve other organs such as the kidneys (3). The development of acute kidney injury (AKI) in patients with COVID-19 infection ranges from 1% to 46% (1).

The most common kidney biopsy findings associated with COVID-19 infection are acute tubular damage, collapsing glomerulopathy (CG) that is a variant of focal

segmental glomerulosclerosis (FSGS), and thrombotic microangiopathy (TMA). In addition, acute tubular injury (ATI) has been reported to be the most common finding in patients with COVID-19 and AKI (3).

There have been a few glomerular diseases associated with COVID-19 infection including CG, minimal change disease (MCD), FSGS, vasculitis (including anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis, anti-glomerular basement membrane (GBM) disease, and immunoglobulin A (IgA) vasculitis with nephritis), membranous nephropathy (MN), lupus nephritis, and TMA. In addition, there have been reports of mixed pathologic renal lesions, acute interstitial nephritis (AIN) as secondary findings, and treatment-related AKI in COVID-19 patients (3).

*Corresponding author: Aiyoub Pezeshgi, Email: Dr.a.pezeshki@gmail.com

Currently, different types of COVID-19 vaccines are being used around the world. The Pfizer BNT162b2 (Pfizer-BioNTech) and Moderna (mRNA-1273) are mRNA vaccines. They both use a lipid nanoparticle nucleoside-modified mRNA encoding the SARS-CoV-2 spike (S) protein that is involved in the host attachment and viral entry. The Astra-Zeneca vaccine is a chimpanzee adenovirus vector that is deficient in replication and contains the SARS-CoV-2 spike (S) protein (4). mRNA vaccines have minimal risk of infection and insertion-induced mutagenesis, produce antiviral neutralizing immunoglobulins (Igs) and stimulate strong immune responses by activating both CD8+ and CD4+ T cells (5). It has been reported that mRNA COVID-19 vaccine results in antibody production, activation of virus-specific CD4+ and CD8+ T cells, and release of cytokines such as interferon- γ (IFN- γ). IFN- γ along with type I INFs inhibits the replication of the SARS-CoV2 virus (6).

With millions of COVID-19 vaccinations administered globally, side effects are being reported (7, 8). In addition, there are concerns about the possibility that COVID-19 vaccines might cause immunologic events, such as autoimmunity or exacerbation of pre-existing autoimmune disorders (9-11). Therefore, pharmacovigilance of COVID-19 vaccines seems important for determining the incidence of the potential complications.

Herein, we give an overview of the reported cases of renal complications following different types of COVID-19 vaccinations.

Methods and Results

To identify the reported cases of renal complications following COVID-19 vaccinations, we conducted a search for relevant case reports in PubMed Central. 36 articles were identified including a total of 49 reported cases (Table 1). These included MCD (n = 17) (12-25), IgA nephropathy (IgAN) (n = 15) (26-34), IgA nephritis/vasculitis (n = 5) (10,30,35,36), ANCA glomerulonephritis/vasculitis (n = 5) (7,8,35,37,38), anti-GBM nephritis (n = 2) (28, 39), 1 case of each granulomatous vasculitis (40), acute tubulointerstitial nephritis (ATIN) (41), scleroderma renal crisis (42), IgG4-related disease nephritis (43), and primary MN (11).

Minimal change disease

MCD has been associated with infections such as viral, parasitic, and mycoplasma pneumonia (44). Recently, there have been reports of MCD in COVID-19 infection (45,46). Kudose et al reported a patient that had MCD, ATI, and endothelial tubuloreticular inclusions (TRIs) following COVID-19 infection. The authors suggested that since there is an association between IFN therapy and MCD, and there are TRIs (also known as IFN footprints) in this patient, cytokines may have a role in the pathogenesis of this patient, who is genetically susceptible, by inducing podocyte injury in COVID-19 infection

(45). Molecular mimicry between the SARS-CoV-2 spike protein and self-antigens on podocytes, cytokine release, and abnormal activation of the immune system may be possible underlying causes of MCD following COVID-19 infection (18,46).

Furthermore, MCD has been associated with various vaccines including influenza, tetanus-diphtheria-polio, pneumococcal, and recombinant hepatitis B vaccinations (47). This exact pathogenesis of MCD following vaccinations is still unclear. A possible immune response after vaccination causing MCD has been suggested. Systemic dysfunction of the T-cells promotes the production of a glomerular permeability factor that induces the fusion of foot processes. This leads to significant changes in the glomerular filter system and, in turn, proteinuria. In addition, podocytes have cytokine receptors; therefore, the immune response after vaccination may influence the cytoskeleton of podocytes, change the podocyte integrity, and result in proteinuria (48).

There have been 17 reports of new-onset/relapse of MCD following COVID-19 vaccinations. Among these, 8 cases were new-onset MCD following Pfizer-BioNTech, Moderna, and Oxford University-AstraZeneca COVID-19 vaccines (12-18). The other 9 reports were MCD relapse cases following Pfizer-BioNTech, AstraZeneca, Moderna, and an inactivated COVID-19 vaccine (20-25).

The exact pathogenesis of MCD following COVID-19 vaccination is still unknown. The development of MCD following mRNA vaccinations does not seem to be due to a direct effect of the vaccine (18). It has been suggested that mRNA COVID-19 vaccines induce an immune response, T-cell activation, cytokine release, and strong antibody response (6,18,20,22). In addition, T cell-mediated immune response to viral mRNA has been suggested to be a possible trigger for podocytopathy (12,13,23). Foot process effacement is the main feature of MCD; therefore, the pathogenesis of MCD may involve the disruption in the integrity of the glomerular filtration barrier. It has been suggested that circulating factors may increase the permeability of the glomerular filtration barrier leading to proteinuria (44). Additionally, it has been reported that during the active phase of MCD, an imbalance in T-cell subpopulations, mostly circulating CD8+ T suppressor cells, aggravate the nephrotic syndrome by cytokine-mediated impairment in mouse models (Th2; IL4, IL5, IL9, IL10, and IL13), which has also been reported by another study on idiopathic nephrotic syndrome in the Buffalo/Mna rat (44,49-51). Interestingly, Özkan et al reported a case of MCD following an inactivated COVID-19 vaccination (25). Moreover, allergies have been reported to cause MCD; therefore, hypersensitivity reactions to vaccines may result in MCD (25). Lebedev et al and Morlidge et al raised questions about the mechanisms involved in the timing of the mRNA vaccinations and the development of MCD that whether cell-mediated

Table 1. Reported cases of the renal complications following COVID-19 vaccinations

Reported cases of the renal complications following COVID-19 vaccinations	Total number of reported cases	Vaccination types	New-onset/relapse/flare occurrences	Number of new-onset/relapse cases	Vaccination types and the number of the new-onset/relapse/flare cases
Minimal change disease	17	<ul style="list-style-type: none"> • Pfizer-BioNTech • Moderna • AstraZeneca • Inactivated SARS-CoV-2 vaccine 	New-onset	8	<ul style="list-style-type: none"> • 5 Pfizer-BioNTech (12-15, 19) • 1 Moderna (16) • 2 Oxford-AstraZeneca (17, 18)
			Relapse/Flare	9	<ul style="list-style-type: none"> • 5 Pfizer-BioNTech (19-23) • 2 AstraZeneca (24) • 1 Moderna (19) • 1 inactivated SARS-CoV-2 vaccine (25)
IgA nephropathy	15	<ul style="list-style-type: none"> • Pfizer-BioNTech • Moderna 	New-onset	1	Moderna (34)
			Relapse/Flare	14	<ul style="list-style-type: none"> • 7 Pfizer-BioNTech (27-29, 31, 33) • 7 Moderna (26, 29, 30, 32)
Primary membranous nephropathy	1	Sinovac (11)	Relapse	-	-
Anti-glomerular basement membrane nephritis	2	<ul style="list-style-type: none"> • Pfizer-BioNTech • Moderna 	New-onset	2	<ul style="list-style-type: none"> • 1 Pfizer-BioNTech (28) • 1 Moderna (39)
IgG4 related disease nephritis	1	Pfizer-BioNTech (43)	Relapse	-	-
IgA vasculitis	5	<ul style="list-style-type: none"> • Pfizer-BioNTech • Moderna 	New-onset	3	<ul style="list-style-type: none"> • 2 Moderna (30, 35) • 1 Pfizer-BioNTech (36)
			Relapse/Flare	2	2 Moderna (10, 30)
ANCA glomerulonephritis/vasculitis	5	<ul style="list-style-type: none"> • Pfizer-BioNTech • Moderna • Oxford-AstraZeneca 	New-onset	5	<ul style="list-style-type: none"> • 2 Moderna (7, 35) • 2 Pfizer-BioNTech (8, 37) • 1 Oxford-AstraZeneca (38)
Granulomatous vasculitis	1	AstraZeneca (40)	New-onset	-	-
Acute tubulointerstitial nephritis	1	Pfizer-BioNTech (41)	New-onset	-	-
Scleroderma renal crisis	1	Pfizer-BioNTech (42)	Flare	-	-

response occurs as early as 3-4 days after vaccination (12,24). For example, Morlidge et al (24) suggested that at two days after vaccination, the vaccine may trigger a cytokine-mediated response (52), and symptoms after four days may be due to a T cell-mediated response to viral mRNA (12,13,53). Nevertheless, there have been reports that the development of the symptoms of MCD following vaccinations may range from 4 days to 4 months after the vaccination administration (48). Furthermore, Lebedev et al and Leclerc et al pointed out that unlike these cases of MCD following mRNA vaccination, MCD is not usually associated with AKI, although it may be a complication of MCD (12,18). Nonetheless, further studies are required to investigate the exact pathogenesis of MCD following COVID-19 vaccination.

IgA nephropathy

IgAN is associated with bacterial and viral upper respiratory or gastrointestinal infections such as cytomegalovirus, toxoplasma, adenovirus, herpes simplex virus, *Haemophilus parainfluenza*, and *Staphylococcus aureus* (54). There is mesangial IgA1 deposition with/without IgG and C3 deposits in IgAN patients. The pathogenesis of IgAN involves poorly-galactosylated IgA1 in the serum and the production of glycan-specific IgG or IgA against them that result in the formation of immune complexes. In addition, there are increased affinity for mesangial cells and deposition in the glomeruli. It is possible that, other factors are also involved in the pathogenesis of IgAN such as gene variants that are triggered with infections or other environmental factors that lead to the production of anti-glycan IgG or IgA (34). Furthermore, it has been reported that severe COVID-19 infection is associated with increased levels of IgA and antiphospholipid IgA (IgA-aPL) antibodies. This association, which is probably triggered in the bronchial mucosa, may have a role in the systemic complications and autoimmunity in patients with COVID-19 infection (55). Likewise, the receptor-binding domain (RBD) of the viral spike protein is a highly specific target of neutralizing antibodies in COVID-19 patients which suggests the presence of an association between them (56).

There are rare reports on IgAN development following vaccinations. Owda and Turney reported a case of IgAN following typhoid vaccination who had a history of nephritis without any relapse (57). In an experimental study, Kavukçu et al showed that repeated vaccination with conjugated *Haemophilus influenzae* type B vaccine (PRP-T), which contains capsular polysaccharides of the organism (PRP) conjugated to tetanus protein (T), causes IgAN in mice (58). However, IgA vasculitis/Henoch-Schönlein purpura (HSP) has been reported following various vaccination administration such as influenza, hepatitis A, and meningococcal vaccines (59-61). In addition, patients with primary IgAN have been reported

to have a significant increase in the IgA1 subclass and have a stronger IgA (IgA1) response to the influenza vaccine (62). In addition, there have been reports of IgA vasculitis following COVID-19 vaccinations (see details in section IgA vasculitis) (10,30,35,36).

There have been 15 reports of relapse/new-onset of IgAN following COVID-19 vaccinations. Among the cases, 14 individuals were IgAN relapses following Pfizer-BioNTech and Moderna vaccinations in adult patients (26-30,32). Two of the other case were IgAN relapse following Pfizer-BioNTech vaccinations in pediatric patients (31) There has only been one report of a new-onset IgAN following Moderna vaccination (34).

The exact underlying pathogenesis of IgAN following COVID-19 vaccinations is unknown. Perrin et al suggested that IgAN cases following COVID-19 vaccinations seem to be unrelated to the anti-SARS-CoV-2 antibody response (29). Rahim et al suggest that the IgAN flares following the second dose of COVID-19 vaccinations may be induced by a delayed-type hypersensitivity reaction (27). Kudose et al suggested that an immune response to vaccination with a systemic cytokine-mediated flare may exacerbate a pre-existing IgAN by inducing IgA1 anti-glycan immune responses (32). An experimental study of mRNA vaccines on mice and non-human primates showed that compared to adjuvant protein and inactivated virus vaccines, mRNA vaccines induce increased levels of T follicular helper (Tfh) and germinal center (GC) B cells, antigen-specific CD4+ T cell responses, neutralizing antibody responses, and long-term B cell memory (63). In this regard, Sahin et al reported that mRNA COVID-19 vaccine results in antibody production, activation of virus-specific CD4+ and CD8+ T cells, and release of cytokines such as IFN- γ . IFN- γ is an important cytokine and with type I INFs inhibits the replication of the SARS-CoV2 virus (6). Another study suggested that COVID-19 mRNA vaccines induce spike-antigen-specific IgG and IgA responses (64). Furthermore, Abramson et al reported the first and only case of a new-onset IgAN following COVID-19 vaccination. They suggested that mRNA vaccines, similar to COVID-19 infection, produce strong antibodies against the RBD of the S1 protein. Therefore, the glycosylation of RBD and the immune response from the mRNA vaccine may be the underlying cause of the production of antibodies that resulted in the deposition of IgA immune-complexes in this patient (34). Moreover, severe COVID-19 infection is associated with a strong IgA response that may be triggered in the bronchial mucosa (55). However, since vaccinations do not stimulate mucosal immune responses, it is unclear if there is any association between COVID-19 vaccinations and IgAN (27, 34). Abramson et al suggested that the development of IgAN following COVID-19 vaccination may be due to the production of anti-glycan antibodies in these patients that cross-react with pre-existing poorly-galactosylated

IgA1 (34). The exact pathogenesis of IgAN following COVID-19 vaccinations is still unclear and requires further investigation.

Primary membranous nephropathy

MN has been associated with various infectious diseases such as HBV, HCV, HIV, parasites, leprosy, syphilis and hydatid disease (65). Kudose et al reported two cases of MN following COVID-19 infection. One of these patients was phospholipase A2 receptor antibody (PLA2R)-positive and the other one was PLA2R-negative. PLA2R is the major target antigen in MN, which is also present in the respiratory tract. Therefore, they suggested that this could be a potential source for antigen presentation to induce or reinforce autoimmune responses against PLA2R, although this association could also be coincidental (45). Moreover, there was another report of a patient with MN following COVID-infection which was PLA2R-negative. The authors suggested that it was possibly due to an exaggerated immune response associated with COVID-19 infection (66).

In addition, Aydin et al mentioned observing two patients who developed anti-PLA2R-positive MN after COVID-19 infection and suggested that a loss of tolerance to the PLA2R antigen may be due to the COVID-19 virus (11).

There have been reports of MN following influenza vaccination (47,67). Patel et al suggested that although the exact pathogenesis of MN following influenza vaccination is unclear, but activation of the immune system triggered by the vaccine may be the cause, as the interaction between the antigen of the influenza vaccine and native antibodies, form or deposit immune complexes in the subepithelial space (67).

To our knowledge, to date, there has been only one report of a relapse of MN by Aydin et al following Sinovac vaccination (an inactivated SARS-CoV-2 virus vaccine) (11).

Anti-glomerular basement membrane nephritis

Canney et al showed that spatial and temporal clustering of anti-GBM cases is associated with an environmental trigger leading to the disease occurrence (68). In addition, there have been reports of an association between anti-GBM disease/Goodpasture's syndrome with influenza infection, and also seasonal outbreaks of the disease associated with viral infections (69, 70). Furthermore, there have been reports of new-onset and recurrence of anti-GBM disease following COVID-19 infection (45,71). Predecki et al demonstrated cases with positive IgM and IgG antibodies of the COVID-19 virus and negative viral RNA and suggested that the viral infection triggers an adaptive immune response that targets the basement membrane (71).

Moreover, Norton et al reported a case of double

seropositive vasculitis following influenza vaccination who was positive for both MPO-ANCA and anti-GBM antibody (72). There have been several hypotheses regarding the occurrence of vasculitis and autoimmune diseases following influenza vaccine such as molecular mimicry or autoimmune syndrome induced by vaccine adjuvants. The authors suggested that in this case, the influenza vaccine may have caused an immunological response stimulating an inflammatory procedure resulting in the double seropositive vasculitis and AKI (72).

Currently, there have been two reports of anti-GBM nephritis following Pfizer-BioNTech and Moderna vaccines (28, 39).

The exact pathogenesis of anti-GBM nephritis following mRNA COVID-19 vaccinations is still unclear, although it has been suggested that it may be due to an immune response to the vaccines (39). Nonetheless, further studies are needed to investigate the cause of this association.

IgG4 related disease (IgG4-RD) nephritis

IgG4-RD is characterized by chronic activation of the immune system and fibrosis. It affects many organs including the kidney. Although it may be an autoimmune disease, the exact etiology is still unknown. In addition, in patients with IgG4-RD, increased IgE and autoantibodies of the IgE isotype have been reported, although the exact association of IgE and IgG4-RD is still unclear (73). To our knowledge, there has only been one report of a relapse of IgG4-RD nephritis following COVID-19 vaccination (43).

Masset et al suggested that there is a possible link between the relapses of immune diseases following mRNA vaccinations. However, they argued that it is still unclear whether the relapses are due to a direct immune activation triggered by the vaccination, or due to a chronic immune activation triggered by an allergic reaction, or both of these (43). The pathogenesis and the association between IgG4-RD nephritis and COVID-19 mRNA vaccination require further investigation.

IgA nephritis/vasculitis

There have been reports of IgA vasculitis/ HSP following bacterial and viral infections, such as streptococcal and human parvovirus B19 infections (74,75). Additionally, there have been case reports of IgA vasculitis/HSP following COVID-19 infection (76-78). It has been suggested that in infections, there is an increase in galactose-deficient IgA1 (gd-IgA1) that recognizes microorganisms and, in turn, develops complexes that deposit and provokes the mesangial cells leading to renal damage. In addition, mucosal infections increase the level of IL-6, which may result in the production of gd-IgA1 and cause renal damage (76,79).

Furthermore, the development of HSP (IgA vasculitis) has been reported following various vaccinations such as influenza, hepatitis A, and meningococcal vaccines (59-

61). Since there is a structural similarity between influenza antigens and vaccine proteins, it has been suggested that the link between vaccines and autoimmunity may be caused by immune responses to the infectious antigen or other vaccine components (80). In addition, it has been reported that patients with primary IgAN have a significant increase in the IgA1 subclass and have a stronger IgA (IgA1) response to the influenza vaccine (62). Besides, there have been reports of IgAN following COVID-19 vaccinations [see details above in section IgAN] (26-32,34).

To our knowledge, there have been three reports of new-onset IgA vasculitis/HSP and two reports of a flare-up/reactivation of IgA vasculitis following mRNA COVID-19 vaccinations (10,30,35,36).

The exact pathogenesis of IgA vasculitis following COVID-19 vaccination is still unknown. Anderegg et al suggested that the trigger in predisposed individuals may be an immune response to the spike protein or the mRNA of the COVID-19 vaccine (35). Obeid et al discussed that in patients with pre-existing IgA vasculitis following COVID-19 vaccination, there may be an association between the increase in anti-SARS-CoV-2 spike IgA and the relapse of pre-existing IgA vasculitis. However, they reported that it is unclear whether the activation of autoreactive B-cells after vaccination is due to the previous autoreactive B-cells producing IgA, new autoreactive B-cells producing IgA, or a combination of both of these (10). Further studies are required to investigate the exact pathogenesis of IgA vasculitis following COVID-19 vaccination.

ANCA glomerulonephritis/vasculitis

Associations between various infections and ANCA-associated vasculitis have been previously reported (81). Recently, ANCA-associated vasculitis has been reported in patients with COVID-19 infection (82,83). COVID-19 infection has been reported to trigger autoimmunity, production of autoantibodies, and autoimmune disorders. The mechanism may be due to some receptors that recognize and bind to the virus RNA which lead to the production of type I interferon (type I IFN) and pro-inflammatory cytokines which are associated with autoimmune diseases, and by activating the transcription factor NF- κ B, the production of type I IFN, interleukin-6 (IL-6), and other pro-inflammatory cytokines are triggered. Furthermore, activation of type I IFN-mediated B-cells result in autoantibody production (84).

In addition, there have been reports of ANCA-associated vasculitis following influenza vaccination (80, 85). Jeffs et al suggested that influenza RNA triggers the production of PR3-ANCA and also activates the Toll-like receptor-7 (TLR7) (RNA is the natural ligand for TLR7). Further, they suggested that the RNA in vaccines, in genetically predisposed individuals, may trigger autoimmunity.

Interestingly, they reported that after treatment with RNase, there was a significant decrease in the PR3-ANCA response to the RNA (85). On the other hand, other studies suggest that influenza vaccination is safe in ANCA-associated vasculitis patients that are in remission (86).

To our knowledge, to date, there have been 5 reports of ANCA vasculitis following COVID-19 vaccinations (2 after Moderna, 2 after Pfizer-BioNTech, and 1 after Oxford-AstraZeneca) (7,8,35,37,38).

The exact pathogenesis of ANCA-associated vasculitis following COVID-19 vaccination is still unknown. Sekar et al suggested that a neutrophilic immune response to mRNA vaccine may trigger ANCA glomerulonephritis which after the second dose and an increase in the immune response, production of PR3 antibodies are triggered (7). Anderegg et al and Shakoor et al suggested that the trigger in predisposed individuals may be an immune response to spike protein or mRNA of COVID-19 vaccine (8,35). However, further investigations regarding the exact cause of ANCA-associated vasculitis following COVID-19 vaccination are required.

Granulomatous vasculitis

Infections such as mycobacterium tuberculosis, mycobacterium leprae, histoplasmosis, candidiasis, toxoplasmosis, Escherichia coli, and Epstein-Barr virus may cause granulomatous interstitial nephritis (GIN) (87). In addition, there has been a report of the development of GIN with some ATI in a patient with COVID-19 infection. The patient had normal kidney function on admission but during his admission, he developed acute kidney failure that required dialysis. In addition, he developed a drug-induced skin rash (possibly due to amoxicillin) simultaneously with his AKI. Therefore, the authors suggested that the cause of GIN in this patient may be drug-related that resulted in AKI. However, they debated that it is unclear that if the COVID-19 inflammation process had affected the disease phenotype or that COVID-19 infection was the cause of the disease itself (88).

In addition, GIN has been reported following treatment with intra-vesical bacillus Calmette-Guérin (BCG) in bladder cancer. This may be due to vesicoureteral reflux or a hematogenous spread of the attenuated bacilli. In addition, in tissues, the attenuated bacilli multiply uncontrollably and cause necrotizing granulomatous reaction (89).

Gillion et al reported a case of acute granulomatous nephritis associated with vasculitis following the first dose of AstraZeneca vaccination (40). The pathogenesis of this disease following COVID-19 vaccination is unclear.

Acute tubulointerstitial nephritis

Various infections have been associated the with

tubulointerstitial nephritis (TIN) such as cytomegalovirus, Epstein-Barr virus, hepatitis, human immunodeficiency virus, and BK polyomavirus, brucellosis, campylobacter, legionella, salmonella, streptococcus, Mycoplasma pneumonia, Mycobacterium tuberculosis, histoplasmosis, leptospirosis, candidiasis, and toxoplasmosis. The exact pathogenesis of infection-associated TIN is unclear; however, it has been suggested that direct damage by the organism or circulating immune complexes may be involved (90). On the other hand, many drugs are also associated with TIN, such as analgesics, antibiotics, antivirals, antiepileptics, antacids, diuretics, allopurinol, cyclosporine, azathioprine, and sulfasalazine (90). Additionally, Panchangam et al reported a case of statin-induced AIN and rhabdomyolysis. The patient had been using atorvastatin which resulted in myopathy and interstitial nephritis followed by AKI (91). Moreover, AIN has been reported to be the most common renal immune-related adverse events (irAE) of immune checkpoint inhibitors (ICIs) (92). However, unlike other drug-induced AINs that are usually due to a type 4 hypersensitivity reaction or delayed hypersensitivity reaction, ICI-induced AIN may be caused by a loss of tolerance against renal antigens (92,93).

Furthermore, Buyansky et al reported a case of ATIN that was triggered by a COVID-19 infection in a patient using an ICI drug. The patient had normal kidney function before admission and was treated with durvalumab, which is an anti-programmed death-ligand 1 (anti-PD-L1) drug. Investigation of the kidney tissue by immunochemistry did not show any SARS-CoV-2 suggesting that ATIN in this patient was not caused by direct SARS-CoV-2 infection of the kidney. The authors suggested that SARS-CoV-2 has caused an immune-regulatory imbalance which triggered ATIN in this patient. Systemic viral infections induce pro-inflammatory reactions that may increase the disruption of self-tolerance and immunity in patients treated with ICIs resulting in cross-reactivity and, in turn, ATIN. However, this patient was administered cloxacillin a few months before her admission which may have had some involvement in the immune-regulatory imbalance (94).

Merino et al reported a case of ATIN following COVID-19 vaccination with Pfizer-BioNTech vaccine. However, it is worth mentioning that the patient was using drugs such as allopurinol, which may have also contributed to the development of ATIN in this patient (41). However, the association of ATIN following COVID-19 vaccination requires further investigation.

Scleroderma renal crisis

A patient with a history of limited cutaneous systemic sclerosis (SSc) was reported to develop

scleroderma renal crisis (SRC) that was complicated with TMA following an Influenza B infection. He did not have any of the known risk factors for SRC and, therefore, the authors suggested that influenza infection could induce

SRC complicated with TMA in patients diagnosed with SSc (95). The pathogenesis of SRC includes endothelial damage, intimal proliferation, and narrowing of the kidney arteries resulting in decreased renal blood flow. As a result, hyperplasia of the juxtaglomerular apparatus occurs leading to increased levels of renin resulting in acute hypertension and renal dysfunction (95).

Furthermore, there has been a case report of systemic sclerosis following a mild COVID-19 infection in a previously healthy patient. However, his renal function was normal, and did not have SRC at admission. The authors suggested that there are some similarities between COVID-19 infection and systemic sclerosis. For example, both may become systemic, have increased levels of IL-6, IL-10, and MCP-1, as well as cause endothelial damage and interstitial lung fibrosis (96). In addition, Ferri et al reported that COVID-19 infection in systemic sclerosis patients may cause direct tissue damage by SARS-CoV-2. On the other hand, they suggested that virus may aggravate the existing presentations of systemic sclerosis during the acute phase of COVID-19 infection. However, it may lead to complicated organ damage in the long-term (97).

Oniszczyk et al reported a case of SRC following mRNA Pfizer-BioNTech COVID-19 vaccination which revealed an undiagnosed and pre-existing diffuse cutaneous systemic sclerosis (42). The association and pathogenesis of SRC following COVID-19 vaccination require further investigation.

Conclusion

The analysis and investigations of the complications and adverse events induced by COVID-19 vaccines could increase our understanding of the underlying pathogenesis of post-COVID-19 vaccination complications. Immediate consideration, diagnosis, and treatment of renal complications in the post-COVID-19 vaccination period may prevent long-term renal complications such as ESRD. In addition, the pharmacovigilance of COVID-19 vaccines may help determine the incidence of side effects. However, these complications are overall extremely rare and the benefit of vaccination outweighs the potential risks.

Authors' contribution

SH conducted the primary draft. AP conducted the primary edit. ADJ and LM conducted the secondary edits. All authors read and signed the final paper.

Conflicts of interest

The authors declare that they have no competing interests.

Ethical issues

Ethical issues (including plagiarism, data fabrication, double publication) have been completely observed by the authors.

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