



Commentary

Clinicopathologic Spectrum of Renal Disease in SARS-CoV-2 Infection and Post-COVID-19 Vaccination

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Abstract

Both SARS-CoV-2 infection and COVID-19 vaccination have been associated with renal complications. In the context of SARS-CoV-2 infection, kidney injury is primarily attributed to a complex interplay of systemic immune dysregulation and vascular damage. Severe infection triggers a cytokine-mediated inflammatory response characterized by elevated levels of pro-inflammatory mediators, resulting in systemic hemodynamic instability, increased capillary permeability, and renal hypoperfusion, ultimately leading to acute tubular injury. In addition, virus-induced endothelial activation promotes a prothrombotic state, microvascular injury, and further impairment of renal perfusion. Collectively, these processes converge to produce acute kidney injury (AKI) and, in severe or prolonged cases, may contribute to chronic tubulointerstitial damage and progressive renal dysfunction. In contrast, renal manifestations following COVID-19 vaccination are relatively uncommon and are thought to arise primarily from immune-mediated dysregulation rather than direct cytopathic effects. Reported cases include minimal change disease, IgA nephropathy, focal segmental glomerulosclerosis (FSGS), acute interstitial nephritis and other glomerulopathies, which present as either new-onset disease or relapses. These lesions are thought to result from transient immune activation following vaccination, including T-cell stimulation and altered humoral responses, which may trigger or unmask an underlying susceptibility to renal injury. Careful post-vaccination monitoring in high-risk populations may be warranted, and further molecular and population-level studies are needed to elucidate the underlying mechanisms and refine risk assessment.

Keywords: COVID-19; vaccine; kidney; adverse events; outcome



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1. Introduction

Both SARS-CoV-2 infection and COVID-19 vaccines can potentially impact renal function through distinct but overlapping mechanisms, with infection posing a greater risk for acute kidney injury (AKI) and long-term renal dysfunction than vaccination does. Both infection and vaccination stimulate the immune system, but infection intensifies and unpredictably, potentially leading to more severe renal injury. Compared with vaccination, SARS-CoV-2 infection triggers a more robust and potentially harmful inflammatory response, including cytokine storms. The virus can directly damage the vascular endothelium and kidney cells, whereas vaccines may affect renal function through altered immune responses.

COVID-19 vaccination has been reported to induce various renal injuries, including minimal change disease, IgA nephropathy, ANCA-associated vasculitis, and acute interstitial nephritis. Although it is too early to estimate the frequency and outcomes of COVID-19 vaccine-induced renal injury, 69% of the 130 reported cases were new-onset kidney diseases, and 31% were relapses of preexisting conditions. AKI and collapsing glomerulopathy are common renal lesions in patients with COVID-19. Both SARS-CoV-2 infection and COVID-19 vaccines can potentially cause kidney inflammation, blood pressure dysregulation, coagulopathy, and AKI. Healthcare providers should monitor patients with existing kidney disease after COVID-19 vaccination, as there is a potential risk of worsening renal function. The exact pathomechanism is not fully clear, but immune activation following SARS-CoV-2 infection or vaccination might trigger *de novo* or exacerbate existing kidney diseases. Additionally, inflammatory responses generated by SARS-CoV-2 infection or vaccination might have adverse effects on various components of the kidney, particularly among vulnerable individuals. The varying renal disorders caused by either SARS-CoV-2 infection or COVID-19 vaccination and the potential clinical impacts of vaccination need further study. From a clinical perspective, healthcare providers should be aware of the possibility of vaccine-related renal injuries; patients who develop renal discomfort and dysfunction after vaccination should be promptly evaluated and treated to prevent further renal deterioration.

A potent vaccine is indeed an effective way to control infectious diseases such as COVID-19. The effectiveness of COVID-19 vaccines depends on several factors, including overall health and the preexisting conditions of an individual [1,2]. Healthy individuals generally experience better tolerance and greater benefits from COVID-19 vaccination, as they are more likely to generate a robust immune response, resulting in better overall protection against infection and/or severe disease. Individuals with chronic diseases or a compromised immune system may have a less optimal response to vaccination. This can potentially result in a decline in vaccine effectiveness. Although studies are underway to evaluate the long-term safety of COVID-19 vaccines beyond initial, relatively shorter clinical trials, the risk of severe side effects may increase with multiple booster doses of the COVID-19 vaccine. The likelihood that COVID-19 vaccines might worsen existing health conditions in certain vulnerable groups highlights the need for medical advice before receiving additional vaccine doses. Renal adverse events have been reported after all types of COVID-19 vaccines were administered. An analysis of published reports showed that the percentages of reported cases were as follows: mRNA vaccine BNT162b2 (Pfizer—BioNTech): 56% (72 out of 128 cases), mRNA-1273 (Moderna—NIAID): 30% (38 cases), ChAdOx1-S (AstraZeneca—Oxford): 10.2% (13 cases), and Ad26.COV2. S (Janssen): 1.5% (2 cases) and the inactivated vaccine CoronaVac (Sinovac): 2.3% (3 cases) [3]. The apparent higher prevalence of post-vaccination kidney injury associated with mRNA vaccines may be due to the greater availability and widespread use of the Pfizer (BNT162b2) and Moderna (mRNA-1273) vaccines during the peak of the pandemic. This increased accessibility likely resulted in more people receiving these specific vaccines, potentially leading to a greater number of reported adverse events than with other vaccine types [4]. In terms of clinical relevance, glomerular diseases unrelated to COVID-19 have also been reported after vaccination. For example, the hepatitis B vaccine is linked to nephrotic syndrome, whereas the Bacillus Calmette–Guérin (BCG) vaccine is linked to membranous nephropathy [5,6].

Although renal complications have been described after both SARS-CoV-2 infection and COVID-19 vaccination, earlier publications have generally examined these entities separately [7]. A comparative clinicopathologic review may help clarify the shared and distinct features of clinical presentation, biopsy findings, and underlying mechanisms, thereby

improving diagnosis and patient counseling. This article briefly summarizes renal lesions that may be caused by or linked to either SARS-CoV-2 infection or COVID-19 vaccines.

2. COVID-19 and Kidney Lesions

Information was collected by searching the literature in the PubMed database between January 2020 and December 2024. The keywords used to guide the search were ‘COVID-19 and kidney lesions’, ‘COVID-19 vaccine and kidney’, ‘COVID-19 vaccine and acute kidney injury’, and ‘COVID-19 and acute kidney injury’. This opinion article synthesizes information collected from human studies to briefly summarize COVID-19 vaccination-associated adverse renal events.

The interplay between genetic factors, viral infection, and the immune response contributes to renal injury in COVID-19 patients. COVID-19 has been reported to trigger various forms of nephritis, with collapsing glomerulopathy being the most common [8]. APOL1 genotypes have been identified as major risk factors for COVID-19-induced collapsing glomerulopathy, especially in patients of African descent [9]. In a study of 990 veterans of African ancestry hospitalized with COVID-19, approximately one in eight (12.6%) carried high-risk APOL1 genotypes. Among those individuals with high-risk genotypes, 51.2% developed AKI, and 19.2% died [10]. These results highlight the potential importance of the APOL1 genotype in predicting outcomes for patients of African ancestry who are hospitalized with COVID-19. Other reported types of renal lesions include acute interstitial nephritis, pauci-immune crescentic glomerulonephritis, and thrombotic microangiopathy. More than 25% of patients hospitalized with COVID-19 develop AKI [11]. AKI was observed in 1835 of the 3993 patients hospitalized with COVID-19, representing 46% of the total. Among these AKI patients, 347 patients (19% of those with AKI) experienced severe kidney dysfunction that required dialysis treatment [12]. The “two hits” hypothesis suggests that a combination of genetic predispositions and cytokine-mediated host responses to COVID-19 contributes to renal injury [8,13]. SARS-CoV-2 is likely to induce kidney damage through direct interactions with kidney cells through ACE2 receptors. ACE2 receptors are expressed in various kidney cell types, including proximal tubule epithelial cells, podocytes, mesangial cells and glomerular endothelial cells (Figure 1) [14,15]. Systemic inflammation and cytokine storms, along with endothelial injury and microvascular thrombi, can also intensify renal structural damage. The clinical presentation of COVID-19 can range widely from uremia to proteinuria to hematuria. Currently, kidney biopsy is still the best approach for confirming specific types of kidney damage. In certain populations, genetic testing for APOL1 risk variants should also be considered. Both the diagnosis and treatment of COVID-19-associated renal diseases pose clinical challenges; a carefully balanced approach is needed to assess the risks and benefits of using immunosuppressive treatments in patients with active COVID-19 infections. The short-term outcomes of COVID-19-associated kidney damage are generally poor, with high morbidity and mortality [12,16]. An analysis of 1835 patients with AKI showed that the in-hospital mortality rate was 50% among COVID-19 patients with AKI versus 8% among those without AKI [12]. Further studies are needed to understand the long-term outcomes and optimal management strategies for patients with COVID-19-associated acute and chronic renal diseases.

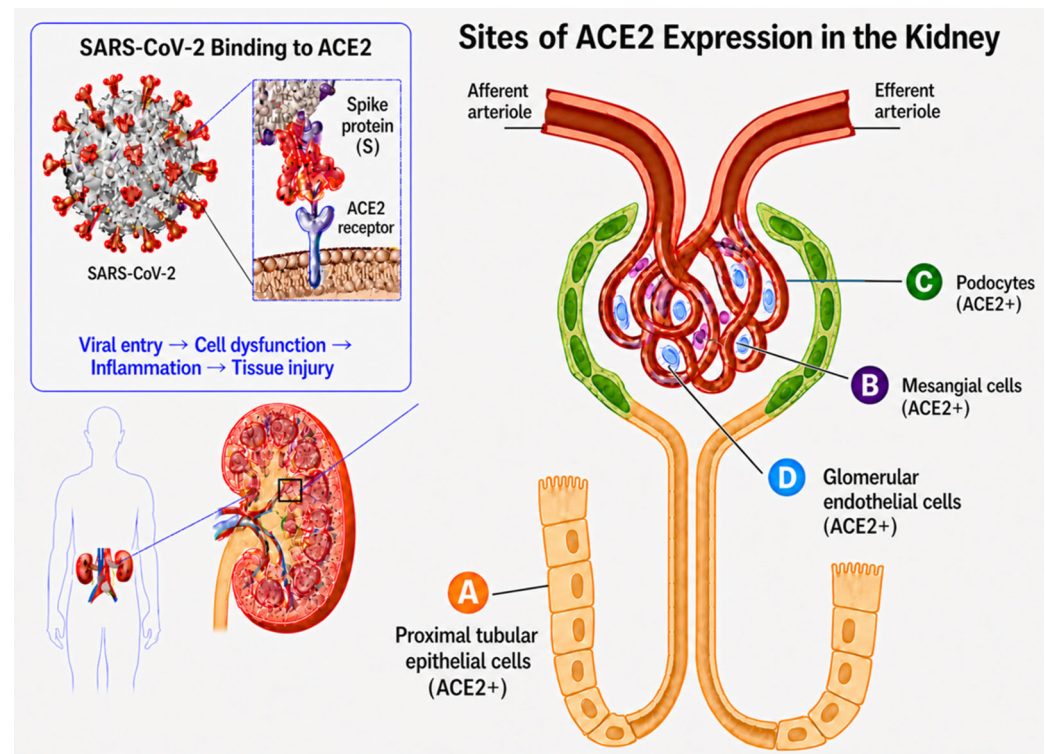


Figure 1. SARS-CoV-2 is thought to contribute to renal injury through direct interactions with renal cells via ACE2 receptors. ACE2 is expressed in multiple renal cell populations, including proximal tubular epithelial cells, podocytes, mesangial cells, and glomerular endothelial cells, thereby providing potential sites for viral binding and subsequent cellular injury.

3. COVID-19 Vaccines and Kidney Lesions

Clinical studies have documented various types of kidney lesions following COVID-19 vaccination (Table 1). Studies have reported minimal change disease (most common), IgA nephropathy, antineutrophil cytoplasmic autoantibody (ANCA) vasculitis, acute interstitial nephritis, crescentic glomerulonephritis, acute tubular injury, and thrombotic microangiopathy [3]. Membranous nephropathy, anti-glomerular basement membrane (anti-GBM) nephritis, and focal segmental glomerulosclerosis (FSGS) are also documented following vaccination. A 21-year-old male with FSGS who underwent kidney transplantation and was stable for the following 8 years experienced a recurrence of FSGS after receiving two doses of the SARS-CoV-2 mRNA vaccine [17]. Current evidence linking COVID-19 vaccination to FSGS is limited to isolated case reports and small case series, and a causal relationship has not yet been established [18]. However, the published literature indicates a temporal association between both *de novo* FSGS and the relapse or recurrence of podocytopathy. A series of biopsy-confirmed kidney diseases have been described following COVID-19 vaccination, suggesting a temporal association with diverse renal manifestations [19]. These include IgA nephropathy, which typically presents with painless gross hematuria and is characterized histologically by mesangial proliferation and dominant glomerular deposition of IgA by immunofluorescence microscopy, and minimal change disease, which typically presents with nephrotic syndrome and shows diffuse podocyte foot process effacement on electron microscopy. COVID-19 vaccination is also linked to thrombotic microangiopathy and acute tubulointerstitial nephritis in patients who present with AKI. In one report, a 44-year-old man developed severe renal dysfunction within one week of receiving the mRNA-1273 SARS-CoV-2 vaccine, with serum creatinine concentration increasing to 4.13 mg/dL, and kidney biopsy subsequently confirmed tubulointerstitial nephritis [19]. In a separate study, a 17-year-old Japanese male

presented with high-grade fever, headache and macrohematuria following a second dose of SARS-CoV-2 mRNA. Renal biopsy of the patient showed mesangial cell proliferation and matrix expansion, endocapillary hypercellularity and crescents, whereas immunofluorescence staining revealed predominantly IgA and C3 deposits in the mesangial areas. The patient was diagnosed with IgA nephropathy, and because of persistent proteinuria and microhematuria, the patient was treated with tonsillectomy with steroid pulse therapy [20]. Notably, an association of IgA nephropathy with vaccines unrelated to COVID-19 has also been reported. For instance, influenza vaccine-linked IgA nephropathy has also been noted in both native and transplanted kidneys [21,22]. An analysis of published cases related to COVID-19 vaccination, showed that 39% (50/128) of patients developed renal symptoms after the first dose, whereas 61% (78/128) of patients developed renal symptoms after the second dose [3]. In a similar line of observation, a 51-year-old healthy Korean man without comorbid diseases experienced body weight gain from 77 kg to 91 kg 20 days after COVID-19 vaccination (Ad26.COV2. S); urinalysis revealed protein 4+ and hematuria; the serum albumin level was low (1.6 g/dL), and the creatinine concentration was 1.13 mg/dL. Renal biopsy, performed 33 days after vaccination, showed diffuse effacement of the podocyte foot processes without any electron-dense deposition (by electron microscopy). Histopathological findings showed that the patient was diagnosed with minimal change disease and was treated with high-dose steroid therapy, which resulted in clinical improvement [23].

Table 1. Partial list of COVID-19 vaccine-induced kidney lesions.

Renal Lesions	Age/Sex (Vaccine Platform)	Morphological Features	References
Acute tubulointerstitial Nephritis	44 yr. Male (mRNA vaccine)	Normal glomeruli, mild tubular atrophy and interstitial fibrosis, with severe inflammatory cell infiltrates in tubular epithelial cells (tubulitis) and interstitium.	[19]
Anti-GBM nephritis	67 yr. Male (mRNA vaccine)	Cellular crescents and fibrinoid necrosis with moderate interstitial fibrosis and tubular atrophy; linear for IgG1 staining of GBM by immunofluorescence microscopy.	[24]
C3 glomerulonephritis	67 yr. Male (Adenoviral vector vaccine)	Interstitial inflammation and proliferation in the mesangium with expansion; moderate C3 staining in glomerular capillary walls and granular staining in blood vessel by immunofluorescence microscopy.	[25]
Chronic thrombotic microangiopathy	69 yr. Female (Adenoviral vector vaccine)	Diffuse thickening of capillary wall with capillary loop doubling, hyaline thrombi in glomeruli, mild infiltration of lymphocytes in the interstitium, and arterial fibrointimal thickening.	[19]

Table 1. Cont.

Renal Lesions	Age/Sex (Vaccine Platform)	Morphological Features	References
Collapsing glomerulopathy	63 yr. Female (Adenoviral vector vaccine)	Segmental glomerular sclerosis, with collapse of capillary loops, podocyte hyperplasia and hypertrophy, along with mild tubular degeneration.	[26]
Crescentic glomerulonephritis	16 yr. Female (mRNA vaccine)	Diffuse fibrocellular to fibrous crescent formation, tubular dilatation, and detachment of epithelial cells, severe interstitial inflammatory cell infiltration, with edema, and mild fibrosis.	[27]
Focal segmental glomerulosclerosis (FSGS)	17 yr. Female (mRNA vaccine)	Glomeruli with segmental sclerosis, synechia, and hyaline deposits in the tubular pole, with mild tubular degenerative changes.	[28]
IgA nephropathy	47 yr. Male (mRNA vaccine)	Mild mesangial expansion and hypercellularity, endocapillary hypercellularity, cellular and fibrocellular crescent formation; diffuse granular mesangial staining for IgA by immunofluorescence microscopy.	[29]
Lupus nephritis	42 yr. Female (mRNA vaccine)	Focal and segmental mesangial hypercellularity; granular immunoreactivity for IgG and C3 along glomerular capillary walls and within the mesangium.	[30]
Membranous nephropathy	70 yr. Male (mRNA vaccine)	Subepithelial electron-dense deposits in GBM with extensive foot process effacement of podocytes by electron microscopy.	[31]
Minimal change disease	52 yr. Male (Adenoviral vector vaccine)	Normal glomeruli, intact tubules and interstitium, with diffuse foot process loss by electron microscopy.	[19]

Of significance, measles and diphtheria pertussis tetanus (DPT) vaccines are also reportedly linked to minimal change disease with urinary protein wasting [32,33]. Furthermore, new-onset lupus nephritis (class III) involving multiple organs was reported following adenoviral COVID-19 vaccination (AZD1222) [34]. One week after receiving the first dose of Pfizer mRNA, a flare-up of a known case of lupus nephritis (class V) has been documented [30]. The patient was treated with immunosuppressive drugs, specifically mycophenolate mofetil and prednisolone; however, protein levels in the urine decreased slowly despite such aggressive treatment [30]. Other vaccines unrelated to COVID-19 have also been linked to lupus nephritis. A unique case of lupus nephritis was reported following hepatitis B vaccination; a 27-year-old woman developed weakness and edema one week after receiving the recombinant hepatitis B vaccine. Laboratory tests revealed low complement levels, positive lupus serology, nephrotic-range proteinuria, hematuria and granular casts in the urine. A kidney biopsy confirmed diffuse proliferative glomerulonephritis (class IV lupus nephritis), and the patient fully recovered after treatment with cyclophosphamide and prednisone [35].

4. Diagnostic Dilemma

Differentiating between vaccine-induced AKI and other causes of AKI can at times be challenging, and clinicians need to consider several factors, including timing: vaccine-

induced AKI typically occurs within days to weeks after vaccination, and most reported cases develop AKI within 1–3 weeks post-vaccination. Additionally, the common causes of AKI, such as dehydration, medications, and infections, should be ruled out, in addition to carefully taking the past and present history, performing thorough physical examination, and working up to exclude other causes. If the etiology remains unclear, especially with severe or prolonged AKI, kidney biopsy needs to be performed; common biopsy findings in vaccine-related cases include acute interstitial nephritis and glomerulonephritis (Figure 2). As mentioned, kidney biopsy is considered the gold standard for diagnosing specific types of kidney diseases, including those potentially induced by vaccines. Biopsy may help to rule out other potential causes of existing kidney injury, in turn strengthening the association with vaccination. While kidney biopsy is invaluable for determining the extent of renal structural damage, it is important to note that it cannot definitively prove causation by the vaccine. The diagnosis of vaccine-induced kidney injury is typically based on a combination of clinical presentation, temporal relationship to vaccination, exclusion of other causes, and biopsy findings. Healthcare providers should consider the overall clinical picture when biopsy results are interpreted in the context of suspected vaccine-induced kidney injury. Corticosteroids remain the first-line treatment for many vaccine-induced kidney conditions, including minimal change disease, acute interstitial nephritis, and some cases of glomerulonephritis. Reports show that 80% of patients with glomerulonephritis following COVID-19 vaccination respond well to treatment, indicating a favorable outcome [36].

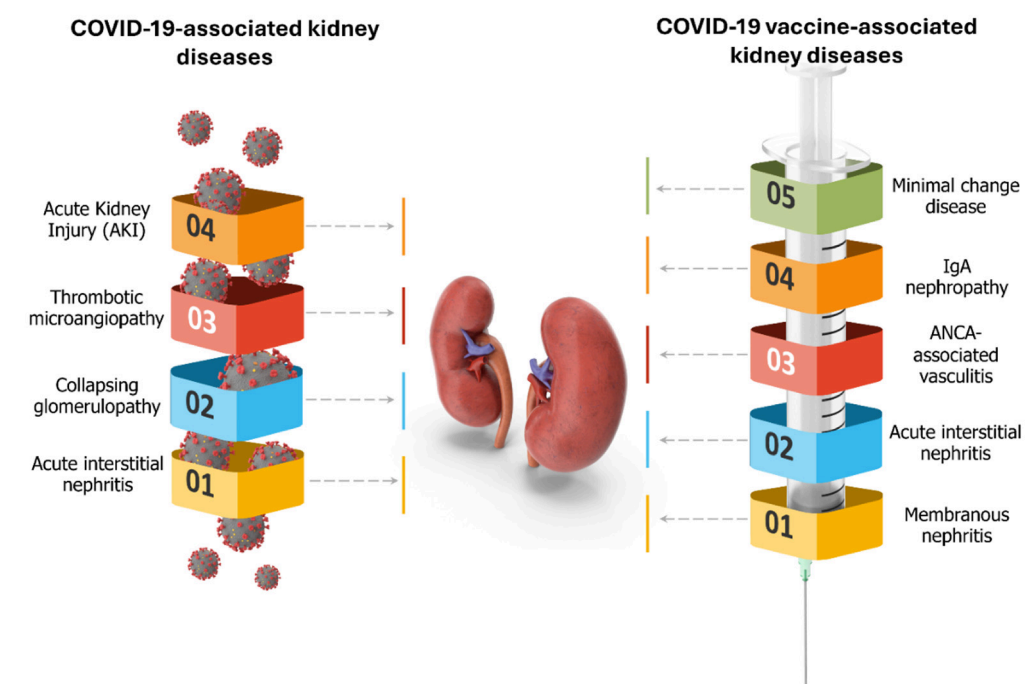


Figure 2. Partial list of COVID-19 and COVID-19 vaccine-associated renal lesions.

Some patients with vaccine-associated severe AKI might need supportive care, such as fluid management, electrolyte correction, blood pressure control or temporary dialysis. Importantly, treatment should be individualized on the basis of the specific diagnosis and severity of kidney injury. In most reported cases, prompt recognition and appropriate treatment led to good outcomes, with many patients showing significant improvement or complete resolution of kidney function. While a link between vaccines and kidney injury may raise suspicion, identifying a direct cause is often difficult without identifying specific biomarkers. Clinicians should maintain a high level of suspicion in the appropriate clinical context and be aware of potential vaccine-related kidney injury in relevant cases [37].

Additionally, diagnosing subclinical renal disease is a clinical challenge, as many cases may be undetected until significant deterioration of renal function occurs. Early signs of renal injury, including slight fatigue and changes in urination, can easily be mistaken for other, more common illnesses, such as dehydration or stress. To detect the early stage of kidney disease before the appearance of signs and symptoms, Japan has adopted low-cost urinalysis as part of mass screening in schools to detect kidney disease, diabetes, and urinary tract infections in the subclinical stage by analyzing the presence of protein, blood, sugar, and bacteria in urine samples [38]. Periodically using such low-cost screening with urine samples after vaccination might be useful for detecting subclinical renal disease.

Infection-related kidney damage mechanisms include direct viral invasion of renal cells, cytokine-induced inflammation, complement dysregulation, hypercoagulation, and microangiopathy. Of relevance, the evidence linking complement activation to infectious disease outcomes remains inconsistent and is derived primarily from observational studies, with only limited evidence from studies investigating nutritional cofactors [39]. In contrast, vaccine-associated renal effects are generally milder and appear to occur less frequently. Notably, the absolute risk of AKI is substantially lower following COVID-19 vaccination than after SARS-CoV-2 infection. After adjustment for potential confounders, SARS-CoV-2 infection has been associated with an approximately 10-fold higher risk of AKI compared with COVID-19 vaccination [40]. Renal manifestations of SARS-CoV-2 infection appear more distinctive and mechanistically heterogeneous than those seen in many other infectious diseases. In COVID-19, kidney involvement commonly includes proteinuria, hematuria, and AKI, with pathology characterized by endothelial injury, microvascular thrombosis, complement activation, and podocyte injury, including collapsing glomerulopathy in susceptible patients. In contrast, in infections such as streptococcal disease, hepatitis B or C, HIV, and malaria, renal injury is more commonly driven by immune complex deposition, direct pathogen-mediated damage, sepsis, or hemodynamic compromise, with acute tubular injury often representing the predominant lesion [41]. Overall, although AKI is a common endpoint across infectious diseases, SARS-CoV-2 is associated with a broader and more complex renal phenotype involving vascular, inflammatory, and glomerular injury [42].

5. Discussion

SARS-CoV-2 infection can cause renal dysfunction by inducing microthrombi formation and renal microvasculature damage. Additionally, infection triggers an increase in inflammatory markers such as NGAL and KIM-1, indicating AKI. COVID-19 is associated with an accelerated decline in kidney function, particularly in hospitalized patients. Moreover, the recurrence of renal diseases after subsequent doses of the vaccine [36,43] support the importance of the vaccine combination. Conclusively determining whether SARS-CoV-2 infection or COVID-19 vaccination directly causes *de novo* kidney injury may not always be possible [44]. However, when urinary abnormalities or renal structural changes appear shortly after infection or vaccination, it is plausible that the immunoinflammatory responses triggered by either event could lead to kidney disorders. Elderly patients and those with preexisting kidney conditions may be at increased risk of SARS-CoV-2 infection or vaccine-induced renal injury. COVID-19 patients who develop AKI have longer hospital stays and higher mortality rates [45]. Current evidence suggests the need for short-term and long-term vaccine safety monitoring. Repeated vaccinations might increase side effects or decrease effectiveness because of “vaccine exhaustion” [46]. Recent reports suggest that SARS-CoV-2 infection or COVID-19 vaccines may have negative effects on various systems, including renal health (Figure 2) [47,48]. While serious short-term renal complications after COVID-19 vaccines are limited, further study is needed to de-

termine whether COVID-19 vaccination can worsen the condition of severely ill patients with preexisting kidney diseases (those who have also been infected with SARS-CoV-2). Addressing whether vaccination may worsen renal outcomes in patients with preexisting kidney disease requires well-designed longitudinal registries. However, their establishment is challenging because it depends on standardized case definitions, harmonized data collection, long-term follow-up, access to vaccination records, and careful distinction between temporal association and causality. The principal barrier is sustainability, as registry development, maintenance, data quality assurance, and periodic analysis require stable financial and institutional support, typically from academic networks, hospital systems, public health agencies, national kidney foundations, and competitive research funding.

Again, AKI can occur as a complication of both SARS-CoV-2 infection and COVID-19 vaccination [3,49]. Finally, determining the short-term and long-term effects of repeated COVID-19 vaccinations on various systems, including kidney health, is important [50]. This is particularly crucial for vulnerable groups with preexisting kidney diseases. Before additional doses of vaccines are recommended to these groups, healthcare providers need to consider potential long-term complications that may not be immediately apparent [13,19,36,51–53].

6. Conclusions

In conclusion, a heterogeneous spectrum of renal lesions has been reported in temporal association with SARS-CoV-2 vaccination, including minimal change disease, IgA nephropathy, ANCA-associated vasculitis, membranous nephropathy, anti-GBM disease, and FSGS. While vaccination provides protection against severe COVID-19 and its renal complications in certain groups of patients, emerging reports of de novo and relapsing glomerular and tubulointerstitial diseases underscore the need for vigilant clinicopathologic correlation, particularly in high-risk populations with preexisting kidney disease, transplant recipients, and immunosuppressed individuals. Systematic assessment of kidney function, urinary protein excretion, and relevant serologic markers after each vaccine dose, coupled with continued pharmacovigilance, is essential to better define the short- and long-term renal safety of COVID-19 vaccines and to guide evidence-based vaccination strategies in vulnerable populations.

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