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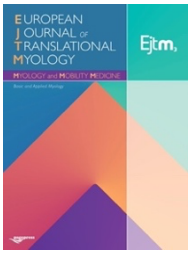

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Acute and chronic kidney injury following COVID-19 infection and vaccination: a narrative review

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Abstract

This narrative review examines acute and chronic kidney injury following COVID-19 infection and vaccination, discussing the mechanism of SARS-CoV-2 entry into host cells through the ACE2 receptor - highly expressed in renal tissues - facilitating the viral invasion. Viral RNA has been detected in the urine of patients infected with SARS-CoV-2, suggesting direct renal involvement. The incidence of acute kidney injuriy (AKI) among hospitalized COVID-19 patients was particularly higher in the early stages of the pandemic and largely varied by 29%-46%, depending on population studied and COVID-19 wave. Pathological findings include acute tubular injury (ATI), collapsing glomerulopathy, and focal segmental glomerulosclerosis. Major risk factors for AKI comprise older age, male sex, diabetes, hypertension, cardiovascular disease, and preexisting Chronic Kidney Disease (CKD). AKI significantly increases mortality of COVID-19 patients, particularly in advanced stages of renal failure. CKD is also associated with

severe COVID-19 outcomes, including increased hospitalization, intensive care admission, and mortality. Patients with CKD show a dose-dependent relationship between disease stage and adverse patient outcomes. This review further addresses renal complications following COVID-19 vaccination, which, although rare, encompass various immune-mediated glomerular diseases. Minimal Change Disease (MCD) is most frequently reported after COVID-19 vaccination, followed by IgA nephropathy, membranous nephropathy, anti-glomerular basement membrane (anti-GBM) nephritis, and ANCA-associated vasculitis. These conditions commonly present with hematuria, proteinuria, or nephrotic syndrome, and many respond to corticosteroid or immunosuppressive therapy. Other less frequent renal complications include thrombotic microangiopathy, acute tubulointerstitial nephritis, and IgG4-related nephritis. In conclusion, both COVID-19 infection and vaccination can be associated with a spectrum of renal manifestations ranging from AKI to CKD and immune-mediated glomerulopathies. Awareness, early detection, and multidisciplinary management are essential to reduce renal morbidity and improve patient outcomes.

Key words: COVID-19, acute kidney injury, chronic kidney disease, minimal change disease, vaccination.

COVID-19 was initially identified in December 2019 in Wuhan, a city within Hubei Province, China. The World Health Organization (WHO) declared COVID-19 a global pandemic on March 11, 2020. Cellular and molecular investigations have illuminated the complex mechanisms underlying the onset of SARS-CoV-2 infection. The virus's entry into host cells is mediated by the interaction between the spike glycoprotein and the Angiotensin-Converting Enzyme 2 (ACE2) receptor, which is abundantly expressed in alveolar type II pneumocytes, cardiomyocytes, and renal epithelial tissues. Once the spike protein binds to ACE2, the transmembrane serine protease 2 (TMPRSS2) facilitates the endocytosis of SARS-CoV-2, thereby initiating its replication within the host cell.[1]

By early 2023, the cumulative global burden of COVID-19 had escalated to approximately 700 million confirmed cases, with over 7 million fatalities reported worldwide.[2] The pathogen's pathological influence extends beyond the respiratory tract, affecting multiple vital organs—including the cardiac, renal, and gastrointestinal systems. Extensive clinical and post-mortem studies have revealed the pervasive distribution of SARS-CoV-2 within various organ systems, emphasizing its multisystemic pathogenic potential.

Notably, the renal system has emerged as a prominent target for SARS-CoV-2 invasion, even in patients without preexisting renal impairment. This susceptibility is attributed to the high expression levels of ACE2, TMPRSS2, and cathepsin L in renal cells, which collectively act as molecular gateways that facilitate viral entry inside them.[3] Polymerase Chain Reaction (PCR) assays have detected viral RNA fragments of SARS-CoV-2 in the urine samples of 21–50% infected patients, typically during the second or third week of illness, suggesting a potential renal tropism of this virus.[4]

The development of vaccines represents a critical milestone in the global effort to combat the rapid spread of this highly contagious pathogen. These sophisticatedly engineered and rigorously evaluated immunogenic formulations are designed to elicit both humoral and cellular immune responses. By targeting essential viral structures—such as the spike glycoprotein and nucleocapsid protein—these vaccines train the immune system to identify and neutralize the virus upon future exposure.[5]

Globally, various strains of COVID-19 immunization modalities have been deployed. The Pfizer-BioNTech (BNT162b2) and Moderna (mRNA-1273) vaccines are lipid nanoparticle–encapsulated, nucleoside-modified mRNA constructs that encode the SARS-CoV-2 spike protein, a key mediator of host cell attachment and entry.[6] In contrast, Vaxzevria (AstraZeneca vaccine) employed a replication-deficient chimpanzee adenoviral DNA-vector encoding the spike protein. mRNA-based vaccines present a minimal risk of infection or insertional mutagenesis, while inducing robust antiviral neutralizing antibody production and strong T lymphocyte activation, including CD4+ helper and CD8+ cytotoxic responses.[7] Their efficacy is demonstrated by vigorous antibody generation, T-cell activation, and cytokine release, particularly interferon-γ, which inhibits SARS-Co-V-2 replication. [8]

Nevertheless, with the widespread implementation of COVID-19 vaccination programs, adverse events have been increasingly reported.[9] These include immunologically mediated complications, such as autoimmune reactions or exacerbations of preexisting autoimmune disorders.[10,11] Hence, stringent pharmacovigilance is crucial to determine the true prevalence and clinical significance of these post-vaccination sequelae.[12,13]

This narrative review aims to synthesize and analyze current evidence concerning renal complications—particularly acute kidney injury (AKI) and chronic kidney injury (CKD) — associated with both COVID-19 infection and vaccination.

Acute kidney injury and COVID-19 infection

Definition of acute kidney injury

According to the Kidney Disease: Improving Global Outcomes (KDIGO) 2012 and 2021 Clinical Practice Guidelines, AKI is defined as an increase in serum creatinine by ≥0.3 mg/dL within 48 hours, or an increase to ≥1.5 times the baseline value within 7 days, or urine output less than 0.5 mL/kg/h for at least six hours.[14]

AKI and acute kidney disease (AKD) were differentiated from chronic glomerular disorders. Consequently, glomerular pathologies—including Minimal Change Disease (MCD), IgA Nephropathy (IgAN), Membranous Nephropathy (MN), anti-Glomerular Basement Membrane (anti-GBM) nephritis, and anti-Neutrophil Cytoplasmic Autoantibody (ANCA) vasculitis—are categorized as glomerular diseases rather than AKI subtypes.

These standardized definitions form the conceptual framework for classifying renal involvement following COVID-19 infection or vaccination, ensuring consistency with international nephrology guidelines throughout this review.

Incidence of acute kidney injury

The incidence of AKI among hospitalized COVID-19 patients was particularly higher in the early stages of the pandemic and largely varied by 29%-46%, depending on population studied and COVID-19 wave.[15]

According to a metanalysis of 79 studies on 49,692 total COVID-19 patients the incidence of AKI was 10.6%, higher than in patients hospitalized without COVID-19 (8%).[16] A further random effect metanalysis of 39 studies published between December 1, 2019 and June 30, 2020, including 25,566 patients, reported an AKI incidence of around 15% among hospitalized COVID-19 patients, increasing to approximately 50% with severe disease.[17] A similar pooled incidence of 12.6% was reported by another metanalysis of 45 studies published in 2019-2020, including 14,358 patients (average age 51 years)[18] and 12.5% in a metanalysis of 58 studies including 13,452 patients hospitalized during 3 coronavirus pandemic waves (SARS-CoV-1; MERS-CoV and SARS-CoV-2).[19]

Another metanalysis including 142 studies and 49,048 hospitalized COVID-19 patients reported a higher pooled incidence of AKI (28.6%; 95%CI: 19.8–39.5) among hospitalized COVID-19 patients from USA and Europe (20 studies) versus 5.5% (95%CI 4.1–7.4) among patients from China (62 studies).[20] Within ICU patients of the latter metanalysis, the pooled incidence of AKI was 29.2% (95%CI; 20.1–40.3, I^2 = 97%) among 23 studies involving 4,330 ICU patients and 1,701 AKI events.[20] Another metanalysis on 28 studies published through June 20, 2020 for 18,043 total patients hospitalized for COVID-19 the overall incidence of AKI was 9.2% (95%CI: 4.6; 13.9) increasing to 32.6% (95%CI: 8.5-56.6) among those admitted to ICU.[21] In another study summarizing 42 systematic reviews (38 with metanalysis) – whose number of primary studies ranged from 4 to 142 and COVID-19 cases from 420-54,173 - the risk of AKI among COVID-19 patients varied from 4.3% to 36.4%.[22]

A more granular examination of specific patient subgroups revealed distinct variations. For instance, the incidence of AKI ranged from 36% to 50% among COVID-19 patients on kidney replacement therapy (KRT) and from 30% to 69% among renal transplant recipients.[22] This high prevalence of AKI underscores the critical need for systematic renal monitoring in severe COVID-19 cases. Immunocompromised individuals demand heightened clinical vigilance, given the delicate interplay between immunosuppressive therapy, graft functionality and COVID-19-related renal insults. [23-28]

Clinical features

Early clinical observations of COVID-19-associated AKI highlighted the frequent occurrence of hematuria and proteinuria.[29,30] For instance, in an investigation on 3,993 COVID-19 patients admitted to Mount Sinai Health System from February 27 to May 30, 2020, 1,835 developing AKI, 84% had proteinuria (defined by a protein-to-creatinine ratio above 0.5, a dipstick reading of ≥1+, or >30 mg/dL on urinalysis) and 81% hematuria (≥1+ at dipstick or urinalysis).[31] In an early study on 701 hospitalized COVID-19 patients from China, 43.9% of patients exhibited proteinuria and 26.7% hematuria on admission.[32] In a multicenter study on 5,980 patients admitted to 20 hospitals from Texas (USA) from March 1, 2020 through January 1, 2021, presence and degree of proteinuria and hematuria at dipstick analysis in combination with pre-existing chronic kidney disease (CKD) or baseline creatinine level accurately predicted both AKI and KRT.[33]

Among 333 COVID-19 patients with dipstick analysis on admission to Tonji hospital (Wuhan, Hubei Province, China) from January, 29 through February 9, 2020, 41.7% had proteinuria and 65.8% proteinuria. The proportion of patients with hematuria increased from 33.3% in those with moderate COVID-19, 39.1% in those with severe disease and 69.6% for critically ill patients. Likewise, the proportion of patients with proteinuria increased from 43.8% in patients with moderate COVID-19 to 81.2% in those with severe disease and 85.7% in those critically ill.[29] In the latter study, among the sub-group of 198 patients with renal involvement followed up for a median time of 12 days, 55.1% exhibited hematuria and 89.4% proteinuria, despite mean basic serum creatinine level of 74 μmol/L and a prevalence of AKI of 17.7% according to KDIGO criteria.[29]

Likewise, among 178 COVID-19 patients hospitalized at Wuhan Union Hospital (China) from 2 February to 29 February 2020, despite urine abnormalities (hematuria, proteinuria, leukocyturia) being more frequent among patients with AKI, a large proportion of patients without AKI exhibited urine abnormalities with normal creatinine level.[34]

Consensus statement supported the use of new biomarkers other than serum creatinine or urine output to classify and manage AKI.[35] In COVID-19-related AKI, low-molecular-weight proteins tend to predominate in urine rather than albumin, indicating a tubular rather than glomerular origin of proteinuria. This distinct pattern may serve as an early biomarker of AKI.[36] Proximal tubule injury could be employed as predictor of AKI.[30]

To advance understanding, future studies should integrate biomarker profiling of glomerular and tubular function with pathological analysis from renal biopsies, thereby elucidating the underlying mechanisms of COVID-19-induced AKI.

In a study on 47 COVID-19 patients from Paris metropolitan area developing AKI or proteinuria and undergoing kidney biopsy between March 8 and May 19, 2020 acute tubular injury (ATI) was the predominant lesion (42.6%) followed by glomerulopathy injury (36.2%), combining collapsing glomerulopathy and focal segmental glomerulosclerosis.[37] Likewise, a variable degree of ATI was confirmed at post-mortem examination in all 26 COVID-19 patients (mean age 69 years; 19 males vs 7 females) died for respiratory failure between February 18 to March 27, 2020 at Union Hospital in Wuhan (Hubei Province, China)[38] and among 12 patients with AKI (stage 2 o 3) examined from April 24, 2020 to May 8, 2020 at Lenox Hill Hospital in New York City (USA).[39]

Risk factors for acute kidney injury

A complex interdependence of demographic and clinical factors influences AKI occurrence.[40,41]

A consistent association between age and AKI risk has been documented by various systematic reviews.[18,40,41] In particular, in a metanalysis of 79 studies published between January 1, 2020 and May 15 2020 including 49,692 patients, the pooled risk of AKI increased in patients aged >60 years (aOR=3.53; 95%CI: 2.92–4.25) and in patients with severe COVID-19 (aOR=6.02; 95%CI: 2.53; 14.58).[41]

According to another systematic review of 38 studies including 42,779 patients, male sex was also a significant determinant of AKI, with a pooled OR of 1.37 (95%: 1.25- 1.49; $p < 0.00001$) of developing AKI.[40] Furthermore significant risk factors of AKI included smoking (pooled OR=1.23; 1.07- 1.42; p=0.004), hypertension (pooled OR=1.85; 95%CI: 1.70–2.02; $p < 0.00001$), diabetes mellitus (pooled OR=1.71; 95%CI:1.59–1.84; $p < 0.00001$), obesity (pooled OR=1.12; 95%CI: 1.01–1.25; p = 0.03), CKD (pooled OR= 4.56; 95% CI 3.63–5.73; $p < 0.0001$), pneumopathy (OR=1.36; 1.16-1.80; p=0.001), cardiovascular comorbidity (pooled OR= 1.98; 956%CI: 1.74-2.34; $p < 0.00001$), cancer (pooled OR=1.26; 95%CI:1.13-1.40; $p<0.00001$),

mechanical ventilation (pooled OR=8.61; 5.83-13.17; p<0.00001) and use of vasopressors vasopressors pooled (pooled OR=8.33; 95%CI: 4.31-14.72; p<0.00001).[40]
Likewise, according to another metanalysis on 142 studies published between December 1, 2019 and May 29, 2020, including 49,048 hospitalized COVID-19 patients and 5,152 AKI events, the independent risk of AKI expressed as OR was 2.15 (95%CI: 1.54–3.00) per mean/median 10-year increase in age, 1.36 (95%CI: 1.07–1.73) per 10% increase in male sex prevalence, 1.53 (95%CI: 1.13–2.08) per 10% increase in prevalence of cardiovascular disease, 1.48 (95%CI: 1.24–1.77) per 10% increase in diabetes prevalence, 1.64 (95%CI: 1.40–1.93) per 10% increase in CKD prevalence and 1.50 (95%CI: 1.33–1.69) per 10% increase in prevalence of hypertension.[20]

A random-effect metanalysis on 14 studies published between January 1, 2020 to December 24, 2020, for 17,876 total patients reported that renin–angiotensin–aldosterone system (RAAS) blockades by either angiotensin-converting-enzyme inhibitor (ACEI) and angiotensin receptor blocker (ARB) was associated with a significantly increased risk of AKI (OR=1.68; 95%CI: 1.19–2.36).[42]

Collectively, the above findings underscore the multifactorial etiology of AKI in COVID-19, where risk factors as age, sex and comorbidities as cardiovascular and metabolic disorders, preexisting CKD, and hypertensive disease intersect synergistically. Hence, clinicians must employ risk-stratified management approaches to mitigate renal complications in vulnerable COVID-19 patients.

Acute kidney injury and mortality

Several studies reported an increased mortality risk associated AKI in COVID-19 patients.[19,23,29,43-60]

Moreover, in a prospective study on 701 hospitalized COVID-19 patients from China a growing graded relationship between AKI stage and mortality risk was observed, progressively increasing from stage 1 (HR=1.90; 95%CI: 0.76-4.76) to stage 2 (HR=3.51; 95%CI: 1.49-8.26) and stage 3 (HR=4.38; 95%CI: 2.31-8.31).[32]

However, among 339 COVID-19 patients older than 60, hospitalized at Renmin Hospital in Wuhan (Hubei Province, China) from January 1 to February 6, 2020, AKI significantly increased the risk of death only at univariable analysis (HR=5.84; 95%CI: 3.30-10.43) but not in the adjusted model (HR=1.2; 95% CI 0.6–2.4), where only age, cardiovascular disease, COPD and cerebrovascular disease were significant risk mortality factors.[61]

A comprehensive meta-analysis summarizing all above studies provided a pooled risk ratio of 4.6 (95%CI: 3.3–6.5, I^2=90%) for AKI-associated deaths, reflecting the substantial prognostic weight of AKI in the context of COVID-19. [20] Likewise, in a subsequent single center study on 997 COVID-19 patients from Tehran, not included in the above review, the adjusted risk of death significantly increased with AKI (aHR=1.91; 1.39-2.63).[62]

Treatment and recovery

A multicenter cohort study on 3,099 critically ill COVID-19 patients admitted to ICU in 67 hospitals across the USA between March 4 and April 11, 2020 reported that 20.6% patients required KRT within 14 days of ICU admission. [63] However, the optimal modality of KRT remains uncertain, as it is often influenced by practice pattern and institutional resource availability.

Continuous KRT may be preferable in hemodynamically unstable patients, especially those requiring prone positioning, whereas peritoneal dialysis may be employed in case of dialysis circuit clotting, coagulation contraindications or limited extracorporeal resources.[64]

Fluid management in COVID-19-associated AKI represents a therapeutic challenge, requiring precise volume optimization to restore perfusion while avoiding fluid overload, which may worsen oxygenation in patients with Acute Respiratory Distress Syndrome (ARDS).[65,66]

Although most cases of COVID-19-related AKI ultimately recover renal function, a substantial subset (35–40%) experience persistent AKI lasting beyond 7 days. Furthermore, approximately 30% of patients undergoing KRT during hospitalization remain dialysis-dependent at the time of discharge.[67,68]

The extent and duration of AKI episodes significantly influence the risk of developing new-onset or progressive CKD and increases overall mortality. Consequently, rigorous post-AKI follow-up is essential to detect long-term renal sequelae and mitigate the progression to CKD.

Notably, data from the pandemic initial phase reported a decline in both AKI severity and KRT utilization rates, even before the roll out of COVID-19 vaccines commenced. This positive trend, which continued through Omicron wave, may reflect advancements in clinical management, improved understanding of disease mechanisms and earlier intervention strategies, in addition to more contagious yet less virulent SAR-CoV-2 strains circulating. [69-72]

The continuously evolving interplay between emerging SARS-CoV-2 variants, host immune responses, and innovative therapeutic interventions shaped incidence, severity and recovery trajectories of AKI in patients afflicted by COVID-19. [17,54,58,59,61,62]

Chronic kidney disease (CKD) and COVID-19 infection

Prevalence

CKD refers to abnormalities in kidney structure or function that persist for a minimum of three months and are associated with health implications, including reduced Glomerular Filtration Rate (GFR), albuminuria or structural abnormalities identifiable on imaging.[73]

Prevalence of pre-existing CKD among individuals infected with SARS-CoV-2 displayed substantial variability according to various systematic reviews with metanalysis, ranging from 0.4% to 49.0% depending on study design, geographical area and clinical setting according to a review of 28 systematic reviews (Supplementary materials, Table 1).[74-82]

For instance, a metanalysis of 22 studies published by June 2020, including 17,391 COVID-19 patients reported a pooled prevalence of pre-existing CKD of 5.2% (95%CI: 2.8-8.1) and 2.3% (95%CI: 1.8-2.8) end stage kidney disease (ESKD).[81] Another metanalysis of 45 studies on 16,561 hospitalized COVID-19 patients from 17 countries and 4 continents reported a pooled prevalence of 10.0% (95%CI: 6.2-14.6) of pre-existing CKD.[83] A further metanalysis of 58 studies on kidney-related outcomes published by 16 June 2020, including 13,452 total patients hospitalized during 3 recent pandemics (SARS-CoV-1, MERS-CoV and SARS-CoV-2) reported

a ESKD prevalence of 30.9% (95%CI: 4.6%–66.8%) associated with COVID-19 and an incidence of coronavirus infection of 7.7% (95%CI: 4.9%–11.1%) in hemodialysis patients.[19]

When considering individual studies not included in the above systematic reviews, the evidence remains less conclusive regarding infection susceptibility among CKD versus non-CKD populations. A nationwide retrospective case-control study on 219,961 patients 18+ years old from South Korea, 7,341 testing positive for COVID-19 by May 15, 2020 reported a lower adjusted odds ratio (OR = 0.50) of COVID-19 infection by CKD.[84]

Another investigation conducted at a major urban dialysis center in the United Kingdom, including 1,530 dialysis patients reported a 19.6% incidence of COVID-19 from March 9 to April 19, 2020. Importantly, 96% of infections occurred in patients receiving in-center dialysis, highlighting the elevated transmission risk in facility-based treatment environments.[85]

Chronic kidney disease and hospitalization risk

According to a metanalysis of 74 qualitative and 75 quantitative studies, with study population ranging from 19 to 44,672 COVID-19 patients, a number of pre-existing comorbidities including CKD increased the risk of hospitalization, though study heterogeneity was moderate to high (I^2=50-90%).[86]

Additional individual studies outside the above reviews corroborated this evidence, consistently showing significant higher risk of hospital admission among COVID-19 patients with pre-existing CKD, with variable ORs.[87-94]

For instance, one study on 5,416 COVID-19 patients testing positive from March 1–June 23, 2020 in the US reported a significantly higher adjusted RR of 4.0 (95%CI: 3.0-5.2) for hospitalization in patients with CKD[89] and another study on 10,371 US veterans testing positive between February 28 and May 14, 2020, the aHR for hospitalization for CKD was 1.21 (95%CI:1.11-1.32).[90] In the Province of Reggio Emilia (Northern Italy) the HR of hospitalization of CKD adjusted for age and sex was 1.9 (95%CI: 1.3-2.9) among 2,653 patients testing positive between February 27 and April 2, 2020.[92]

In Brazil, among 10,713 patients positive to SARS-CoV-2 in the State of Espirito Santo through June 11, 2020, the adjusted OR of hospitalization in relation to CKD was 2.41 (95%CI: 1.59–3.66, < 0.001).[93] Likewise, among 38,324 SARS-CoV-2 positive patients testing positive in Mexico between 01 January to 13 May 2020 the adjusted OR of hospitalization for CKD was 2.58 (95%CI: 2.08–3.20).[94]

These above results underscore the vulnerability of patients with advanced CKD and the need for intensified monitoring and preventive care.

Chronic kidney disease (CKD) and mortality

A number of systematic reviews and large studies have examined mortality risk in COVID-19 patients with pre-existing CKD.[82,95,96, 98-103] Among these, Dorjee *et al.*[82] provided the most current and methodologically robust meta-analysis, identifying a RR of 2.52 (95% CI: 2.11–3.00) for COVID-19 related mortality among CKD patients—a finding featured by moderate certainty (I^2= 72%).[82]

Another meta-analysis of 19 studies published until April 23, 2020, including 7,216 COVID-19 patients reported and independently higher mortality risk associated with CKD (RR= 3.47; 95%CI: 1.36-8.86; p < 0.009; I2=14%, p=0.32).[95]

A population based cohort study conducted in England from 2017 through 2020 reported a progressively higher mortality with increasing CKD stages among COVID-19 patients with type 1 diabetes, with HRs of 2.07 (95%CI: 1.48- 2.89) , 2.46 (95%CI: 1.72- 3.52), 3.71 (95%CI: 2.47-5.58), and 8.35 (95%CI: 5.50- 12.72) for CKD stages 3A–5, respectively.[96] A similar dose-dependent relationship was documented in type 2 diabetes, with adjusted HRs of 1.39 (95%CI: 1.30- 1,59), 1.76 (95%CI: 11.63- 1.89), 2.31 (95%CI: 2.10- 2.54), and 4.91 (95%CI: 4.84; 5.56) for the corresponding CKD stages.[96]

Likewise, an OpenSAFELY study conducted in England on 17,278,392 patients [97] identified an adjusted HR of 3.69 (95%CI: 3.09–4.39) of mortality in ESKD patients compared to non-ESKD counterparts.[97] Additional studied conducted in the early months of the pandemic reported mortality odds ratio of 2.02 (95%CI: 1.27- 3.20) among 9,807 COVID-19 patients aged >60

from Brazil during 2020,[98] 1.86 (95% CI: 1.30–2.64) among 1,305 COVID-19 patients hospitalized in South Michigan (USA) from March 1 2020 through April 17, 2020 [99] and 3.07 (95%CI: 1.43–6.61) among 9,148 COVID-19 patients diagnosed by PCR in South Korea up to March 26, 2020.[100] In another study on 10,482 COVID-19 patients diagnosed in New York between March 1, 2020, to April 27, 2020, significantly higher mortality was found among 419 COVID-19 patients with ESKD (OR=1.37; 95%CI: 1.09- 1.73).[101]

In the European kidney replacement therapy population on 4,298 COVID-19 patients diagnosed between February 1, 2020 through April 30, 2020 from 7 European countries, dialysis patients experienced a 20% attributable 28 day mortality rate and a 21.1 (95%CI: 18.6- 23.9) higher death risk compared to propensity-score matched controls.[102] Likewise, in a study on 4,264 COVID-19 patients admitted to 68 different ICU in the USA, dialysis patients exhibited ad higher risk of 28-day in-hospital death (aHR= 1.41; 95%CI: 1.09-1.81), while patients with non-dialysis-dependent CKD had an intermediate risk (aHR=1.25; 95%CI: 1.08-1.44) compared to patients without pre-existing CKD.[103]

Chronic kidney disease and COVID-19 severity

Several systematic reviews have evaluated the impact of CKD on COVID-19 severity.[82, 104-107] Among them, a comprehensive meta-analysis of 77 studies on 38,906 patients reported a RR of 1.56 (95%CI: 1.31- 1.86) of developing severe COVID-19 among CKD patients.[92] Further metanalyses reported much higher pooled ORs of severe COVID-19 associated with CKD, going from 2.20 (95%CI: 1.27-3.80) in 124 studies published by July 2020 CKD[103] to 3.03 (95%CI: 1.09–8.47) in another contrite metanalysis of four studies published by March 9, 2020[106] and 1,389 SARS-CoV-2 patients (19.7% with severe COVID-19)[105] and up to 5.60 (95%CI: 4.13-7.60) in 13 studies on 99,817 patients.[106]

In addition, three relatively large individual studies not covered in those reviews also reported significantly higher OR of severe COVID-19 ranging from 2.1 to 4.28 [84,87,108,109] In particular, the adjusted OR of severe COVID-19 increased from 2.052–2.178 among 7,341 COVID-19 patients (13.0% severe) with CKD or ESKD testing positive through May, 15 in South Korea,[84] to 3.6 (95%CI: 2.2–5.8) among 4,322 COVID-19 patients (31.5% hospitalized) from Atlanta

metropolitan area positive between March 2 and May 31, 2020,[87] 3.40 (95%CI: 1.67–6.92) among 7,339 COVID-19 patients (mean age 47.1 years),12.6% hospitalized, from South Korea testing positive by 15 May 2020,[108] and 4.28 (95%CI: 1.31–13.92) in 2,0387 patients (34% with severe disease) testing positive from January 27 2020 and March 21, 2020 at Tonjii Hospital in Wuhan (China).[109]

However, definitions of "severe COVID-19" varied across studies, introducing heterogeneity in interpreting CKD-related risk patterns.

Chronic kidney disease and intensive care unit admission

The above mentioned meta-analysis of 34 studies published between 6 May 2020 and 18 May 2021 reported no association between CKD and ICU admission (OR=1.44, 95%CI 0.94–1.94; I^2= 53.1%).[106]

Three studies[87,110.111] not included in the above review reported significantly greater ICU admission risks with CKD, with ORs increasing from 1.7 (95%CI: 1.2-2.3) among 4,322 COVID-19 patients diagnosed in Atlanta Metropolitan Area (Georgia, USA) between March 2 and May 31, 2020[87] to 2.99 (95%CI: 1.89-4.64) on 4,261 patients hospitalized for suspected COVID-19 in Moscow between 8 April and 28 May 2020 (2.99; 1.89-4.64)[110] and 3.6 (95% CI 1.57-8.08) among 5,000 consecutive patients diagnosed with COVID-19 in Qatar between February 28 and April 17, 2020.[111] However, other studies did not confirm the latter evidence (OR=2.68; 95%CI: 0.65; 11.07)[112] and according to an overview of 69 systematic reviews and 66 primary studies with high AMSTAR index did not confirm the association between CKD and ICU admission in COVID-19 patients.[74]

This variability underscores the complexity of assessing critical care needs in CKD patients, influenced by comorbidities, treatment access, and disease stage.

Chronic kidney disease and mechanical ventilation

The risk of mechanical ventilation increased with CKD (RR= 34.39; 95%CI: 4.63-255.51; $p < 0.0005$) according to the above meta-analysis of 19 studies on 7,216 patients.[95] Two

additional large studies not included in the latter review reported OR of mechanical ventilation associated with CKD of 1.22 (95%CI: 1.05-1.43) among 11,721 patients admitted to 245 hospitals across 38 US states between 15 February and 20 April 2020[112] and 1.44 (95%CI: 1.07-1.94) in 38,324 SARS-CoV-2 positive patients diagnosed in Mexico from January 1 to May 12, 2020.[94] Although 27% needed mechanical ventilation, over half of 15 kidney transplant recipients admitted to Columbia University Medical Center through March 17, 2020 were discharged home by the end of follow-up.[23] Furthermore, in a longitudinal cohort study including 10,131 patients testing positive for SARS-CoV-2 at PCR between February 28 and May 14, 2020, and followed up through June 22, 2020 in the Department of Veterans Affairs (VA) national health care system, CKD increased the risk of hospitalization (aHR=1.21; 95%CI: 1.11-1.32) and death (aHR=1.25; 95%CI: 1.08-1.45) yet not mechanical ventilation (aHR=1.16; 95%CI: 0.96- 1.41).[90] Likewise, among 10,482 COVID-19 patients admitted to 13 Ney York hospitals from March 1, 2020, to April 27, 2020, and followed through May 27, 2020, those 419 with ESKD were more likely to die (aOR=1.37, 95%CI: 1.09 - 1.73; p=0.003) but not to receive mechanical ventilation (aOR=0.84; 95%CI: 0.84-1.38; p=0.56).[101]

Although the available evidence does not definitively link CKD with an increased requirement for ventilatory support, individualized patient assessment remains essential due to clinical variability in renal and respiratory function.

Chronic kidney disease and multiple outcomes

Four systematic reviews have collectively investigated multiple adverse outcomes associated with pre-existing CKD in COVID-19 patients. A metanalysis of 16 studies published by April 2020 including 3,994 patients reported a pooled OR of 5.32 (95%CI: 1.86 - 15.19; p= 0.002) for a composite outcome of serious adverse events encompassing ICU admission, acute respiratory distress syndrome (ARDS), mechanical ventilation, pneumonia, or mortality among CKD patients versus controls.[113]

Likewise, another metanalysis of 19 studies for 7,216 total COVID-19 patients reported a pooled RR of 2.63 (95%CI: 1.33-5.17; p=0.03; I^2=51%) for a composite poor outcome including

mortality, severe COVID-19, ARDS, ICU admission, and mechanical ventilation in patients with CKD.[95]

A further metanalysis of 13 studies published Until July 15, 2020 including 12,999 COVID-19 patients reported a pooled RR of 1.64 (95%CI: 1.28- 2.09) for a composite outcome defined as severe illness, critical illness or death associated with CKD.[114]

Emerging primary data reinforce these findings: among 38,324 testing positive to SARS-CoV-2 between January 1 and May 12, 2020 in Mexico, those with CKD exhibited an increased risk of hospital admission (OR=4.65; 95%CI3.61–5.98), pneumonia (OR=2.14; 95%CI: 1.69–2.72) and death (OR=2.23; 95%CI: 1.50–3.31).[89] Additionally, ESKD increased the odds ratio of length of stay of 7+ days both at crude (OR=1.62; 95%C: 1.27-2.06) and adjusted analysis (aOR=1.57; 95%CI:1.22-2.02) among 10,482 COVID-19 patients (419 with ESKD) admitted to 13 New York hospitals from March 1, 2020, to April 27, 2020, and followed through May 27, 2020.[101]

Collectively, these findings underscored CKD as a pivotal prognostic factor influencing multiple outcomes as disease severity, clinical course, mortality in COVID-19 patients and length of hospital, stay, thereby emphasizing the necessity for vigilant renal monitoring and integrated multidisciplinary management.

Glomerular diseases following COVID-19 vaccination

Minimal change disease **(MCD)**

MCD is the leading cause of nephrotic syndrome (NS) in children, resulting in massive proteinuria.[115] Approximately 60% of MCD cases exhibit acute tubular or interstitial injury.[116]

The precise pathogenesis of MCD remains uncertain, although podocyte effacement and glomerular permeability may be induced by T-lymphocyte dysfunction.[117] Experimental data demonstrated an increase in $CD8^+$ suppressor T-cells and cytokine-mediated glomerular injury during the active phase of MCD.[117,118]

Although MCD is primarily idiopathic, secondary triggers—including viral, parasitic or mycoplasma pneumoniae infections, pharmaceutical agents, immunizations, malignancies and

allergic stimuli—have been indicted as possible culprits precipitating or exacerbating primary glomerulonephritis.[119-121]

MCD - which in some cases may present with AKI - is the most frequently reported glomerular disease following COVID-19 vaccination (Supplementary materials, Table 2). Ninety-four cases of MCD following COVID-19 immunization (mean age 45 years) were reported - 59.1% males (=55/93) versus 40.9% females (= 38/93) - one being a relapse after the second dose.[122] Fifty-three-point-one percent (=59/94) of latter cases were newly developed MCD versus 46.9% (=44/93) relapses. Information on previous SARS-CoV-2 infection was not available for 79.5% (=74/93) patients, and 2/19 of the remaining had reportedly been SARS-CoV-2 infected before. Patients were predominantly immunized with Comirnaty (58.5%= 55/94), followed by Spikevax (20.2%=19/94), Vaxzevria (14%=13/94), Janssen (3.2%= 3/94) and CoronaVac (1%=1/94). Most patients developed symptoms after the first COVD-19 vaccine dose (55.5%= 52/94), followed by the second (39.3%= 37/94), third (2.2%= 2/94), booster dose (1%= 1/94), both first and second doses (1%= 1/94), while in one case information was not available (1%= 1/94).[122]

Direct cytopathic effect of vaccines on podocytes has been hypothesized to explain post-immunization MCD. Vaccine-induced T-cell responses heighten interferon-gamma (IFN-γ) and interleukin-2 (IL-2) production, paralleling the acute inflammatory phase of idiopathic NS, eventually leading to podocyte damage. Additionally, vaccine adjuvants might induce immune cross-reactivity or hypersensitivity reactions via molecular mimicry. [123-125]

Although being the leading cause of NS in children, post-COVID-19 vaccination MCD predominantly affects adults. [126] Symptoms onset of MCD generally occurs within three weeks after the first COVID-19 vaccine dose, and subsequent doses may elicit a stronger immunologic response. Nearly 77% of patients reportedly developed AKI after their first vaccine dose, with edema being the most common presenting symptom.[127]

Therapeutic management of MCD primarily involves systemic corticosteroids, although a subset of patients requires hemodialysis.[117] In steroid-resistant cases, rituximab is recommended as a second-line intervention.[127,128] Following treatment, a significant proportion of MCD patients achieve complete or partial remission within three months.[127,130]

Overall, the prognosis of MCD following COVID-19 vaccination is favorable, with the majority of patients achieving complete or partial remission under standard corticosteroid therapy.[122]

Immunoglobulin A nephropathy (IgAN)

In the available body of evidence, IgA nephropathy (IgAN) ranks as the second most prevalent etiology of AKI associated with COVID-19 vaccination (Supplementary materials, Table 2).

IgAN is a chronic glomerulonephritis characterized by mesangial deposition of IgA1 immune complexes, often precipitated by bacterial or viral infections of the upper respiratory or gastrointestinal tracts.[132] Common pathogens include cytomegalovirus, adenovirus, herpes simplex virus, *Toxoplasma gondii*, *Haemophilus parainfluenzae*, and *Staphylococcus aureus*.[132,133]

Patho-physiologically, aberrantly galactosylated IgA1 molecules form autoantibodies (IgG or IgA) against exposed glycan epitopes, leading to immune complex formation and glomerular deposition.[133] Genetic polymorphisms and environmental cofactors may also influence susceptibility.

Although the overall complication rate of IgAN is low, several reports describe coexistent Acute Interstitial Nephritis (AIN).

Gross hematuria remains the predominant clinical feature of IgAN, and management strategies range from conservative observation to steroid therapy, depending on disease severity. Importantly, more than 80% of patients with IgAN experience complete remission after treatment.[128,131]

Severe COVID-19 infection has been associated with elevated serum IgA level and antiphospholipid IgA antibodies (IgA-aPL), possibly originating from the bronchial mucosa. Furthermore, the spike protein's Receptor-Binding Domain (RBD) serves as a key antigenic target for neutralizing antibodies, which may partially overlap with IgA immune activity.[134]

Although rare, several case reports have documented IgAN onset or relapse following vaccinations, including those for typhoid, Haemophilus influenzae type B, and influenza.[135,136]

Notably, 15 post-COVID-19 vaccination cases of IgAN have been reported, particularly following Comirnaty and Spikevax mRNA vaccines, with gross hematuria as a dominant presentation.[131,132,137-142] However, only one case was a newly-developed IgAN following Spikevax.[133] The other fourteen cases were IgAN relapses, either after Comirnaty (N=7)[131,139-142] or Spikevax (N=7) m-RNA vaccines in adults.[137-140]

Moreover, 127 patients presenting with visible hematuria across 22 centers in Japan were reported, mostly developed within 3 days since COVID-19 immunization. Thirty-five cases were flares of pre-existing IgA nephropathy. Seventy out of 90 incident cases of IgA nephropathy were confirmed at renal biopsy.[143,144]

The underlying mechanisms of vaccine-related IgAN remain ambiguous. Mechanistically, mRNA vaccines induce strong T-follicular helper (Tfh) and germinal center B-cell activation, resulting in enhanced antigen-specific T-cell responses, neutralizing antibody production, and long-term immune memory.[8,145] Vaccine-induced immune activation may trigger mucosal IgA overproduction, cross-reaction of anti-glycan antibodies with hypo-galactosylated IgA and deposition of IgA immune-complexes.[131,146]

Perrin *et al.*[140] argued that IgAN cases were unrelated with anti-SARS-CoV-2 antibody response, whereas Rahim *et al*.[141] suggested a delayed-type hypersensitivity reaction following the second COVID-19 vaccine dose. Kudose *et al*.[139] proposed that systemic cytokine surges induced by vaccination may exacerbate pre-existing IgAN by stimulating IgA1 anti-glycan responses.

New cases of IgAN may result from unmasking an undiagnosed IgAN owing to robust immune activation rather than development of new deposits.

Three patients with well controlled pre-existing rheumatioid arthritis reportedly developed novel IgAN – with microscopic hematuria and proteinuria - following m-RNA COVID-19 vaccination, one after the third dose, a second patient one month after the fourth dose, and the third 8 weeks after the 5th dose.[144] Urinary alterations resolved after switching biologics from abatacept or a TNF-α inhibitor to an IL-6 inhibitor, with or without short-term glucocorticoid co-administration or methylprednisolone pulse therapy, suggesting that COVID-19 mRNA vaccination may be implicated, due to mesangial cell proliferation induced by high IL-6 levels.[144]

Taken together, the above evidence highlights a potential immunologic overlap between mRNA vaccine responses and IgA-mediated glomerular injury, though further studies are required to clarify causal mechanisms.

Membranous nephropathy

Membranous Nephropathy (MN) is rare yet well-documented form of GN featured by peripheral edema and immune complex deposition along the glomerular basement membrane, endorsing an autoimmune pathogenic mechanism.[147]

MN - frequently associated with anti-phospholipase A2 receptor (PLA2R) antibodies - has been linked to a variety of infections, including hepatitis B and C viruses, HIV, leprosy, syphilis, and hydatid disease.[150]

Kudose *et al.*[139] reported two MN cases post-COVID-19 infection, one positive while the other negative to anti-PLA2R antibodies, suggesting immune heterogeneity. Since PLA2R antigens are also expressed in the respiratory tract, airway exposure during infection may initiate autoimmune sensitization. Another case of MN was reported in the USA in an 81-year-old Hispanic man PLRA2 negative with pre-existing stage 3 CKD, developing ATI following COVID-19, questioning whether SARS-CoV-2 can trigger or worsen an underlying MN through excessive immune response.[151]

Likewise, Aydin *et al.*[149] described two anti-PLA2R-positive MN following COVID-19 infection, hypothesizing loss of tolerance to PLA2R epitopes triggered by SARS-CoV-2.

Vaccination-associated MN has also been reported, particularly following influenza vaccines, where immune system hyperactivation may provoke immune complex formation between vaccine antigens and host antibodies.[152-154.] In particular, a case of MN following influenza vaccination was reported in a 56-year-old male developing influenza-like illness and acute-onset Nephrotic Syndrome (NS) approximately 20 days following immunization with 2009 H1N1 influenza vaccine.[153] Another case report presented a patient developing acute-onset massive proteinuria with NS and severe AKI soon after receiving the 2009 H1N1 influenza vaccine. The patient encountered a relapse (confirmed by MN and AIM at kidney biopsy) soon after

completely a first initial corticosteroid treatment course. A second prolonged steroid course successfully achieved full clinical remission of NS and retrieval of renal function.[154]

The only case of MN following COVID-19 vaccination was a relapse documented in a 66 year old woman immunized with inactivated Sinovac vaccine, endorsing vaccine-induced immune activation as a plausible mechanism.[149]

Moreover, a recent study compared 186 MN patients immunized with inactivated COVID-19 vaccine versus 167 unvaccinated. There was no difference in the rate of relapse or worsening between the two groups, 10 (13%) in the vaccinated group against 11 (15%) in the unvaccinated group (HR= 0.98, 95%: 0.42–2.33).[155]

Conservative management combined with targeted immunosuppressive therapy are commonly employed against mild MN. Unfortunately, in most instances serum creatinine fails to return to baseline, with incomplete renal recovery.[128,129,148,149]

Overall, these findings reinforce the need for post-vaccination surveillance and longitudinal renal monitoring in MN patients, especially those with autoantibody-mediated disease.

IgG4-related disease (IgG4-RD) nephritis

IgG4-RD is a fibrotic disease of unknown etiology affecting multiple organs, including the kidney, and featured by chronic activation of the immune system and elevated IgE levels.[156] A relapse of IgG4-RD nephritis following mRNA COVID-19 vaccination was reported 2 weeks following a second dose of Comirnaty in a 66 year old male, possibly caused by direct immune activation induced by vaccination, or an allergic reaction or both.[157] Another case of IgG4-RD was reported in a 69-year-old Japanese male admitted with edema the day after second dose of Comirnaty, initially diagnosed with MN based on renal biopsy.[158] However, one month following successful prednisone treatment, the patients was readmitted for a relapse, presenting with very high levels of IgG4 (2,320 mg/dL), which prompted diagnosis of IgG4-RD and resumption of prednisone therapy, which resolved symptoms in a few days.[158]

Nevertheless, the evidence from case reports is clearly insufficient to establish a definitive causality relationship.

Further studies are clearly warranted to explore immune tolerance mechanisms and clarify any causal relationship between IgG4-RD nephritis and COVID-19 vaccination.

IgA vasculitis (Henoch–Schönlein Purpura)

IgA vasculitis, also known as Henoch–Schönlein purpura (HSP), has been associated with bacterial and viral infections, such as streptococcus and parvovirus B19.[159,160]

The pathophysiological process of HSP involves elevated galactose-deficient IgA1 (gd-IgA1) that binds microbial antigens, forming immune complexes that deposit within mesangial cells and induce renal injury. Mucosal inflammation also elevates interleukin-6 (IL-6), further driving gd-IgA1 production and glomerular deposition.[161-164]

Historically, HSP has followed various vaccinations, including influenza, hepatitis A and meningococcal immunizations, possibly through structural antigen mimicry or immune hyperreactivity.[133,165]

Five cases of IgA vasculitis following m-RNA COVID-19 vaccines were reported, 3 newly developed, one in a 40 year old female 3 weeks following 2nd dose of Comirnaty,[166] one in a 67 year old male one month after the first dose of Spikevax[138] and one in a 39 year old man immediately after the 2nd dose of Spikevax.[167] Additionally, two relapses of IgA vasculitis were reported, one in a 79 year old woman 7 days after the first dose of Spikevax[11] and one in a 22 year old females 48h hours after 2nd dose of Spikevax.[138] Anderegg *et al.* suggested that a spike protein–induced immune activation may trigger vasculitic flares in predisposed individuals, while Obeid et al. highlighted spike-specific IgA overproduction as a potential driver of disease recurrence.[11,166-168]

However, in a population-based study on 496,432 Norwegian adolescents, 87,086 unvaccinated, 181,556 vaccinated with one dose, 168,698 with two and 59,092 with 3+ doses of COVID-19 vaccine during April 2021-September 2022, no cases of IgA were recorded during the risk window, which was established at 42 days post COVID-19 immunization.[169]

Although rare, these observations highlight the importance of post-vaccine monitoring in patients with a history of IgA-mediated disorders.

Anti-glomerular basement membrane (anti-GBM) nephritis

Recent case reports described new-onset of anti-glomerular basement membrane (anti-GBM) nephritis following COVID-19 vaccination, typically presenting with gross hematuria, hypertension, anorexia, nausea, fever, and other systemic manifestations (Supplementary materials, Table 2).

Treatment regimens of anti-GBM nephritis often include steroids, cyclophosphamide (CyC), mycophenolate mofetil, and plasma exchange (PLEX). Despite these interventions, clinical outcomes of anti-GBM nephritis remain poor, with approximately 75% of patients showing suboptimal recovery and 25% requiring long-term hemodialysis.[128,129,170]

Anti-GBM nephritis, including Goodpasture's syndrome, has shown temporal associations following both infections and vaccines. [171,172] Epidemiologic clustering during influenza outbreaks supported a potential environmental trigger.[173]

Both COVID-19 infection and mRNA vaccination have been linked to new-developed or recurrent anti-GBM disease.[174,175]

In particular, 2 cases of newly developed anti-GBM nephritis - one 1 day following the 2nd dose of Comirnaty[142] while the other 2 weeks after receiving the second dose of Spikevax 170 - were reported, arguably attributable to vaccine-induced immune response.[170] A further case was reported in a 78 year old female presenting to the emergency department with a one day history of haemoptysis, cough, fever and shortness of breath two days after 2nd dose of Comirnaty.[176]

Molecular mimicry, bystander activation, or autoantigen exposure following immune stimulation may play causative roles.

These findings call for enhanced pharmacovigilance and further mechanistic research to delineate how vaccination-induced immunity may interact with GBM-specific autoantibodies.

ANCA-associated vasculitis

Five cases of newly-developed ANCA-associated vasculitis have been reported following 1st or 2nd dose of Spikevax (N=2)[167,177] 1st, 2nd or 3rd dose of Comirnaty (N=4)[182-186] or 1st dose of Vaxzevria (N=1).[187] Symptoms presented within 1-4 weeks since immunization and included asthenia, anorexia, headache, hematuria, proteinuria, AKI.

The link between infections and ANCA-Associated Vasculitis (AAV) is well-established. Viral RNA recognition by Toll-like receptor-7 (TLR7) may activate type I interferon pathways, leading to autoantibody generation and neutrophilic inflammation. Influenza vaccination has previously been associated with AAV flares, and Jeffs *et al*.[178] demonstrated that viral RNA can induce proteinase 3-ANCA (PR3-ANCA) expression, an effect attenuated by RNAse treatment.[179-182]

The proposed mechanism of vaccine-induced AAV involves mRNA vaccine–driven neutrophil immune response with production of PR3 antibodies[9] or immune response against the spike protein of SARS-CoV-2 precipitating ANCA production in susceptible individuals.[167,183]

Encouragingly, patients with granulomatous forms (including myeloperoxidase (MPO)-positive and ANCA-negative variants) generally respond favorably to immunosuppressive therapy, showing improvement in serum creatinine.[128]

Although extremely infrequent, these AAV cases still emphasize the necessity for continued vigilance and immunologic investigation to delineate vaccine-autoimmunity relationships.

Other renal complications following COVID-19 infection or vaccination

Thrombotic microangiopathy

Thrombotic Microangiopathy (TMA) denotes a clinic-pathologic syndrome of microvascular thrombosis, endothelial injury, microangiopathic hemolytic anemia, thrombocytopenia, frequently associated with AKI. In severe COVID-19 the combination of systemic complement activation, endothelial dysfunction and inflammatory cytokine release has been implicated in secondary TMA-like presentations.[188]

Cases of TMA temporally associated with SARS-CoV-2 infection and a number of reports describing renal TMA responding to complement blockade (eculizumab) underly the relevance of complement-mediated pathways in selected patients.[189]

Post-COVID-19 vaccination syndromes phenotypically overlapping with TMA—most notably Vaccine-Induced Immune Thrombotic Thrombocytopenia (VITT) after adenoviral-vector vaccines—are mediated by platelet-activating antibodies (anti-PF4) and can yield thrombotic microangiopathic lesions in multiple organs, including the kidney. Early recognition and management (anticoagulation treatment, intravenous immunoglobulin, and methylprednosolone therapies) are critical for a favorable prognosis.[190]

Renal histopathology in TMA cases commonly shows glomerular capillary thrombosis, endothelial swelling, mesangiolysis and fibrin thrombi with variable deposition of terminal complement factors (C5b-9). These findings support a model in which complement dysregulation and endothelial injury converge to produce irreversible microvascular damage in predisposed hosts.[191]

Acute tubulointerstitial nephritis

Acute Tubulointerstitial Nephritis (ATIN) and Acute Interstitial Nephritis (AIN) is an immune-mediated inflammatory condition of the renal interstitium and tubules commonly triggered by drugs, infections, or immune checkpoint perturbation. Renal biopsies of patients with AIN classically show interstitial edema, neutrophil-rich tubulointerstitial inflammatory infiltrate, and tubulitis.[192]

Clinical presentation typically includes acute rise in serum creatinine, sometimes with fever, rash, or sterile pyuria; withdrawal of the offending agent and short-course corticosteroids often lead to disease recovery.[193]

AIN has been reported after SARS-CoV-2 infection.[193-195] Notably, immune-checkpoint inhibitor–associated ATIN reported in the context of COVID-19 infection illustrates that infection-triggered loss of self-tolerance may unmask renal autoimmunity even when direct viral particles are not identified in renal tissue.[192]

Several cases of ATIN have also been reported following different COVID-19 vaccines (predominantly mRNA formulations).[195] In particular, 22 cases of newly developed AIN were reported following SARS-CoV-2 infection and 36 after COVID-19 vaccination (35 confirmed at

renal biopsy).[195] Patients were predominantly European (47.2%), followed by Americans (25%) and Asians (22.2%). Nineteen (52.8%) patients were immunized with Comirnaty, 8 (22.2%) with Spikevax, 7 (19.4%) with Vaxzevria, 1 with Sinovac and 1 with Coronavac.[195] Twelve patients developed symptoms after first dose, 21 after a median time of 7 (range 1-82) days since 2nd COVID-19 vaccine dose and only 3 following a median time of 21 (21.30) days since the third dose of m-RNA vaccines (2 after Comirnaty, 1 after Spikevax).[195]

The potential mechanism proposed to explain ATIN induced by COVID-19 vaccination was formation of a hapten combining vaccine and peptides, with subsequent activation of dendritic cells and immunological cascade.[195]

Nonetheless, the causal relationship between ATIN and COVID-19 vaccination remains challenging due to the impact of pre-existing comorbidities and medications. For instance, Merino et al reported a case of ATIN following COVID-19 vaccination with Pfizer-BioNTech vaccine in a patient aged 78 years old male. However, the patient had pre-existing hypertension, hyperuricemia, dyslipidemia, diabetes mellitus with good metabolic control, and stage 3a-b/A3 CKD. Moreover, the patient was taking a number of medications including allopurinol, which may have also contributed to increase the risk ATIN.[196]

Scleroderma renal crisis

Scleroderma Renal Crisis (SRC) is a severe microvascular complication of systemic sclerosis, caused by decreased renal blood flow due to endothelial damage, intimal proliferation, and narrowing of the kidney arteries, resulting in increased level of renin, malignant hypertension, rapidly progressive renal failure and microangiopathic hemolysis.[197] Although SRC is classically linked to disease phenotype and corticosteroid exposure, case reports have described SRC occurring or relapsing after influenza or even COVID-19 infection.[197-199] A case of SRC was reported following COVID-19 in a patient in long remission before the infection and without any risk factors for SRC, suggesting the triggering role of systemic immune activation and endothelial injury in susceptible individuals.[199] COVID-19 can trigger production of autoantibodies and in genetically predisposed patients may cause the onset or exacerbation of autoimmune diseases.[198] Beyond direct tissue damage, SARS-CoV-2 might also amplify the

ongoing systemic sclerosis manifestations during the acute phase of infection, contributing to organ damage.[200]

SRC has been linked also post COVID-19 vaccination.[201,202] In particular, a case of SRC was reported in a 34 years old woman - without history of SARS-CoV-2 infection - one week following immunization with Comirnaty. Twenty-four hours after vaccination she suffered from acute vision loss and hypertension (220/110 mmHg) and was diagnosed with stage 1 hypertensive retinopathy.[201] Secondary ischemic glomerular changes without thrombotic glomerular microangiopathy were identified at renal biopsy.[201] Blood pressure and renal function normalized after one week of antihypertensive medications yet a second dose of COVID-19 vaccine was refrained.[201]

Management of SRC remains centered on immediate renin–angiotensin system blockade (ACE inhibitors) and supportive care, whose early initiation markedly improves outcomes. Recognition of potential triggers (including infection or immune stimulation) is critical when monitoring patients with systemic sclerosis.[203]

Although further evidence is needed to confirm the causal association, monitoring vascular complications in patients with systemic sclerosis following COVID-19 vaccination is recommended.

Granulomatous interstitial nephritis

Granulomatous interstitial nephritis (GIN) has been described after several bacterial infections[204] and even after intravesical bacillus Calmette-Guerin therapy for bladder cancer.[205]

A case of GIN associated with vasculitis was reported following the first dose of Vaxzevria, although the pathogenesis could not be explained.[206]

When observed post-COVID-19 infection or vaccination, (GIN) likely reflects macrophage/Th1 hyperactivation and requires histologic confirmation for accurate diagnosis and targeted therapy.[207]

Integrative perspective

Collectively, glomerular diseases as MCD, IgAN, MN, anti-GBM disease and ANCA-associated vasculitis share overlapping mechanistic themes: immune dysregulation, endothelial injury, complement activation, and—occasionally—autoantibody formation. Recognizing these mechanistic patterns supports a rational diagnostic approach (timely biopsy and hematologic/complement testing where indicated) to guide targeted therapy (e.g., immunosuppression, plasma exchange, complement inhibitors) in selected patients. Future research should prioritize systematic registries, case–control studies, and genetic/biomarker investigations to identify individuals at heightened risk and to define evidence-based management algorithms.[192]

Clinical implications and recommendations for practice

COVID-19–associated AKI carries significant prognostic implications, particularly in patients with pre-existing risk factors as CKD, diabetes, hypertension, cardiovascular disease, older age, or kidney transplantation; therefore, early recognition through routine monitoring of serum creatinine, urine output, and urinalysis—even in the absence of overt dysfunction—is essential. High-risk patients require careful volume and blood pressure management, avoidance of nephrotoxins and individualized adjustment of immunosuppressive therapy. Although vaccine-associated glomerular diseases such as MCD, IgAN, MN, anti-GBM disease, and ANCA-associated vasculitis have been reported, these events are relatively rare, often respond to corticosteroid-based therapy, and occur far less frequently than AKI from COVID-19 infection. Therefore, COVID-19 vaccination is still strongly recommended, with post-immunization monitoring restricted to individuals with prior glomerular disease. Patients who develop AKI during infection should receive long-term follow-up due to substantial risk of progression to CKD or dialysis, and multidisciplinary collaboration among nephrology, infectious disease and immunology consultants can optimize diagnosis and treatment. Ultimately, integrating early detection, risk stratification, careful management of comorbidities and proactive patient follow-up into clinical practice may reduce kidney-related morbidity and improve patient outcomes within the context of COVID-19 infection and vaccination.

Conclusions

This comprehensive narrative review underscored the critical importance of recognizing, monitoring, and effectively managing renal complications arising from COVID-19 infection or vaccination. The global emergence of COVID-19 has redefined the landscape of clinical nephrology, emphasizing the interconnectedness between infectious diseases and renal pathophysiology.

While the development and worldwide deployment of COVID-19 vaccines has been instrumental in reducing infection rates, hospitalizations and SARS-CoV-2 related mortality, clinicians must remain vigilant to potential renal sequelae that may manifest following infection or immunization.

The evidence presented in this review revealed that COVID-19 can precipitate a broad spectrum of renal disorders, ranging from AKI to CKD. These outcomes are often exacerbated by pro-inflammatory, prothrombotic, and endothelial-damaging mechanisms induced by SARS-CoV-2. Moreover, although vaccine-associated renal adverse events are infrequent, their occurrence warrants careful evaluation, particularly in patients with pre-existing renal conditions or autoimmune predispositions.

The findings of this review highlight the necessity for continuous pharmacovigilance, renal function surveillance and longitudinal cohort studies to fully elucidate the short- and long-term renal effects of both SARS-CoV-2 infection and COVID-19 vaccination. Such research will be indispensable for developing evidence-based clinical guidelines that optimize patient safety and therapeutic decision-making.

In clinical practice, integrating renal monitoring protocols into COVID-19 management pathways can facilitate early detection of nephrotoxicity and enable timely therapeutic intervention. Furthermore, a multidisciplinary approach—involving nephrologists, infectious disease specialists, and immunologists—is essential to navigate the evolving challenges posed by COVID-19.

Ultimately, by fostering a deeper understanding of the renal manifestations linked to both COVID-19 infection and vaccination, healthcare systems can enhance patient outcomes,

minimize long-term complications, and strengthen preparedness for renal challenges posed by eventual further viral pandemics.

List of Abbreviations

AAV, ANCA-associated vasculitis
ACE, Angiotensin-converting enzyme
ACE2, Angiotensin-Converting Enzyme 2 receptor
ACE2, Angiotensin-Converting Enzyme 2 receptor
ATI, Acute tubular injury
AIN, Acute Interstitial Nephritis
AKI, Acute Kidney Injury
AKD, acute kidney disease
ANCA, anti-Neutrophil Cytoplasmic Autoantibody
anti-GBM, anti-Glomerular Basement Membrane nephritis
ARDS, Acute Respiratory Distress Syndrome
ATIN, Acute Tubulointerstitial Nephritis
BCG, Bacillus Calmette-Guérin
CKD, Chronic kidney disease
CyC, cyclophosphamide
ESKD, End-stage kidney disease
FSGS, focal segmental glomerulosclerosis
gd-IgA1, galactose-deficient IgA1
GFR, Glomerular Filtration Rate
GIN, granulomatous interstitial nephritis
HR, hazard ratio
HSP, Henoch–Schönlein purpura
KRT, Kidney Replacement Therapy
ICU, Intensive care unit
IFN-γ, interferon-gamma
IgA-aPL, antiphospholipid IgA antibodies
IgA-aPL, antiphospholipid IgA antibodies
IgAN, IgA Nephropathy
IgG4-RD, IgG4-related disease
IL-2, interleukin-2
IL-6, interleukin-6
KDIGO, Kidney Disease: Improving Global Outcomes
MCD, Minimal Change Disease
MN, Membranous Nephropathy
MPO, myeloperoxidase
mRNA, messenger ribonucleic acid
NS, nephrotic syndrome
OR, Odds Ratio
PLA2R, anti-phospholipase A2 receptor

PLEX, plasma exchange
PCR, Polymerase Chain Reaction
PR3ANCA, proteinase 3-ANCA
RAAS, renin–angiotensin-aldosterone system
RBD, Receptor-Binding Domain
RR, relative risk
SRC, Scleroderma renal crisis
TAC, Tacrolimus
Tfh, T-follicular helper
TLR7, Toll-like receptor-7
TMA, Thrombotic Microangiopathy
TMPRSS2, transmembrane serine protease 2
USRDS, U.S. Renal Data System
WHO, World Health Organization
VITT, Vaccine-Induced Immune Thrombotic Thrombocytopenia

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Online supplementary materials

Table 1. Acute kidney injury (AKI) and chronic kidney disease (CKD) associated with COVID-19 infection: incidence, prevalence, risk factors, clinical pattern and patient outcome.

Table 2. Renal complications following COVID-19 vaccination.