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# Kidney injury following JN.1; a mini-review on current evidence

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**Abstract**
The SARS-CoV-2 JN.1 variant, a descendant of the Omicron BA.2.86 lineage, emerged as a globally dominant COVID-19 strain by early 2024. This virus characterized by heightened transmissibility and immune evasion, which represents as an evolution in the virus's ability to adapt across with maintaining lower severity versus earlier variants. Since direct evidence on JN.1 and renal involvement is lacking, the variant is expected to cause kidney injury through the well-characterized SARS-CoV-2 mechanisms of direct renal infection and systemic inflammatory damage observed in COVID-19 patients in general.
**Keywords:** SARS-CoV-2 JN.1 variant, Acute kidney injury, Chronic kidney disease, COVID-19, Apolipoprotein L1
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## Introduction

The SARS-CoV-2 JN.1 variant, a descendant of the Omicron BA.2.86 sublineage, has emerged as a dominant global strain due to its enhanced transmissibility and immune evasion capabilities (1). This virus was first identified in August 2023 and the classified as a variant of interest by the WHO in December 2023 (2). Prior studies found that, JN.1 accounts for over 90% of sequenced cases in multiple regions as of early 2024 (3). Though, this new variant pretends a lower risk of severe disease compared to earlier variants, however its rapid spread emphasizes the need for sustained vigilance (4). This virus carries over 30 spike protein mutations, including the critical L455S mutation in the receptor-binding motif (5). This mutation reduces ACE2 receptor-binding affinity while enhancing infectivity and immune evasion by hindering antibody neutralization (5,6). Preliminary studies found that JN.1's spike protein adopts conformational changes that improve host cell entry, particularly in lung cells, resembling pre-Omicron variants like Delta (2,7). The transmissibility of JN.1 is 1.5–2 times higher than BA.2.86, driven by its ability to thrive in cold, dry climates and evade population immunity (8). Reports showed that, between November 2023 and March 2024, it caused surges across 121 countries, peaking at 95.1% of global sequences by mid-2024 (2). Common symptoms are resembling to COVID-19 and are sore throat, fever, dry cough, fatigue, nausea/vomiting and loss of taste/smell (9). The regions most affected by the JN.1 variant's spread are primarily in Asia, Singapore, Hong Kong, Thailand, China, and India with significant surges and increased healthcare burdens reported in these areas (10). Beyond Asia, JN.1 has been documented in over 41 countries, including the United States, United Kingdom, France, Canada, and Sweden, where it has become one of the fastest-growing variants, but the most intense recent impact and public health concern remain focused in Asian countries (11). Recent studies found that, JN.1 exhibits moderate antibody escape, reducing neutralizing antibody titers compared to ancestral strains (3,12). Therefore, updated mRNA vaccines targeting Omicron lineages maintain an appropriated effectiveness against symptomatic infection and a suitable protection against severe disease (13,14). However, declining immunity and low-booster uptake have facilitated its spread (15). It is postulated that, vulnerable populations like elderly, immunocompromised, diabetics and those with comorbidities remain at higher risk of severe outcomes and its complication including kidney involvement (16).

## Search strategy

For this study, we searched PubMed, Web of Science, EBSCO, Scopus, Google Scholar, Directory of Open Access Journals (DOAJ) and Embase, using different keywords including SARS-CoV-2 JN.1 variant, acute kidney injury, chronic kidney disease, COVID-19 and apolipoprotein L1.

## JN.1 versus other Omicron sub-variants

The SARS-CoV-2 JN.1 variant exhibits significantly higher infectivity compared to other Omicron sub-

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

The SARS-CoV-2 JN.1 variant is expected to affect kidney function through mechanisms similar to those established for SARS-CoV-2 infection. Its renal effects from other variants. SARS-CoV-2 JN.1 could affect kidney function primarily by direct infection of ACE2-expressing renal cells leading to acute tubular injury and glomerular damage, combined with systemic inflammatory and vascular effects that contribute to acute kidney injury and long-term renal function decline.

variants, particularly its direct ancestor BA.2.86 (1). This condition increased infectivity is primarily attributed to the unique L455S mutation in the spike protein's receptor-binding motif, which enhances viral entry efficiency into human nasal epithelial cells and improves spike protein cleavage (1,17). Despite a decreased binding affinity to the ACE2 receptor relative to BA.2.86, JN.1 compensates with enhanced cell entry mechanisms and immune evasion, making it more transmissible (18). Laboratory studies demonstrate that JN.1 infects primary human nasal epithelial cells more effectively than BA.2.86, a key factor in its rapid global spread and dominance (19). This mutation alters the spike protein's interaction with ACE2 and increases the number of hydrogen bonds within the spike structure, facilitating better viral attachment and fusion (20). Compared to other Omicron sub-variants, the JN.1 transmissibility is estimated to greater, contributing to its rapid replacement of earlier variants worldwide since late 2023 (1). While it retains immune evasion features typical of Omicron lineages, JN.1's enhanced infectivity distinguishes it as one of the most transmissible variants currently circulating (21). The immune evasion mechanism of the SARS-CoV-2 JN.1 variant differs from previous variants primarily due to the novel L455S mutation in the spike protein's receptor-binding motif (22). This mutation alters the spike protein structure, decreasing its binding affinity to the human ACE2 receptor compared to its ancestor BA.2.86, yet paradoxically enhances infectivity and immune escape (23). Unlike earlier variants, JN.1's L455S mutation significantly hinders the ability of neutralizing antibodies, especially those targeting the receptor-binding domain to attach effectively to the virus (24). This circumstance leads to a more robust evasion of antibody-mediated immunity, including a marked reduction in neutralization by antibodies derived from previous infections or vaccinations (25). Studies show JN.1 exhibits a greater fold reduction in neutralizing antibody titers than BA.2.86 and other Omicron sub-variants, indicating stronger immune escape (26). Moreover, JN.1 evades a broad range of neutralizing antibodies, including those derived from common B cell germlines (IGHV3-53/3-66), which were effective against earlier variants (27). This immune evasion is partly compensated by preserved Fc effector functions mediated by class 3 antibodies binding outside the ACE2 interface, which may contribute to the relatively lower severity of disease despite immune escape (28).

## Kidney injury following JN.1

There is no specific published evidence directly linking the SARS-CoV-2 JN.1 variant to unique or distinct renal involvement compared to other variants (29). However, according to the previous evidence on SARS-CoV-2 kidney pathology, the mechanisms by which COVID-19 causes kidney injury are likely applicable to JN.1 as well (30). SARS-CoV-2 infection can cause kidney damage through several pathways by direct infection of renal cells via ACE2 receptors expressed on podocytes and proximal tubular cells, leading to acute tubular injury and collapsing glomerulopathy (31). In addition, indirect effects including systemic inflammation, cytokine storm, endothelial dysfunction, hypercoagulability, and hypoxia, which contribute to acute kidney injury (AKI) (32). Moreover, residual inflammation and acute injury can result in progressive kidney function decline, even in mild-to-moderate COVID-19 cases (33). In fact, AKI occurs in a significant portion of hospitalized COVID-19 patients which ranging from 5% to over 30% in severe cases (34,35). This condition is associated with higher mortality and risk of chronic kidney disease progression (36). Histopathological studies have detected viral RNA and particles in kidney tissue, supporting direct viral involvement (37). Given that JN.1 retains the ability to infect human cells via ACE2 and causes systemic infection, it is plausible that it can induce kidney injury through similar mechanisms as earlier variants (38). However, no current data specifically describe renal complications uniquely attributable to JN.1 (39). Previous studies demonstrated that, SARS-CoV-2 impacts renal tissue, following direct viral infection of kidney cells (40). This virus binds to ACE2 receptors, which are highly expressed on renal proximal tubular epithelial cells, podocytes, mesangial cells, and parietal epithelial cells (41). This allows direct viral entry and replication in these cells, causing cytopathic effects and acute tubular injury (42). One of the presentations of renal involvement is AKI, which is common in COVID-19 patients, mainly in severe cases (43). Acute kidney injury results from direct viral damage, immune dysregulation, cytokine storm, endothelial dysfunction, and hypercoagulability (32). Viral particles have been identified in renal tubular cells and podocytes, supporting direct kidney tropism (44). Studies show that COVID-19 accelerates decline in estimated glomerular filtration rate, especially in hospitalized patients, increasing risk for chronic kidney disease progression (45). However, study regarding indirect systemic effects, detected that, hypoxia, coagulation abnormalities, multi-organ injury, and nephrotoxic medications also contribute to renal impairment during infection (46). Since JN.1 retains the ability to infect human cells via ACE2 and causes systemic

infection, it likely induces kidney injury through these established pathways (38). Therefore, JN.1 can cause acute kidney injury, proteinuria, and accelerated kidney function decline similar to other SARS-CoV-2 variants (47).

## Focus on genetic factors

Several studies showed that, individuals with two risk alleles of apolipoprotein L1 (APOL1) variants, (G1 and G2), predominantly found in people of West and Central African ancestry, have a significantly increased risk of developing kidney diseases such as focal segmental glomerulosclerosis (FSGS), collapsing glomerulopathy, and HIV-associated nephropathy (48). These APOL1 risk alleles confer an increased risk for certain kidney diseases and are associated with worse outcomes in viral-associated nephropathies (49). A "second hit" such as viral infection or inflammation often triggers disease in genetically susceptible individuals (50). Other podocyte and glomerular gene mutations are consisted of mutations in genes affecting podocyte structure and function such as NPHS1, NPHS2, WT1, TRPC6, INF2, that predispose individuals to nephrotic syndrome and glomerular diseases, which can be exacerbated by viral infections (51). In this regard, a genetic predisposition to AKI is also recently explained (52). Variants in genes related to immune response and kidney function may influence susceptibility to AKI during systemic infections like COVID-19 (53).

## Conclusion

There are no specific studies yet identifying genetic factors that make individuals more susceptible to kidney damage specifically from the SARS-CoV-2 JN.1 variant. However, based on general knowledge of genetic susceptibility to kidney disease and COVID-19-related kidney injury, some genetic variants are known to increase the risk of kidney damage in viral infections, which likely applies to JN.1 as well. Since JN.1 shares the fundamental mechanism of kidney cell infection via ACE2 and induces systemic inflammation similar to other SARS-CoV-2 variants, individuals with high-risk genetic backgrounds (especially those with APOL1 risk alleles) may be more vulnerable to kidney injury or collapsing glomerulopathy triggered by JN.1 infection.

### Conflicts of interest

The author declares that she has no competing interests.

### Ethical issues

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

### Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the authors utilized Perplexity to refine grammar points and language style in writing. Subsequently, the authors thoroughly reviewed and edited the content as necessary, assuming full responsibility for the publication's content.

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