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Systematic Review

Coronavirus Disease-2019 Infection-Associated Glomerular Diseases

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ABSTRACT

Background

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) virus pandemic of 2019 (coronavirus disease-2019 (COVID-19)) has led to unimaginable global deaths and serious morbidities among survivors. From a renal perspective, COVID-19 was also reported to be complicated by glomerular diseases. We herein present a systematic review of the distributive pattern of glomerular diseases (GNs) reported in association with COVID-19 infection.

Methods

We searched National Library of Medicine's (NLM), Excerpta Medica Database (EMBASE) and the World Health Organization's (WHO) COVID-19 database for case reports, case series, and observational studies that reported GNs associated with COVID-19 infection. Two reviewers independently extracted relevant data. The current study was registered with Research Registry #1736 on November 20, 2023.

Results

Of the 1261 articles identified, fifty-eight articles pertained to COVID-19 infection-related GNs. One hundred sixty-two *de novo* GN cases were claimed to be related to the COVID-19 infection. Among these, the top 3 were focal segmental glomerulosclerosis (focal segmental glomerulosclerosis (FSGS), 90 out of 162, 55.6%), antineutrophil cytoplasmic autoantibody (ANCA)-associated GN (34 out of 162, 21.0%), and IgA nephropathy (IgAN) and thrombotic microangiopathy (TMA), including thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, or not specified) (both with 12 out of 162, 7.4%). In comparison, excluding diabetic kidney disease, the top 3 GNs globally in the pre-COVID era were FSGS (17.4%), IgAN (16.5%), and membranous nephropathy (MN) (12.1%).

Conclusion

Although most commonly reported, FSGS was not the only GN associated with COVID-19. The distributive pattern of GNs reported to be associated with COVID-19 infection differed significantly compared with that in the pre-COVID era. This difference may lend support to COVID-19 related effects on the most commonly reported GNs.

Keywords

COVID-19; SARS; Glomerular diseases; Focal segmental glomerulosclerosis; ANCA vasculitis; IgA nephropathy; Thrombotic microangiopathy; Thrombotic thrombocytopenic purpura.

BACKGROUND

The severe acute respiratory syndrome coronavirus 2 (SARS-CoV 2) virus pandemic of 2019 has claimed over 6.9 million lives in less than 3.5 years and left at least 65 million individuals with chronic sequelae involving both pulmonary and extrapulmonary organ systems, known collectively as "*long coronavirus disease (COVID)*".[1,2] Acute kidney injury (AKI) with subsequent function

decline and the development of chronic kidney disease have been reported to be serious sequelae. According to a 2020 systematic meta-analysis, the incidence of AKI in COVID-19-infected individuals was 8.9%.[3] During the early peak of the COVID-19 pandemic, acute kidney injuries were reported to affect up to 70-80% of patients admitted to the intensive care unit.[4] In a cohort of over 1.7 million US veterans, including 89,216 patients who were 30-day survivors of COVID-19 patients, Bowe et al[5] reported an excess estimated glomerular filtration rate decline of -3.26 (-3.58 to -2.94, 95% CI), -5.20 (-6.24 to -4.16), and -7.69 (-8.27 to -7.12) mL/min per 1.73 m^2 per year, respectively, in non-hospitalized, hospitalized, and those admitted to intensive care during the acute phase of COVID-19 infection compared with non-infected individuals. The pathogenesis of AKI was attributed to the direct effects of the virus within the kidneys, leading to endothelial damage, coagulopathy, complement activation, inflammation, and the development of collapsing glomerulopathy, as well as indirect effects involving associated severe hemodynamic instability and presumed organ crosstalk.[4,5]

The earliest reports of kidney biopsy findings in infected individuals confirmed a predominance of acute tubular injury but also revealed collapsing glomerulopathy and thrombotic microangiopathy.[6] We herein perform a systematic review to study the epidemiology of GNs associated with COVID-19 infection.

METHODS

A This systematic review was conducted according to the preferred reporting items for systematic reviews (PRISMA) guideline.[7]

Search Criteria Included

COVID, SARS, or coronavirus (MeSH Terms) AND ((y_5 (Filter)) AND (fha (Filter)) AND (casereports (Filter)) OR dataset (Filter) OR observational study (Filter)) AND (humans (Filter)) AND (English (Filter)) AND (alladult (Filter)) AND (glomerular disease or glomerulonephropathy or glomerulitis or glomerulonephritis or nephropathy or nephritis or glomerulonephritides or nephritides) in title, abstract, and keywords. The data were accessed on April 11, 2023, from the National Library of Medicine (NLM) and EMBASE. Duplicate articles were manually removed from the Microsoft-ExceL spreadsheet following the alphabetization of all authors' names and article titles from the combined dataset. Data from the World Health Organization's (WHO) COVID-19 database were accessed on April 21, 2023. The same search criteria used above were applied to the WHO database, but with the EMBASE database removed. The current study was registered with Research Registry #1736 on November 20, 2023.

Article Selection

Only articles that reported GN cases in adults in the English language related to the COVID-19 infection were selected. Articles related to reviews, mechanisms of disease, epidemiology, treatment strategy, acute kidney injury not related to GNs, non-renal-related topics, COVID-19-vaccine-related GNs, and pediatric and kidney transplant cases were excluded. Two independent reviewers extracted data, with the senior reviewer confirming the accuracy of the data acquisition by the junior reviewer.

RESULTS

Our search identified 1261 articles. After duplicate removal, title screening, and abstract and full text reviews, 117 articles met criteria for inclusion (Figure 1). There were 162 cases with the actual COVID-19 infection (Table 1).

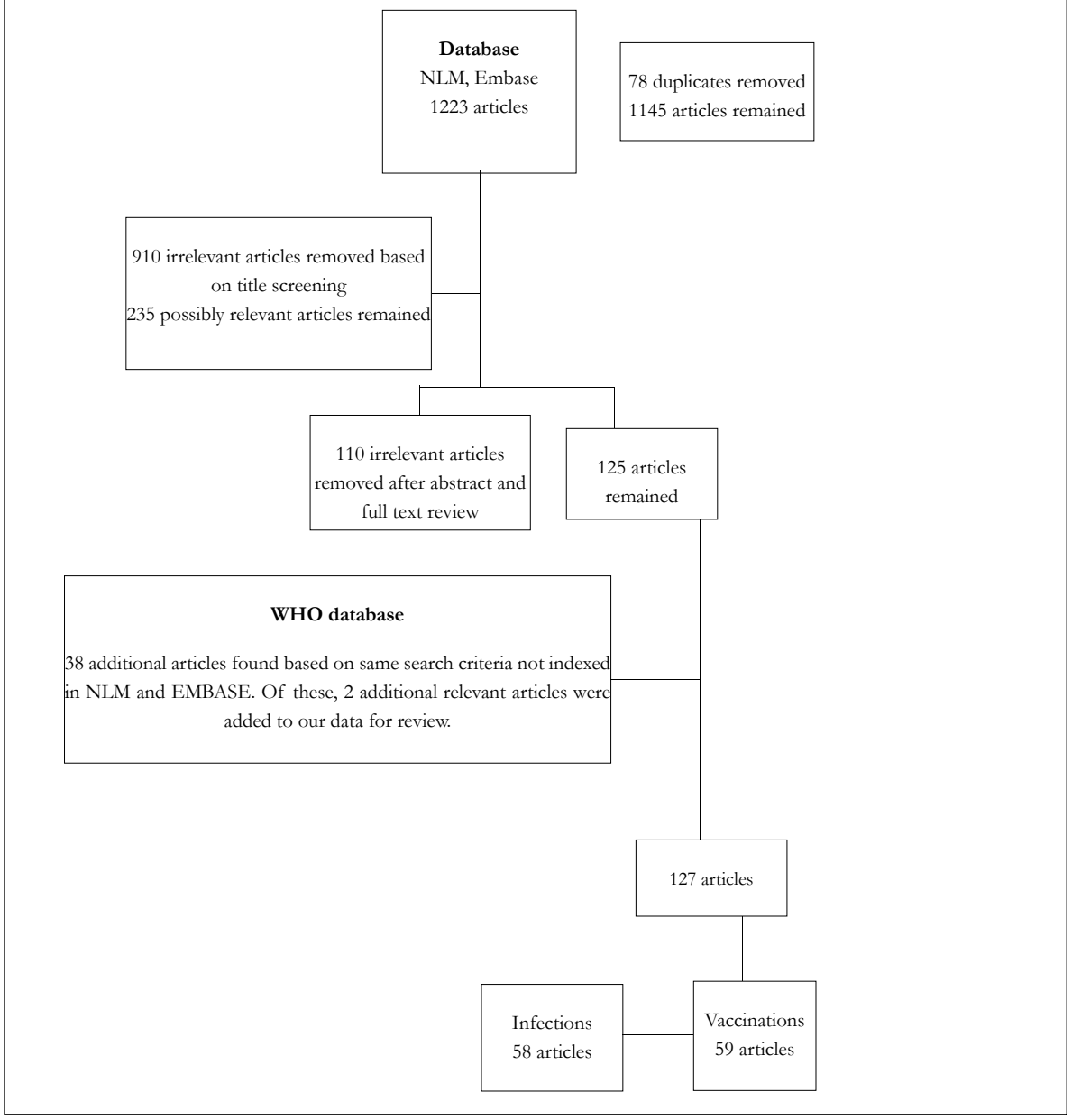
The top 3 most common GNs associated with COVID-19 infection were FSGS (90 out of 162, 55.6%), ANCA-associated GN (34 out of 162, 34%), and IgAN and TMA, including thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, or not

Table 1. *Global Distribution of Glomerular Diseases Reported in the pre-COVID Era and the Distribution of Reported COVID-19 Infection-related Glomerular Diseases during the Pandemic*

Glomerular Diseases	Pre-COVID Global Distribution (%)	COVID-19 Infection (%)
FSGS	17.4	55.6
IgAN/HSP	16.5	7.4
DGS	13.9	0
MN	12.1	3.7
LN	12.2	0.6
ANCA	6.2	21
MCD	5.2	1.2
MPGN/C3GN	3	0
Renal amyloid	2.9	0.6
TTP/HUS	2.5	7.4
Others	8.1	2.5
Total	100	100

FSGS: Focal segmental glomerulosclerosis; IgAN/HSP: IgA nephropathy/Henoch Schönlein purpura; DGS: diabetic glomerulosclerosis; MN: Membranous nephropathy; LN: Lupus nephropathy; ANCA: Anti-neutrophil cytoplasmic antibody associated glomerulonephritis; MCD: Minimal change disease; IC-MPGN/C3GN: Immune complex-mediated membranoproliferative glomerulonephritis or C3 glomerulonephropathy; TTP/HUS: Thrombotic thrombocytopenic purpura or hemolytic uremic syndrome.

Figure 1. *Search criteria: COVID, SARS, or coronavirus (MeSH Terms) AND ((y_5(Filter)) AND (fha (Filter)) AND (case reports (Filter) OR dataset (Filter) OR observational study (Filter)) AND (humans (Filter)) AND (english (Filter)) AND (alladult (Filter)) AND (glomerular disease or glomerulonephropathy or glomerulitis or glomerulonephritis or nephropathy or nephritis or glomerulonephritides or nephritides) in title, abstract, and keywords; the National Library of Medicine (NLM) and EMBASE databases were accessed on April 11, 2023; and the World Health Organization database was accessed on April 21, 2023.*



specified TTP/HUS (both at 12 out of 162, 7.4%).

Fifty-eight out of the 90 cases (64.4%) of FSGS associated with COVID-19 infection were reported to be collapsing FSGS (cFSGS). Among studies that reported race for patients affected by cFSGS, 42 out of 45 (72% of patients) were of African descent.

Among 34 (21%) cases of ANCA-associated GN, six were positive for antibody against proteinase 3, and five cases were positive for antibody against myeloperoxidase. Twenty-three cases did not document ANCA specificity.

There were six cases of membranous nephropathy (3.7%). Three cases were positive for the anti-phospholipase A2 receptor antibody, and three were negative for the antibody. Other GNs reported are summarized in the table.

DISCUSSION

Focal segmental glomerulosclerosis, a non-specific pattern of glomerular injury, is known to be associated with a wide array of causes, including podocytopathic circulating factors, inherited mutations involving podocyte structural proteins, viral infections, drugs, or adaptive responses associated with acute kidney injury or

severe hemodynamic compromise. Collapsing FSGS is an FSGS variant that may be attributed more specifically to viral infections, drugs, renal ischemia, and high-risk variants of the *APOL1* gene.[8] In the pre-COVID era, FSGS was reported to occur at 17.4% of all GNs reported globally.[9]

Our data revealed that FSGS was the most commonly reported COVID-19-associated GN, which occurred in 90 out of 162 infected patients (55.6%). Of note, 64.4% (58 out of 90 reported cases) of all FSGS cases reported were of the collapsing variant. The observation that FSGS was the most common GN that occurred in infected individuals is consistent with our current understanding that the presence of the virus within podocytes per se is likely the primary inciting factor. The associated severe hemodynamic instability and systemic inflammatory response to acute viral infections, with or without the presence of high-risk variants of the *APOL1* gene, likely secondarily contribute to the severe impairment of basic podocyte cellular functions leading to cFSGS.[4,8,10-12]

Of interest, in a multicenter retrospective case series of patients with COVID-19 infections who underwent kidney biopsy in Paris and its metropolitan area, *APOL1* genotyping in 11 patients (7 cFSGS, 4 FSGS not otherwise specified) revealed all 7 out of 7 patients with cFSGS harbored the high-risk combinations of *APOL1* variants, including either G1/G1 or G1/G2, while only 2 of the 4 patients with other FSGS variants had the G1/G2 combination.[13] The study highlighted the association between APOL1 high-risk alleles and cFSGS.

Notably, in the current systematic review, studies that reported race revealed a predominance (42 out of 45 (72%)) of African descent among patients affected with cFSGS, a group whose prevalence for the high-risk *APOL1* alleles G1/G2 has been reported to be 20-22% and 13-15%, respectively.[14] The predominance of African descent among patients affected by cFSGS in the current analysis is consistent with the link between high-risk *APOL1* variants and cFSGS.

Anti-neutrophil cytoplasmic antibody against either proteinase 3 or myeloperoxidase-associated glomerulonephritis was the second most common GN reported to be associated with COVID-19 infection, occurring at 18.9% (21 out of 111). It is noteworthy that ANCA-associated GN only occurred in 6.2% of all reported GNs globally in the pre-COVID era.[9]

It is known that the inciting event for ANCA-vasculitis is cytokine priming of neutrophils, typically in association with an acute infection, in individuals with autoantibody production against two well-recognized antigens, including proteinase 3 and myeloperoxidase.[15] Coincidentally, in COVID-19 infection, large cytokine release is a known systemic response.[15,16] More specifically, interleukin-6 (IL-6), a cytokine known to be elevated in COVID-19 infections, has been suggested to play a central role in the pathogenesis of ANCA-related vasculitis.[17,18] Elevated serum IL-6 levels and increased expression of IL-6 at lesion sites have been shown in patients with ANCA-associated vasculitis. Additionally, in the pre-COVID-19 era, response to the IL-6 inhibitor tocilizumab in patients with ANCA-associated vasculitis has also been reported.[18,19] Individuals who developed ANCA-vasculitis with or without glomerulonephritis during the COVID-19 pandemic were likely those who could produce autoantibodies against proteinase 3, myeloperoxidase, or both, whose disease was activated following neutrophil priming by the great cytokine release associated with both infections.

Another proposed mechanism for the development of ANCA-associated vasculitis involves the formation of neutrophil extracellular traps (NETs) that may be triggered by COVID-19 disease or vaccination. Elevated levels of circulating NETs have been shown in patients with ANCA-associated vasculitis as well as within the kidney biopsies of affected individuals. NETs contain pro-inflammatory proteins that have been implicated in causing direct endothelial cell damage and complement activation, leading to tissue inflammation and damage as well as hypercoagulability and thrombosis.[20-22]

Twelve out of 162 (7.4%) COVID-19-associated GN cases were reported as IgAN.

In terms of IgAN, the pathogenesis is complex and thought to involve the deposition of circulating IgG2 and, to a lesser extent, IgA1 complexed with circulating mucosal secretory galactose-deficient IgA1 (Gd-IgA1) in the mesangium of susceptible individuals, followed by complement activation and resulting glomerular injury. Reported serologic risk factors for the development of IgAN include altered glycosylated (galactose-deficient) IgA1 (Gd-IgA1), high serum IgA levels, and the presence of serum IgG autoantibodies specific for Gd-IgA1.[23-26]

Whether the observed IgAN cases were truly related to COVID-19 infection and associated inflammatory respiratory and intestinal mucosa is unknown because IgAN is one of the most common GNs observed worldwide, independent of COVID-19.

Thrombotic microangiopathy involving thrombotic thrombocytopenic purpura, hemolytic uremic syndrome, or not specified occurred at 7.4% among infected individuals compared with a pre-COVID rate of 2.5%. Whether COVID-19 infection reduces ADAMTS-13 activity due to viral infection-induced production of ADAMTS-13 autoantibodies or predisposes to direct endothelial injury and complement dysregulation that could precipitate TTP and HUS, respectively, among susceptible individuals remains to be elucidated.[27-29] Nonetheless, there is evidence of decreased ADAMTS-13 levels secondary to COVID-19 inflammation.[30,31] A recent systematic review pertaining to COVID-19-associated TTP involving 11 cases characterized the condition as atypical and refractory to routine therapy, where affected patients required longer sessions of plasma exchange therapy and half of the patients received immunosuppressive therapy.[32]

Less commonly reported GNs include conditions that are associated with infectious antigen-driven glomerular injury, such as MN, membranoproliferative pattern of glomerular injury (MPGN), and IgA-dominant infection-related GN.[33] Our data did not reveal significant differences among these GNs compared

with corresponding GNs in the pre-COVID-19 era. Six out of 162 (3.7%) infected cases involved MN, compared with 11% of MN in the pre-COVID era. MN occurrences related to both anti-PLA2R and non-anti-PLA2R were reported.[34] MPGN and IgA-dominant infection-related GN occurrences were low in infected individuals. Other glomerular diseases reported, including AL and AA amyloidosis, anti-glomerular basement membrane, polyarteritis nodosa, myeloma kidney, "extracapillary glomerulonephritis", lupus nephritis, and post-infectious glomerulonephritis, comprised less than 2% of reported cases.

STUDY LIMITATIONS

This is a systemic review based solely on reported cases of GNs associated with COVID-19 infection. Reporting of GN cases may have been biased based on regional practices in terms of performing kidney biopsies in patients with active COVID-19 disease, under-reporting due to a severe shortage of healthcare workforce during the peak of the pandemic, low reporting from developing countries, or a combination of all factors, among others. Additionally, we cannot determine if reported COVID-related cases were truly "related" or just coincidental.

CONCLUSION

The COVID-19 pandemic has claimed the lives of 1,130,662 individuals within the US (Centers for Disease Control (CDC)[35] and over 6.9 million individuals globally.[1] Survivors of COVID-19 infection may further suffer from long-term sequelae such as chronic pulmonary fibrosis and hypoxia, thrombotic and cerebrovascular disease, type 2 diabetes mellitus (T2DM), myalgic encephalomyelitis/chronic fatigue syndrome, dysautonomia, and chronic kidney disease.[2,5] Our systematic review revealed that a great number of infected individuals may have also suffered from associated glomerular diseases other than FSGS. Of interest, the distributive pattern of glomerular diseases reported in association with the COVID-19 infection differs significantly from that reported in the pre-COVID era. Based on our understanding of the pathophysiology of GNs, it is possible that there were COVID-19-dependent effects of the top 3 reported COVID-19-associated GNs.

ETHICAL STATEMENT

I, Phuong-Chi Pham, assure that the following is fulfilled for the manuscript entitled "*COVID-19 associated glomerular diseases*":

- The material presented is the authors' own original work, not previously published elsewhere.
- The paper is not currently being considered for publication elsewhere.
- The paper reflects the authors' own research and analysis in a truthful and complete manner.
- The paper properly credits the meaningful contributions of co-authors.
- All sources used are properly disclosed (correct citation).
- All authors have been personally and actively involved in substantial work leading to the paper and will take public responsibility for its content.

CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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