COVID-19 and KIDNEY DISEASE

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OUTLINE OF KIDNEY INVOLVEMENT IN COVID 19

- Mechanism of SARS COV-2 entry
- COVID associated AKI
  - Pathogenesis
  - Incidence
  - Risk factors
  - Management: RAAS inhibitors in COVID-19, Standard management, pharmacological management, kidney replacement therapy
- Special considerations
  - CKD/ESRD
  - COVID associated glomerular disease
  - COVID -19 vaccine and kidney disease

MECHANISM OF SARS COV-2 ENTRY

- Severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2), the cause of coronavirus disease 2019 (COVID-19), gains entry into target cells through the angiotensin-converting enzyme 2 (ACE2) receptors.

- ACE2 receptors are present in the kidneys as well as the lungs, heart, and intestinal cells

- The renin-angiotensin system plays an important role in human physiology.

- Angiotensin I is cleaved from angiotensinogen by renin and converted to angiotensin II by ACE.

- Angiotensin II causes systemic vasoconstriction and also enhances inflammation, endothelial cell dysfunction, oxidative stress, collagen synthesis in fibroblasts, and fi brosis in target organs.

- ACE2 is a counterregulatory enzyme that breaksdown angiotensin II to form angiotensin 1–7, which mediates vasodilation and attenuates angiotensin II-mediated inflammation

- SARS-CoV-2, downregulates expression of ACE2 after it enters the cell, and without the counterregulatory effects of ACE2, the deleterious effects of angiotensin II are believed to lead to lung disease, including severe acute respiratory distress syndrome.

- The kidney has an abundance of ACE2 receptors and therefore may be one of the primary targets of SARS-CoV-2 infection.

- ACE2 is expressed in the kidney much more than in the lungs, specifically on the brush border apical membrane of the proximal tubule and also at lower levels in the podocytes

COVID-19 ASSOCIATED AKI

INCIDENCE

- In a meta-analysis of approximately 13,000 mostly hospitalized patients, the incidence of AKI was 17 percent, although the range of AKI incidence in the included studies was broad (range 0.5 to 80 percent).

- Approximately 5 percent of patients required kidney replacement therapy.

- The incidence seems to vary by geographic location and proportion of critically ill patients included in each study.

Acute kidney injury (AKI) in patients hospitalized with COVID-19

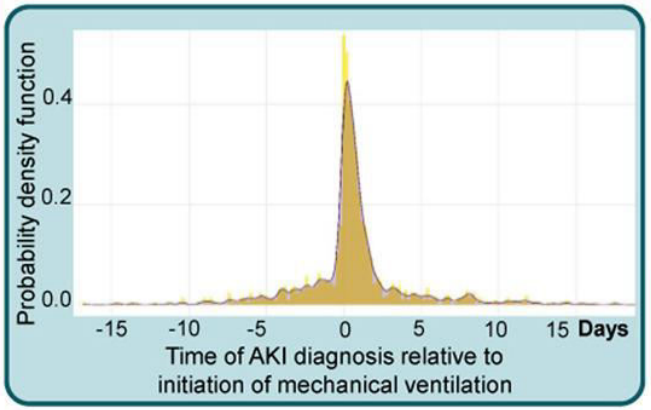
Methods & Cohort

- Retrospective cohort
- 13 hospitals in New York
- SARS-CoV-2 positive
- N = 5449
  Age = 64 (52, 75)
- March 1 – April 5, 2020

Results

All AKI
1,993 (37%)

Stage 1	Stage 2	Stage 3
47%	22%	31%



Independent risk factors for AKI

Older age
Black race
Hypertension
Diabetes mellitus
Cardiovascular disease
Vasopressor use
Need for ventilation*

*OR=10.7
(95%CI 6.8 -16.7)

Disposition of patients with AKI

- Still admitted
  780 (39%)
- Discharged
  519 (26%)
- Died
  694 (35%)

CONCLUSION:

AKI occurs frequently among patients with COVID-19. It occurs early and in temporal association with respiratory failure. AKI in COVID-19 is associated with a poor prognosis.

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Hirsch et al, 2020

RISK FACTORS FOR AKI WITH COVID-19 INFECTION

- Risk stratification is important to tailor monitoring and initiate prevention and/or early treatment strategies

- Among patients with COVID-19, those with AKI are more likely to require vasopressors as well as mechanical ventilation

RISK FACTORS FOR AKI IN COVID 19 PATIENTS

Demographic risk factors

- Older age
- Diabetes mellitus
- Hypertension
- Cardiovascular disease or congestive heart failure
- High body mass index
- Chronic kidney disease
- Genetic risk factors (e.g. APOL1 genotype; ACE2 polymorphisms)
- Immunosuppressed state
- Smoking history

Risk factors for AKI during hospitalization

- Nephrotoxins (medications, contrast exposure)
- Vasopressors
- Ventilation, high positive end-expiratory pressure
- Fluid dynamics (fluid overload or hypovolaemia)

Risk factors for AKI at admission

- Severity of COVID-19
- Degree of viraemia
- Respiratory status
- Non-respiratory organ involvement, e.g. diarrhoea
- Leukocytosis
- Lymphopaenia
- Elevated markers of inflammation, e.g. ferritin, C-reactive protein, D-dimers
- Hypovolaemia/Dehydration
- Rhabdomyolysis
- Medication exposure, e.g. angiotensin-converting-enzyme (ACE) inhibitors and/or angiotensin-receptor blockers (ARBs), statins, nonsteroidal anti-inflammatory drugs (NSAIDs)

PATHOGENESIS OF ACUTE KIDNEY INJURY

- **Acute lung injury** could lead to AKI through-
  - hemodynamic changes and reduced cardiac output with high intrathoracic pressure,
  - inflammatory cytokines that lead to systemic inflammation, and
  - reduction in kidney medullary perfusion due to hypoxemia.

- **Sepsis** due to viral or bacterial infection, is associated with increased risk of AKI
  - Higher levels of inflammatory cytokines including interleukin 6 (IL-6)
  - Maladaptive immune responses leading to microvascular dysfunction, increased vascular permeability, and tissue damage
  - Hypoperfusion affecting the kidney microcirculation.
  - Bacterial co-infections have been reported in severely ill COVID-19 patients, which raises the possibility of sepsis playing a role in AKI in these patients.

- Hemodynamic alterations
  - Cardiorenal syndrome is another possible mechanism of AKI in patients with COVID-19
  - Viral myocarditis and cardiomyopathy with left ventricular dysfunction could lead to decreased perfusion to the kidneys, resulting in AKI.

- Cytotoxic effects leading to tubular and podocyte injury
  - SARS-CoV-2 can potentially injure tubular cells and podocytes, leading to proteinuria, hematuria, and AKI.
  - ACE2 is expressed in high amounts in the proximal tubular cells and podocytes, which may be the site of viral entry in the kidneys
  - However, there is conflicting evidence regarding the possible direct cytopathic effects of SARS-CoV-2 on tubular epithelial cells.

- A few studies from China have reported the presence of corona virus like particles observed under electron microscopy in proximal tubular epithelium and podocytes, along with occasional frank tubular necrosis

- Larsen et al reported that in situ hybridization for SARS-CoV-2 did not detect viral RNA in the kidney

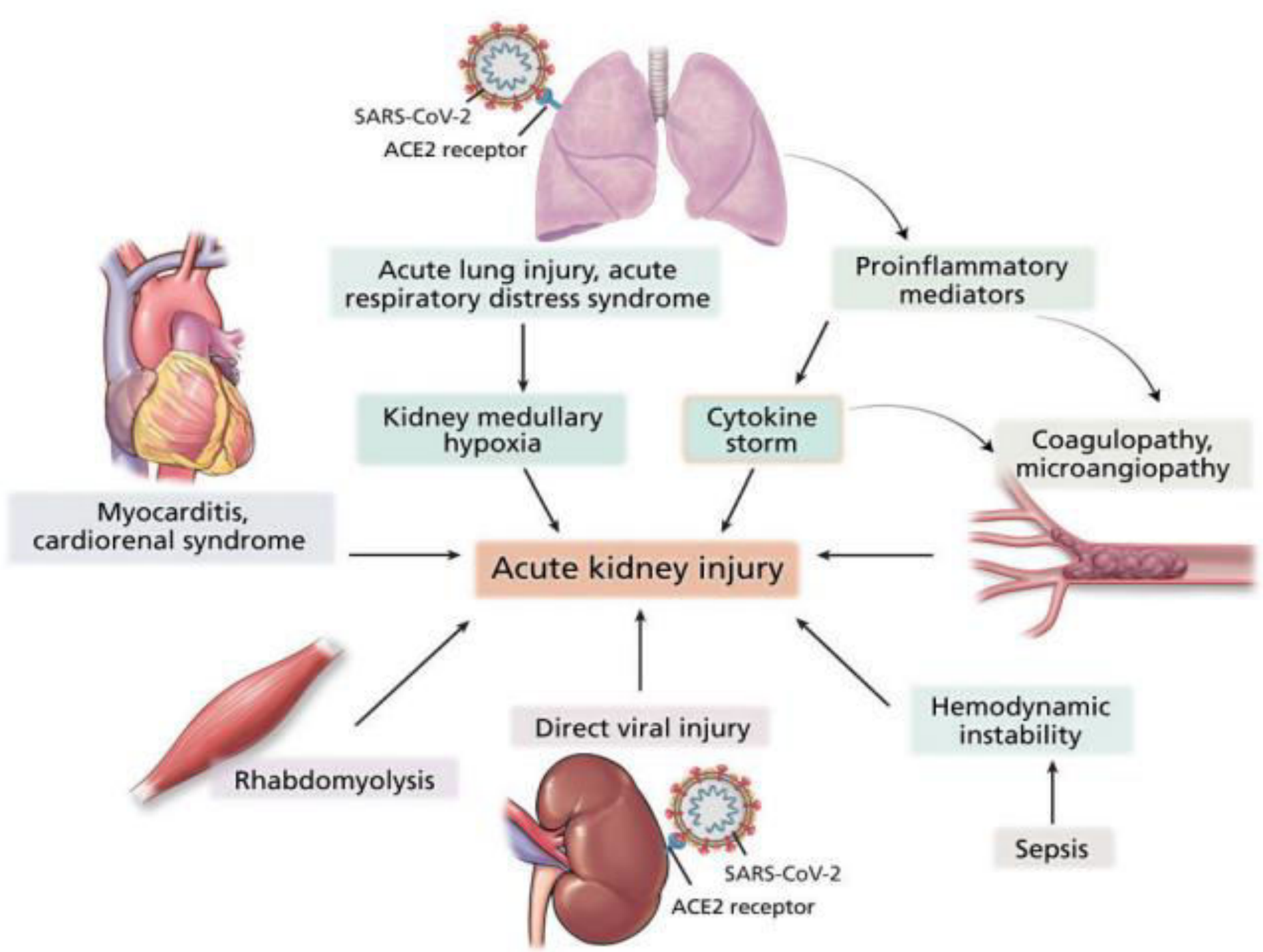
- **Cytokine release syndrome or hyperinflammation:**
  - Viral and bacterial infections are known to cause excessive release of inflammatory cytokines that lead to organ damage
  - As described above, patients with acute lung injury have an increased risk of AKI due to hemodynamic changes, hypoxia, and inflammatory cytokines

- **Rhabdomyolysis**
  - Postmortem kidney histopathologic analysis of COVID-19 patients has shown pigmented casts in the kidney tubules and increased creatine kinase, possibly representing rhabdomyolysis of unclear etiology

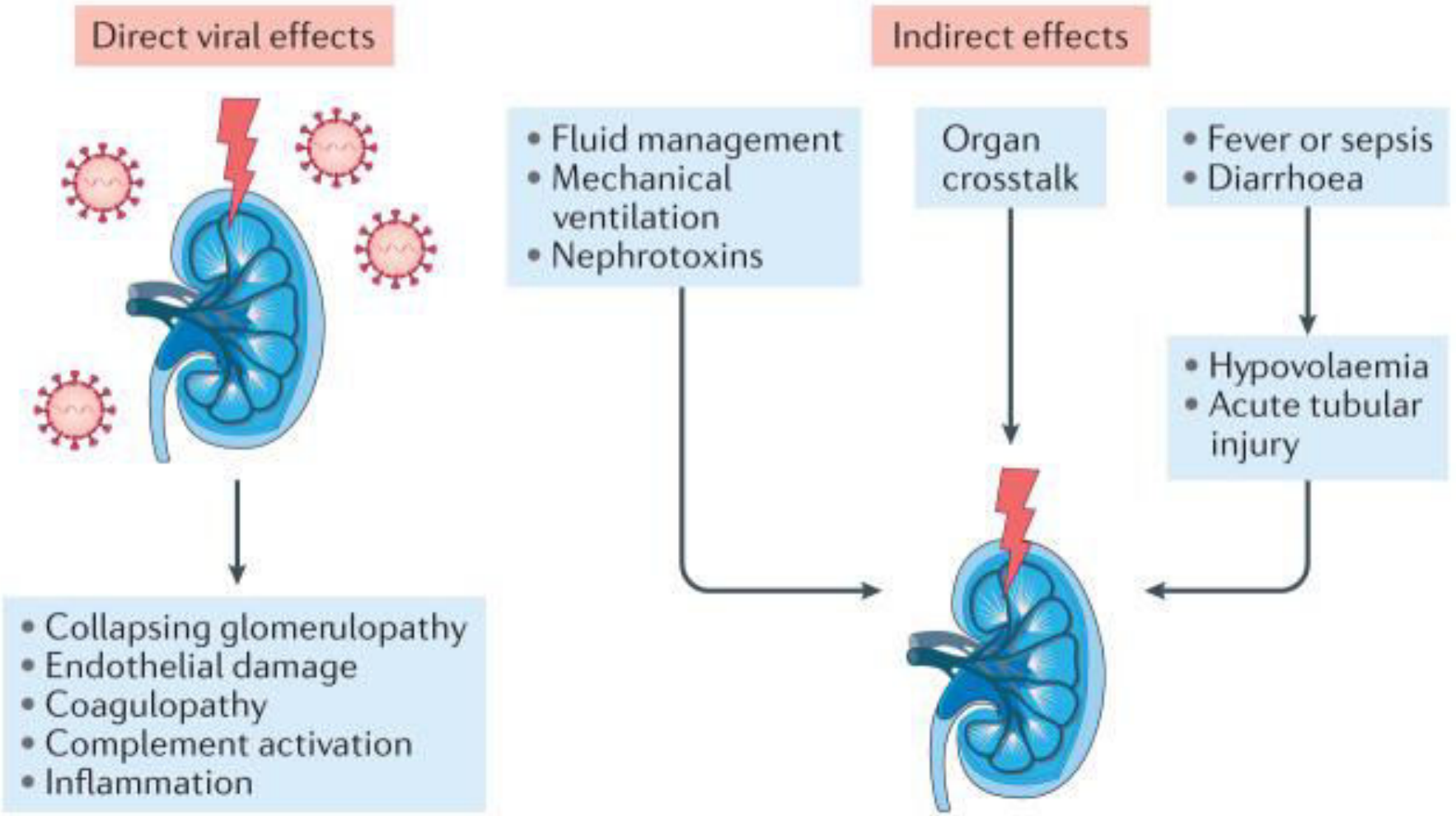
Coagulopathy and microangiopathy

- Coagulopathy has been noted in COVID-19 patients, with altered prothrombin time, activated partial thromboplastin time, D-dimer levels, fibrinogen levels, and fibrin degradation product levels and disseminated intravascular coagulation
- Release of inflammatory mediators and the uninhibited effects of angiotensin II can possibly trigger the coagulation cascade and predispose to hypercoagulability
- Thus, endothelial cell dysfunction leading to activation of the coagulation cascade and thrombosis of the microcirculation may also play a role in AKI

- **Collapsing glomerulopathy**
  - Has been reported in kidney biopsies of patients with COVID-19.
  - Tubuloreticular inclusions have been observed, which can be associated with viral infections.
  - It is hypothesized that either the direct viral effect or presence of increased cytokines from the systemic inflammatory response, or both, can lead to a collapsing variant of focal segmental glomerulosclerosis, especially in patients with high-risk alleles of the APOL1 gene


SARS-CoV-2
ACE2 receptor
Acute lung injury, acute respiratory distress syndrome
Proinflammatory mediators
Kidney medullary hypoxia
Cytokine storm
Coagulopathy, microangiopathy
Myocarditis, cardiorenal syndrome
Acute kidney injury
Rhabdomyolysis
Direct viral injury
Hemodynamic instability
Sepsis
SARS-CoV-2
ACE2 receptor

Mechanism for AKI



MANAGEMENT OF ACUTE KIDNEY INJURY

- Management of patients with a confirmed diagnosis of COVID-19 and AKI begins with an evaluation of the cause of AKI.

- A broad framework of prerenal, renal, and postrenal causes should be considered

- Medications should be carefully reviewed, and any potentially nephrotoxic agents should be discontinued if possible

Cause	Supporting evidence
Prerenal (volume depletion)	Increased blood urea nitrogen: creatinine ratio (> 20), urine sodium < 20 mmol/L, fractional excretion of sodium < 1%
	Urine sediment may show hyaline casts
Acute tubular injury	Urine sodium > 20 mmol/L, fractional excretion of sodium > 1%
	Urine sediment with granular or muddy brown casts
Acute interstitial nephritis	Rash, eosinophilia, white blood cells on urine microscopy
	Urine sediment with white blood cell casts (urine eosinophils are not sensitive or specific)
Postrenal (obstruction)	Bladder scan with high postvoid residual volume, oliguria improving with Foley catheter placement
	Kidney ultrasonography showing hydronephrosis
Rhabdomyolysis	Increased serum creatine kinase and myoglobin in urine
	Positive urine dipstick for blood, no red blood cells on microscopy
Abdominal compartment syndrome	Increased intra-abdominal pressure (> 20 mm Hg)
Coagulopathy	Elevated prothrombin time, partial thromboplastin time, D-dimer, fibrinogen
Cardiorenal syndrome	Jugular venous distention, low ejection fraction on echocardiography, urine sodium < 20 mmol/L

Renin-angiotensin system inhibitors and COVID-19

- Initial concerns were raised regarding a possible association of renin-angiotensin system inhibitors (including ACE inhibitors and angiotensin II receptor blockers) with increased risk of COVID-19, due to possible increased expression of ACE2 based on animal models

- However, the upregulation of ACE2 receptors was noted only in animal studies using various ARBs or ACEIs at doses higher than typically used in humans.

Potential Benefits of RAAS Inhibitors in COVID-19

- Following the initial binding of the S-protein of SARS-CoV-2, there is downregulation of the membrane-bound ACE2
- This downregulation of ACE2 receptor activity in the lungs leads to unopposed accumulation of angiotensin II, the substrate for ACE2
- Accumulated angiotensin II leads to increased neutrophil accumulation, increased vascular permeability, and exacerbated pulmonary edema, and eventually leads to ARDS
- Consequently, by reducing angiotensin II levels, ACEIs and ARBs may protect against lung injury in individuals with COVID-19

- Hence, the decision to discontinue renin-angiotensin system inhibitors should be based on hemodynamic and clinical status, as well as kidney function trend

Optimizing hemodynamic and volume status

- Early volume resuscitation should be initiated in hemodynamically unstable patients to reverse hypoperfusion to vital organs, particularly when patients first present with evidence of volume depletion due to fever and respiratory distress.

- Balance needs to be achieved, avoiding overly aggressive fluid resuscitation and fluid overload

- Active approach with early resuscitation and early termination of fluid resuscitation should be implemented

- Vasopressor therapy is needed to support blood pressure in patients with shock.
- Diuretic therapy should be considered if volume overload is suspected in a hemodynamically stable patient, as in cardiorenal syndrome.
- Volume overload refractory to diuresis requires ultrafiltration, a form of kidney replacement therapy.

Kidney replacement therapy - hemodialysis

- Up to 31% of critically ill patients with COVID-19 require kidney replacement therapy for severe AKI
- The indications for it in COVID-19 patients with AKI are the same as for other AKI patients
- There is no evidence to suggest a benefit for starting it early versus later

- A major challenge during continuous kidney replacement therapy in COVID-19 patients is frequent circuit clotting, thought to be due to upregulation of the coagulation system by inflammatory cytokines

- Circuit clotting seems to improve with heparin use, particularly when administered through the circuit (prefilter)

- A short catheter length with the tip in the superior vena cava can lead to slow flows and catheter dysfunction
- The optimal location of a dialysis catheter to optimize blood flow, which is essential in patients with increased clotting
- Hemodialysis in patients with acute respiratory distress syndrome who require prone positioning needs a coordinated and sequential timing protocol to provide adequate ventilatory support in the prone position and dialysis therapy in the supine position.

Continuous kidney replacement therapy

- Continuous kidney replacement therapy (CKRT) remains preferred among critically ill patients with AKI.

- Even among patients who are hemodynamically stable and could tolerate intermittent hemodialysis (IHD), CKRT or prolonged intermittent kidney replacement therapy should be performed instead, depending upon machine and staffing availability.

Use of extended tubing

- CKRT machines can either be placed inside an isolation room as per standard practice or outside the room with the use of extended tubing.

- Placing the machine outside of the room minimizes the need for repeated entry to troubleshoot and manage the machine, and therefore reduces wastage of personal protective equipment.

- However, extended tubing is a scarce resource.

- In addition, use of extended tubing requires additional tubing connections and increases the likelihood that tubing will become disconnected.

- Extended tubing also decreases the sensitivity of pressure alarms to detect disconnection of the venous lines and potentially increases the risk of clotting due to the longer tubing length

- If CKRT capacity at an institution is overwhelmed, CKRT machines can be used to deliver prolonged intermittent treatments (eg, 10 hours rather than continuous) with higher flow rates (eg, 40 to 50 mL/kg/hour).

- The machine can be rotated between patients every 24 hours or whenever the circuit clots, so that use of new filter sets is minimized.

- This will enable the CKRT machine to become available sooner for care of another patient after terminal cleaning.

Peritoneal dialysis

- Other options emergently starting peritoneal dialysis using bedside tunneled peritoneal dialysis catheter placement.
- Nontunneled acute peritoneal dialysis catheter placement, may be an option in dire situations
- When peritoneal dialysis is used for management of AKI in patients with COVID-19, automated peritoneal dialysis with a cycler should be used, if available. This minimizes the contact between health care personnel and the patient.

Modality	Advantages	Disadvantages
IHD	Widely available Allows treatment of several patients with the same machine in a given day Higher blood flow may reduce risk of clotting	Less effective in reaching daily fluid balance goals Can lead to or exacerbate haemodynamic instability Usually requires a dedicated HD nurse or other staff in addition to an ICU nurse (increasing staff exposure to the isolation environment)
CRRT	Achieves steady-state control of small solutes and acid-base status Least likely to exacerbate haemodynamic instability Easy to achieve net negative fluid balance and achieve fluid balance targets with greater haemodynamic stability Can often be performed by the patient's bedside in the ICU, limiting staff contact with the isolation environment	Not as widely available as other modalities outside of resource-rich settings or tertiary centres Requires one machine per patient per day Requires ICU settings and may require 1:1 nursing ratio depending on institutional policies Given the procoagulable nature of COVID-19, anticoagulation is recommended and may require systemic therapeutic anticoagulation Increased frequency circuit clotting may lead to a lower delivered dose, inability to achieve fluid balance targets and increased resource utilization (which may have supply chain impacts)

Modality	Advantages	Disadvantages
PIRRT: IHD or CRRT	Less likely than other modalities to exacerbate haemodynamic instability Allows treatment of several patients with the same machine in a given day Option for higher blood flow, which may reduce risk of circuit clotting	Not as widely available as other modalities (i.e. hospital protocols are not widely established) Given the procoagulant nature of COVID-19, systemic anticoagulation may be necessary Challenges and uncertainty of drug dosing, especially for antimicrobial and/or COVID-19 therapeutics

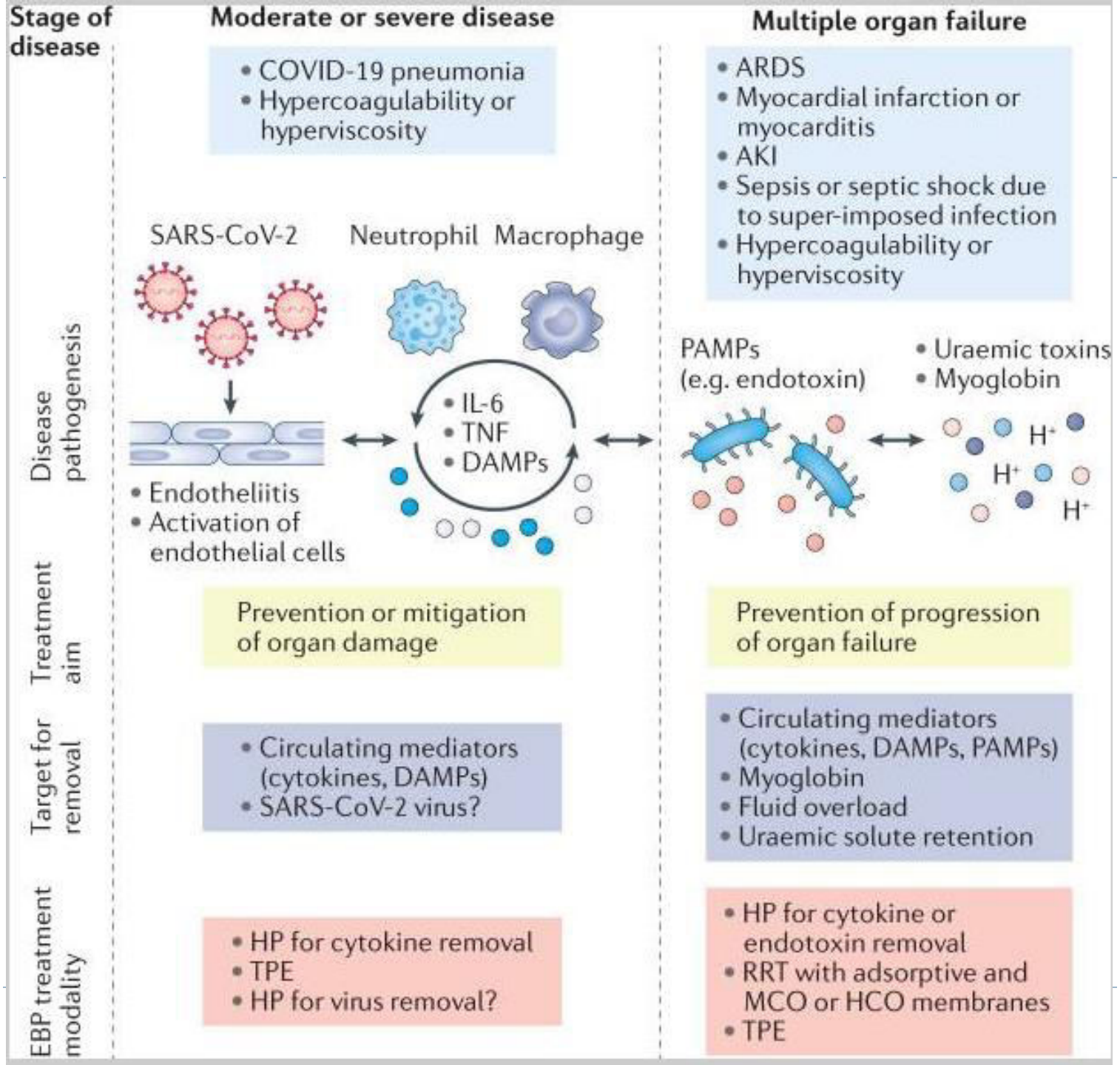
Modality	Advantages	Disadvantages
PD	Widely available No circuit clotting concerns No venous access required Less likely to exacerbate haemodynamic instability Less nursing exposure with the use of automated cycler	May be more challenging in patients in prone positions Risk of peri-catheter leaks Protocols and policies for acute PD are not available at all sites Requires technical expertise to place catheters May require rapid implementation of training regimen for renal nurses and clinicians

Extracorporeal blood purification

Biological rationale:

- Inflammatory cytokines, DAMPs, pathogen-associated molecular patterns(PAMPs), including endotoxins and SARS-CoV-2 particles, potentially contribute to the development of multiple organ failure and mortality in critically ill patients with COVID-19.

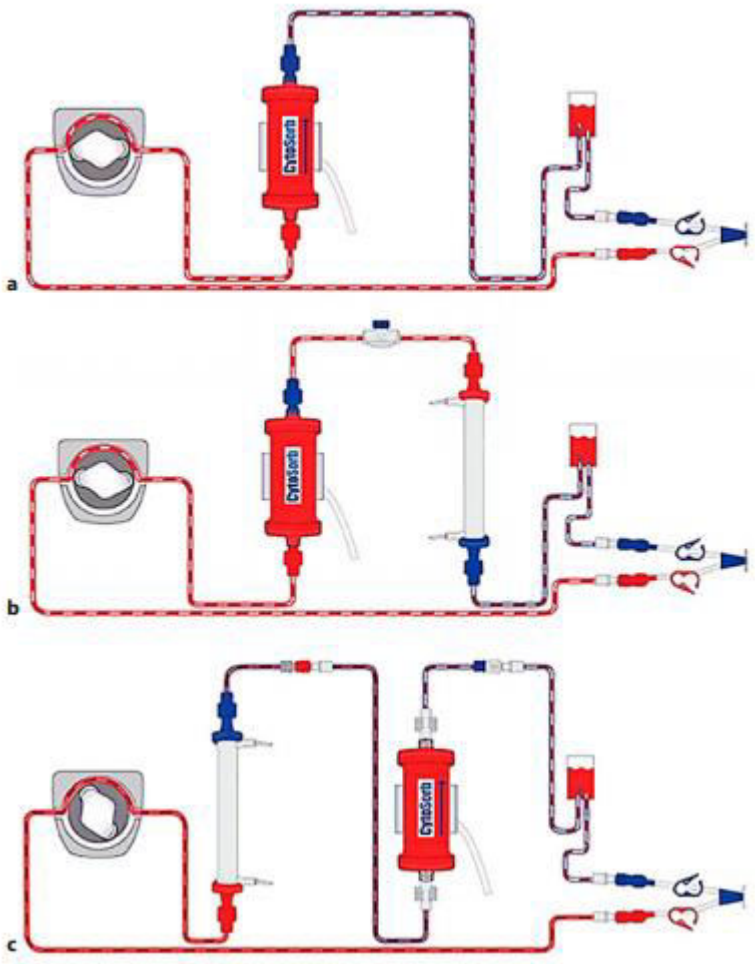
- EBP techniques have been shown to remove cytokines, DAMPs and PAMPs, including endotoxins and circulating viral particles


Stage of disease
Moderate or severe disease
Multiple organ failure
• COVID-19 pneumonia
• Hypercoagulability or hyperviscosity
• ARDS
• Myocardial infarction or myocarditis
• AKI
• Sepsis or septic shock due to super-imposed infection
• Hypercoagulability or hyperviscosity
Disease pathogenesis
SARS-CoV-2
Neutrophil
Macrophage
• IL-6
• TNF
• DAMPs
• Endotheliitis
• Activation of endothelial cells
PAMPs (e.g. endotoxin)
• Uraemic toxins
• Myoglobin
H+
H+
H+
Treatment aim
Prevention or mitigation of organ damage
Prevention of progression of organ failure
Target for removal
• Circulating mediators (cytokines, DAMPs)
• SARS-CoV-2 virus?
• Circulating mediators (cytokines, DAMPs, PAMPs)
• Myoglobin
• Fluid overload
• Uraemic solute retention
EBP treatment modality
• HP for cytokine removal
• TPE
• HP for virus removal?
• HP for cytokine or endotoxin removal
• RRT with adsorptive and MCO or HCO membranes
• TPE

- Haemoperfusion techniques can remove inflammatory molecules, DAMPs and PAMPs, including SARS-CoV-2 particles.

- There are many extracorporeal techniques like oxiris, cytosorb and high cutoff membranes

- The oxiris membrane (Baxter, IL - USA) is a highly biocompatible heparin-coated hemodiafilter which can be used for unselective removal of cytokines and endotoxin. It also reduces clotting during treatment.



- CytoSorb is a hemoadsorption column able to remove inflammatory mediators from the blood. It contains highly absorbent coated beads, coated with polyvinylpyrrolidone.
- It can be configured as standalone, or added to the extracorporeal circuit, pre-dialyzer, or post-dialyzer

- Therapeutic plasma exchange (TPE) can remove inflammatory mediators and proteins associated with hypercoagulability.

- CRRT with surface-modified AN69 or polymethylmethacrylate membranes can remove target molecules by adsorption, whereas CRRT with medium cut-off or high cut-off membranes can remove target molecules by diffusion or convection.

- No consensus exists on the use or thresholds of specific biological and clinical criteria for initiating, monitoring or discontinuing EBP in critically ill patients with COVID-19

Follow up

- Patients with COVID-19 AKI be followed over a period of 2–3 months post-discharge, depending on the severity and acute needs of the patient, to evaluate kidney recovery

SPECIAL CONSIDERATIONS

CHRONIC KIDNEY DISEASE

- In a meta-analysis of four studies and 1389 infected patients (including 273 patients with severe disease), the prevalence of underlying CKD was more frequent among those with severe disease (3.3 versus 0.4 percent; odds ratio 3.03, 95% CI 1.09-8.47)

- **Dialysis access planning in advanced CKD** — Patients with stage 4 or 5 CKD who are referred for dialysis access placement should undergo these procedures as planned (and not have their planned procedure deferred)

COVID-19 ASSOCIATED GLOMERULAR DISEASE

- Glomerular lesions were reported in a minority of patients with COVID-19, with collapsing focal segmental glomerulosclerosis (FSGS), also called COVID-associated nephropathy (COVAN), being the most common.

- Such patients present with nephrotic-range proteinuria and acute kidney injury (AKI)

- Thrombotic microangiopathy is another uncommon finding among patients who develop AKI and nephrotic-range proteinuria

- There are case reports of other glomerular diseases associated with COVID-19, including antineutrophil cytoplasmic antibody (ANCA)-associated vasculitis, anti-glomerular basement membrane antibody disease, and IgA nephropathy

Modifications to the management of pre-existing glomerular disease

- Patients with glomerular disease who are treated with immunosuppressive therapies may be at a heightened risk of infections (COVID-19 or other infections).

- However, there are no rigorous studies to guide pandemic-related modifications to the treatment of glomerular disease

- Patients at **low risk of acquiring COVID-19:**
- Ability to self-isolate (eg, ability to work from home and have limited or no interaction with outsiders)
- Continue with their previously planned treatment for their glomerular disease
- Reinforce the risks and benefits of undergoing immunosuppressive therapy in the context of the pandemic to ensure adherence to strict isolation while on treatment

- Patients **at risk of acquiring COVID-19** due to regional transmission rates and other factors (eg, inability to quarantine because of their occupation):

- If the patient's glomerular disease is being treated with calcineurin inhibitors or hydroxychloroquine, no treatment modification is necessary

- Patients not yet on immunosuppressive therapy, postpone treatment in following groups:
  - membranous nephropathy who have uncomplicated nephrotic syndrome and preserved eGFR
  - IgA nephropathy who do not have features associated with a high risk of progression (eg, heavy proteinuria, impaired eGFR, or crescents on histopathology)
  - glomerular diseases for which it is unclear that immunosuppressive therapy is beneficial (eg, infection-related glomerular disease)

- For patients who were initiated on immunosuppressive therapy before the pandemic and who are not yet in remission:
  - arrangements should be made for administration of necessary intravenous (IV) infusions at home rather than in an institutional infusion center.
  - When possible, IV infusions should be changed to equivalent oral alternatives

- For patients who were initiated on immunosuppressive therapy before the pandemic and who are already in remission, lower the doses of their immunosuppressive medication to a minimum level that will maintain remission

- For patients with glomerular disease who are receiving immunosuppressive therapy that includes antimetabolites and who have suspected or confirmed COVID-19, discontinue antimetabolites for 7 to 10 days after symptom onset.

COVID-19 VACCINES AND KIDNEY DISEASE

- Patients with kidney diseases should be prioritized for COVID-19 vaccination and the available data suggest that replication-defective viral-vectored vaccines and mRNA vaccines are safe to use.

- As vaccine responses are likely to be lower in patients with kidney diseases than in the general population, highly potent vaccines should be preferred.

- As patients with kidney disease commonly have compromised immune systems, live replicating microbial-vectored vaccines should be avoided.

- However, Covaxin (Bharat biotech) which is inactivated virus-based COVID-19 vaccine, replication-defective viral-vectored vaccines such as ChAdOx1 nCoV-19 (Covishield; Oxford-AstraZeneca), Gam-COVID-Vac (Sputnik V) and the mRNA vaccines BNT162b2 (Pfizer-BioNTech) and mRNA-1273 (Moderna) are safe to use.

- Compared with inactivated vaccines, both mRNA vaccines and viral-vectored vaccines have the advantage of inducing balanced humoral and T cell immunity.

- A potent CD8+ T cell response is usually induced by viral-vectored vaccines and is expected for mRNA vaccines

- In patients with no or weak induction of seroconversion and/or T cell immunity after vaccination, theoretical options include an additional booster dose, or respiratory mucosal vaccination.

- Mucosal vaccination induces strong immunological memory mediated by tissue-resident innate and adaptive immune cells

- Mucosal vaccination might therefore be an effective vaccine strategy for patients who are immune compromised.

- **Patients receiving immunosuppression**
- Patients with autoimmune kidney diseases on chronic immunosuppression were excluded from all major trials of COVID-19 vaccine candidates

- Timing of vaccination and vaccine readiness is relevant in this regard, particularly in patients receiving treatment with anti-CD20 therapy (e.g. rituximab), which is known to abrogate immune responses to vaccinations

- In patients with active autoimmune disease, treating this disease should take priority and vaccination should be delayed

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LETTER TO THE EDITOR | VOLUME 99, ISSUE 6, P1487, JUNE 01, 2021

Gross hematuria following vaccination for severe acute respiratory syndrome coronavirus 2 in 2 patients with IgA nephropathy

Lavinia Negrea • Brad H. Rovin

Published: March 23, 2021 • DOI: https://doi.org/10.1016/j.kint.2021.03.002 • Check for updates

Patient no.	Age, yr	Sex	Race	Year IgAN diagnosed	Treatment	Gross hematuria events during disease course	Persistent microscopic hematuria	Proteinuria in 2020, g/d	Proteinuria between SARS-Cov-2 vaccine doses, g/d	Proteinuria 3 weeks after last SARS-CoV-2 vaccine dose, g/d
1	38	F	W	2005	RAASi	At presentation; during 1 episode of gastroenteritis; occasionally after yearly influenza vaccine	Yes	0.63	0.82	1.40
2	38	F	W	2019	Cyc + Pred (6 mo), then RAASi	At presentation only	Yes	0.43	0.59	0.40


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Is COVID-19 vaccination unmasking glomerulonephritis?
Hui Zhuan Tan • Ru Yu Tan • Jason Chon Jun Choo • ... Carolyn Shan-Yeu Tien • Puay Hoon Tan • Keng Thye Woo • Show all authors
Published: May 21, 2021 • DOI: https://doi.org/10.1016/j.kint.2021.05.009

- Previously healthy individuals who presented with macroscopic hematuria shortly after COVID-19 vaccination and were diagnosed with IgAN and crescentic glomerulonephritis, respectively.

SUMMARY

- AKI is common in COVID-19 and is associated with poor outcomes.
- The kidney may be a primary target for SARS-CoV-2 infection owing to its abundance of ACE2 receptors
- SARS-CoV-2 can damage the kidney through several mechanisms, including acute lung injury, sepsis, hemodynamic alterations, cytotoxic effects, cytokine release syndrome, rhabdomyolysis, coagulopathy, microangiopathy, and collapsing glomerulopathy.
- Patients with acute lung injury are at risk of AKI due to hemodynamic changes, hypoxia, and inflammatory cytokines

- Glomerular disease presenting as proteinuria with or without AKI is an important presentation of COVID-19 infection and may be associated with a high-risk APOL1 genotype

- Patients with kidney diseases should be prioritized for COVID-19 vaccination

- Replication-defective viral-vectored vaccines and mRNA vaccines are safe to use

THANK YOU