

# BIBLIOGRAPHIE RECHERCHE COVID 19

## NEPHROLOGIE

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JOURNAL AUTEUR	TITRE	PRINCIPALE QUESTION	POINTS CLES
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<p>Bo Diao MedRxiv 4th March 2020</p>	<p>Human Kidney is a Target for Novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Infection</p>	<p>Is the kidney a target of SARS Cov2 ?</p> <p><b>Retrospective</b> analysis of eGFR and other clinical parameters from <b>85 patients</b>. Kidney tissues from <b>six patients with post mortem examinations</b></p>	<ul style="list-style-type: none"> <li>- <b>27.06%</b> (23/85) patients exhibited <b>acute renal failure</b> (ARF). The elderly patients and those with comorbidities such as hypertension and heart failure more developed ARF more frequently (65.22% vs 24.19%, <math>p &lt; 0.001</math>; 69.57% vs 11.29%, <math>p &lt; 0.001</math>, respectively).</li> <li>- H&amp;E staining demonstrated kidney tissues from postmortems have <b>severe acute tubular necrosis, luminal brush border sloughing and vacuole degeneration, and lymphocyte infiltration</b>. Dilated capillary vessels were observed in the glomeruli of these 6 cases</li> <li>- Immunohistochemistry showed that <b>SARS-CoV-2 NP antigen was accumulated in kidney tubules</b>.</li> <li>- Viral infection not only induces <b>CD68+ macrophages infiltrated</b> into tubulointerstitium, but also enhances <b>complement C5b-9 deposition</b> on tubules.</li> </ul>
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<p>Hua Fan medRxiv 9th March 2020</p>	<p>Retrospective Analysis of Clinical Features in 101 Death Cases with COVID-19</p>	<p>Organ failure in death due to SARS-CoV 2</p>	<p>- Liver and <b>kidney damages</b> were not significant at the time of admission and at ICU admission, but <b>a significant deterioration occurred 48h before death.</b></p> <p>At admission / ICU admission / 48h before death</p> <p><b>Serum creatinine</b></p> <p>74.40(64-94.30) / 74.95(61.80-101.43) / <b>173.70(96.60-350.70)</b></p>
<p>Qiao Shi medRxiv 4th March 2020</p>	<p>Clinical characteristics of 101 non-surviving hospitalized patients with COVID-19—A single center, retrospective study</p>	<p>Organ failure and clinical characteristics in death from SARS-CoV 2.</p> <p>Comparison between death within 3 days or after 3 days in hospital</p>	<p>- <b>Retrospective study on 101 patients with comorbidities</b> including hypertension (58.42%), cardiovascular disease (23.76%), diabetes (21.78%), chronic pulmonary disease (13.86%), cerebrovascular disease (12.87%), chronic kidney disease (10.89%) and malignancy (6.93%)</p> <p>- <b>acute kidney injury occurred in 23 (22.77%)</b> of patients</p> <p>- elevated C-reactive protein (123 ± 10 vs 94 ± 9 mg/L), procalcitonin (2.26 vs 0.58 ng/ml), hsTnI (1.98 vs 0.2 ng/ml), Creatine kinase-MB (6.14 vs 2.78 ng/ml), myoglobin (437.7 vs 216.8 ug/L) and <b>BUN (15.2 vs 10.08 mmol/L)</b> were over normal range and <b>significantly higher in patients died within 3 days of admission</b></p>

<p>Bicheng Zhang medRxiv 26th February 2020</p>	<p>Clinical characteristics of 82 death cases with COVID-19</p>	<p>Organ failure in death due to SARS-CoV 2</p>	<ul style="list-style-type: none"> <li>- Retrospective study of 82 death cases. Median time from initial symptom to death : 15 days (11-20)</li> <li>- <b>comorbidity (76.8%)</b>, including hypertension (56.1%), heart disease (20.7%), diabetes (18.3%), cerebrovascular disease (12.2%), and cancer (7.3%).</li> <li>- Respiratory failure was the leading <b>cause of death</b> (69.5%), following by sepsis syndrome/MOF (28.0%), cardiac failure (14.6%), hemorrhage (6.1%), and <b>renal failure (3.7%)</b>.</li> <li>- respiratory, cardiac, hemorrhage, hepatic, and <b>renal damage</b> were found in 100%, 89%, 80.5%, 78.0%, and <b>31.7% of patients</b></li> <li>- Creatinine &gt;133µmol/L ; 11/72 cases (15.3%)</li> </ul>
<p>Anti-2019- nCoV Volunteers, Zhen Li et al medRxiv February 12nd, 2020</p>	<p>Caution on Kidney Dysfunctions of 2019- nCoV Patients</p>	<p>Kidney function among patients infected by SARS-CoV 2</p>	<p>63% (32/51) of the patients exhibited proteinuria, by dipstick analysis</p> <p>9% (11/59) of the patients had increased plasma creatinine (&gt;200 µmol/L in all 3 deceased patients )</p>

<p>Yichun Cheung</p> <p>Kidney Int</p> <p>16th March, 2020</p>	<p>Kidney disease is associated with in-hospital death of patients with COVID-19</p>	<p>Prevalence and prognostic value of kidney involvement in SARS-CoV 2 infection</p>	<p>Prospective cohort study of 701 patients with COVID-19 admitted in a tertiary teaching hospital</p> <p><b>On admission</b>, 43.9% of patients had proteinuria and 26.7% had hematuria. The prevalence of elevated serum creatinine, and eGFR&lt;60 ml/min/1.73m<sup>2</sup> were 14.4 and 13.1%, respectively</p> <p><b>During the study period</b>, AKI occurred in 5.1% patients</p> <p>The incidence of <b>in-hospital death</b> in the patients with elevated baseline serum creatinine was 33.7%, which was significantly higher than in those with normal baseline serum creatinine (13.2%)</p> <p>After adjustment (for age, sex, disease severity, comorbidities and lymphocyte count), proteinuria, hematuria, elevated baseline serum creatinine, peak serum creatinine &gt; 133μmol/L, and AKI over stage 2 were all associated with in-hospital death</p>
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<p>Bo Diao medRxiv</p> <p><a href="https://doi.org/10.1101/2020.03.04.20031120">https://doi.org/10.1101/2020.03.04.20031120</a></p>	<p><a href="#">Human Kidney is a Target for Novel Severe Acute Respiratory Syndrome Coronavirus 2 (SARS-CoV-2) Infection</a></p>	<p>Apart from the respiratory system, it is unclear whether SARS-CoV-2 can also directly infect other tissues such as the kidney or induce acute renal failure.</p>	<p>6 patients autopsies</p> <p>Necrose tubulaire et infiltrat lymphocytaire, sans anomalie glomérulaire ou vasculaire majeure</p> <p>déposition de complément mais uniquement sur les tubules</p> <p>pas d'explication évidente aux protéinuries glomérulaires majeures constatées en clinique</p> <p>Comments:</p> <ol style="list-style-type: none"> <li>1. Is there any relationship between renal dysfunction and respiratory function? No statistical study or not shown.</li> <li>2. Is the primary antibody against viral NP specific? Probably since negative tissue controls have been used and are negative. <b>The signal in kidney tissues (and lung as positive control) support the presence of the virus within the renal tissue:</b> but it does not demonstrate definitively that it is locally (in situ) pathogenic → limitation of the message.</li> <li>3. Tissue studies from most severe cases leading to death, so with previous severe hypoxemia, shock, ... Are the lesions specific or secondary to severe hypoxia and</li> </ol>
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