

Acute cortical necrosis in transplant recipient
with Covid-19 infection

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INTRODUCTION

- Kidney transplant recipients are considered a high-risk population for severe SARS-CoV-2 infection due to coexisting comorbid diseases and permanent immunosuppression.
- The common renal biopsy findings of renal transplant patients infected with Covid19 are acute tubular necrosis and acute rejection.
- Here we report a case of acute cortical necrosis in a Covid-19 positive renal transplant patient.

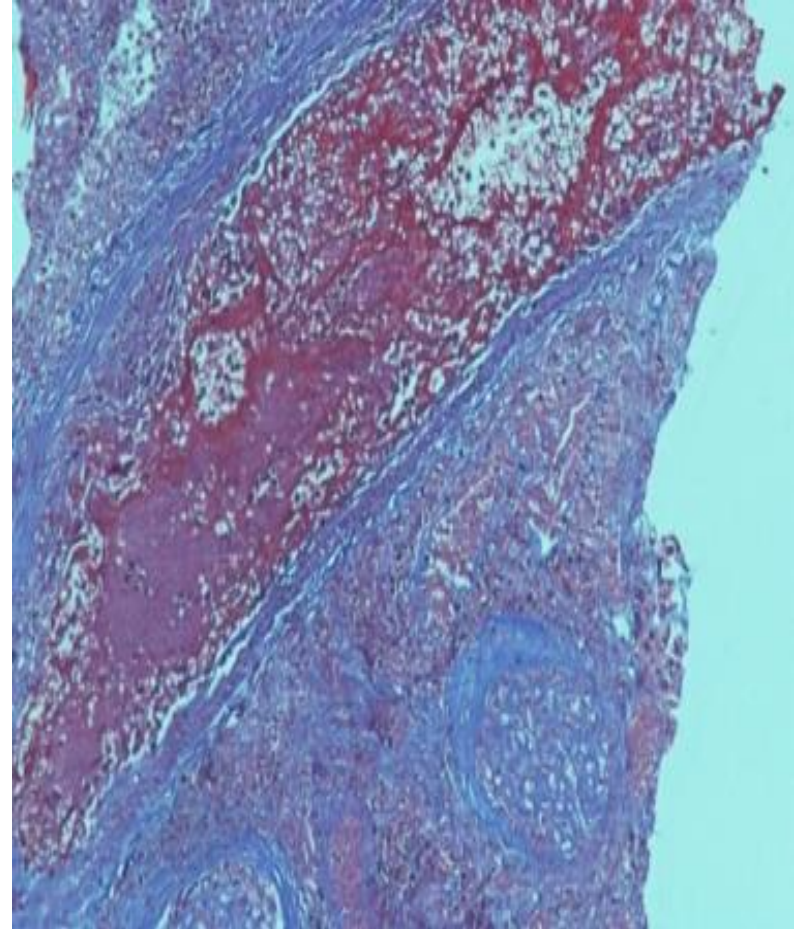
CASE REPORT

- A 47 year male ABO compatible renal transplant patient with good graft function (baseline creatinine 0.9mg/dl) for 17 years of duration got admitted with Covid19 infection(CTscore-16/25).
- He was on triple immunosuppressive drugs (Prednisolone 5mg OD, Cyclosporine 50mg 1-0-1/2, Azathioprine 100mg OD).
- On admission S.creatinine was 2.7 mg/dl and his inflammatory profile (CRP,d-Dimer and Ferritin) was elevated.
- LDH & peripheral smear -normal

- Cyclosporine level was 79.5 ng/ml.
- CMV PCR was negative.
- He received
 1. Inj.Methyprednisolone 125mg iv for 3 days
 2. 5 doses of Inj.Remedesvir and
 3. Inj Heparin 5000 units IV TDS.
- Tab.Azathiopurine,Tab Prednisolone was stopped
- Tab.Cyclosporine was continued(same dose)

- Next day he became oliguric (creatinine -6mg/dl) and was initiated on hemodialysis.
- There was no evidence of septic or ischemic ATN.
- Allograft doppler was normal.
- He underwent renal biopsy after 2 HD session.

95% of biopsied tissue showing evidence of renal cortical necrosis with medium vessel showing organising thrombus



- After 10 days ,he was discharged with oral anticoagulant (Tab.Apixiban 2.5 mg 1-0-0) and a urine output 200ml/day and
- His immunosuppression was
 1. Tab. Prednisolone 10mg 1-0-0
 2. Tab. Cyclosporine 50mg 1-0-1/2.
- He remained dialysis dependent for a month.
- As creatinine showed downward trend he was off dialysis and presently he has a creatinine of 2.7mg/dl

DISCUSSION

- The causes of AKI in patients with COVID-19 are multifactorial.
- Potential mechanisms include indirect renal damage by virus-induced cytokine release, direct cytotoxic effects on renal tubules and endothelium, angiotensin II pathway activation, fluid balance disturbances (prerenal AKI), cardiorenal syndrome, and drug nephrotoxicity.
- Studies describe endothelial activation by SARS-CoV-2 providing an explanation for vascular complications and thromboembolism.

CONCLUSION

- Covid infection is a pro-thrombotic state and our patient developed ACN inspite on adequate anticoagulants.
- Cortical necrosis should be considered as a possible etiology for Acute graft dysfunction with long term graft deterioration in covid-19 transplant patients.

REFERENCES

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