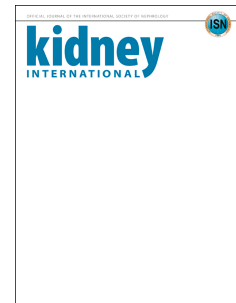


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COVID-19 infection in kidney transplant recipients

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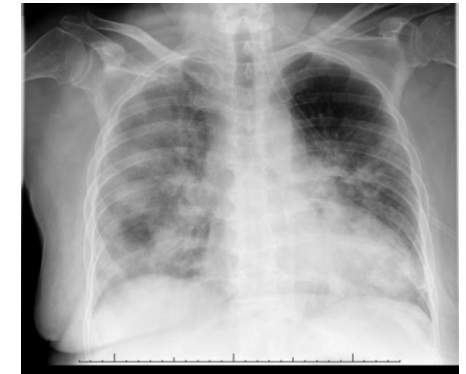
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COVID-19 infection in kidney transplant recipients

SARS-CoV-2 a virus similar to SARS and MERS is causing a pandemic. We report 7 cases of COVID 19 in kidney transplant recipients from South London , UK

- All presented with fever & respiratory symptoms
- 2 patients were within 3 months of transplant
- Most treated with supportive care and reduced immunosuppression
- 4 needed ITU admission, 1 died
- High D dimer, ferritin, troponin levels and lymphopenia are seen in severe cases, are likely to be of prognostic value
- Extra pulmonary involvement contributes to mortality

Case	Transplant Date	Diabetes	Immuno suppression	Outcome
 48y	1989		Pred,AZA	Home
 67y	03/19	Diabetes	Pred,Tac,MMF	ITU, died
 54y	12/19	Diabetes	Pred,Tac,MMF	ITU
 65y	08/18		Pred,Tac,MMF	ITU, ward
 69y	02/20	Diabetes	Pred,Tac,MMF	ITU, ward
 54y	05/13		Tac,MMF	Home
 45y	09/17		Pred,Tac,Aza	Ward



Chest Xray for case 5

CONCLUSION:

COVID 19 infection in kidney transplant patients can cause severe illness, requiring ITU admission with high rate of AKI

Prompt reduction in immunosuppression is required in severe cases

Older and diabetic patients may be at higher risk

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Title: COVID-19 infection in kidney transplant recipients

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Abstract

By 21 March 2020 infections related to the novel coronavirus SARS-CoV-2 had affected people from 177 countries and caused 11,252 reported deaths worldwide. Little is known about risk, presentation and outcomes of SARS-CoV-2 (COVID-19) infection in kidney transplantation recipients, who may be at high-risk due to long-term immunosuppression, comorbidity and residual chronic kidney disease. Whilst COVID-19 is predominantly a respiratory disease, in severe cases it can cause kidney and multi-organ failure. It is unknown if immunocompromised hosts are at higher risk of more severe systemic disease. Therefore, we report on seven cases of COVID-19 in kidney transplant recipients (median age 54 (range 45-69), three females, from a cohort of 2082 managed transplant follow-up patients) over a six-week period in three south London hospitals. Two of 32 patients presented within three months of transplantation. Overall, two were managed on an out-patient basis, but the remaining five required hospital admission, four in intensive care units. All patients displayed respiratory symptoms and fever. Other common clinical features included hypoxia, chest crepitation, lymphopenia and high C-reactive protein. Very high D dimer, ferritin and troponin levels occurred in severe cases and likely prognostic. Immunosuppression was modified in six of seven patients. Three patients with severe disease were diabetic. During a three week follow up one patient recovered, and one patient died. Thus, our findings suggest COVID-19 infection in kidney transplant patients may be severe, requiring intensive care admission. The symptoms are predominantly respiratory and associated with fever. Most patients had their immunosuppression reduced and were treated with supportive therapy

Key words: Kidney Transplantation, SARS-CoV-2 infection, COVID 19, immunosuppression

Introduction

The novel corona virus (2019-nCoV or COVID 19) infection, which originated in the city of Wuhan, in Hubei province, China in December 2019 shares close similarities in its genomic structure with the virus (SARS CoV) that caused the SARS global pandemic in 2003 and the MERS epidemic in 2012 (MERS CoV), and even closer similarities to bat SARS-like betacoronavirus (bat-SL-CoVZC45 betacoronavirus) and bat-SL-CoVZXC21. (1, 2)

Between 31 December 2019 and 27 March 2020 532,692 COVID-19 cases and 24,077 deaths worldwide have been identified as being caused by a newly identified enveloped RNA virus named SARS-CoV-2. (3) In UK between 31 January 2020 and 20 March 2020 3,983 cases were identified with 177 (4% of tested patients) deaths. (4) Due to widespread nature, COVID 19 was declared as a pandemic by World Health Organisation on 11 March 2020, and 176 countries are affected as of 27 March 2020 (3).

The SARS pandemic was reported to affect both paediatric and adult kidney transplant recipients in Hong Kong, with less severe disease in the paediatric population. (5) One liver transplant patient died with the SARS-Cov infection in 2003. (6) The MERS coronavirus infection had a variable impact on kidney transplant recipients. In one report of two kidney transplant patients, one died of progressive respiratory disease and acute kidney injury while the other survived. (7). To the best of our knowledge, only one patient with kidney transplantation has been reported in the literature who suffered from COVID 19 infection in Wuhan China and improved 13 days after hospital admission. (8) The 63-year-old kidney transplant recipient presented with fever, chest pain, cough, low lymphocyte, high serum C-reactive protein (CRP), and abnormal chest CT scan on 2 February 2020. Tacrolimus and mycophenolate administration was discontinued. He was treated with oxygen,

methyl prednisolone, umifenovir, moxifloxacin, biapenem, intravenous immunoglobulin, inhaled interferon α and pantoprazole. He made a successful recovery and was discharged on day 13.

We report here the first seven cases of COVID-19 in kidney transplant recipients in south London hospitals.

Cases

We have seen seven cases of kidney transplant recipients with proven COVID-19 infection in south London in March 2020. These patients are described below and main characteristics summarised in Tables 1 and 2.

1. A 48-year-old man with deceased donor kidney transplant in 1989 with failing transplant kidney, (eGFR 15-18 ml/min/1.73m²) called in NHS (111) helpline in the first week of March 2020 with cough, fever and mild shortness of breath. He tested COVID-19 positive by nose and throat swabs taken on March 2. As he was clinically well, he was asked to stay at home and self-isolate. His immunosuppression was azathioprine 75 mg OD and prednisolone 5 mg OD which was not changed. He was not on ACEI/ARB at the time of presentation. He has made a full recovery. The transplant kidney function remained stable.

2. A 67-year-old woman with insulin-dependent type 2 diabetes and end stage kidney disease (ESKD) on haemodialysis therapy for four years received a deceased donor kidney transplant (DBD donor) in March 2019. Her estimated glomerular filtration rate (eGFR) was 45-55 ml/min/1.73m². She was maintained on Tacrolimus with levels of between 5-8 ng/ml, Mycophenolate mofetil (MMF) 250 mg BD and prednisolone 5 mg OD. Her other medications included Ramipril, Aspirin, Alfacalcidol and Amiloride. She presented on 5th March with cough, fever and shortness of breath. Chest X-ray revealed bilateral patchy consolidation (Fig 1A). SARS-CoV-2 RNA PCR tests from nose and throat viral swabs were positive. Bronchial washing for pneumocystis PCR was negative, as was blood PCR for CMV DNA. There was no other positive microbiological diagnosis. She was hypoxic with peripheral oxygen saturation of 86% and a respiratory rate of 26/min, so she was transferred to intensive therapy unit (ITU) and commenced non-invasive ventilation (CPAP for type 1 respiratory failure) and subsequent intubation and ventilation as her clinical condition deteriorated. Serum CRP on admission was 83 mg/l, haemoglobin 110 g/l, with normal total white cell count and mild lymphopenia (lymphocyte count 0.8x10⁹/L). She was treated with broad spectrum antibiotics. No

specific antiviral drugs were given. Mycophenolate mofetil (MMF) was ceased. Low dose tacrolimus was initially continued but stopped one day before death. On day 3 post admission, she developed AKI, with a serum creatinine increase to 225 $\mu\text{mol/L}$. She remained stable on the ventilator with reducing oxygen requirements and improvement in lung infiltrates on chest X-ray (Fig 1B) but deteriorated markedly on 16th March with high serum lactate and LDH levels and an acute rise of CRP to 190. She developed severe metabolic acidosis resistant to correction on CVVHDF, probably owing to an intra-abdominal event (bowel infarction/intra-abdominal sepsis). She deteriorated rapidly and died on 17th March .

3. A 54-year-old female with a history of adult polycystic kidney disease, ESKD in 2012, on haemodialysis for 7 years, received a deceased donor kidney transplant in December 2019. Soon thereafter, she experienced an episode of CMV infection and developed post-transplant diabetes mellitus (PTDM). Her medications included twice daily doses of tacrolimus 11 mg and MMF 500 mg, a once daily dose of prednisolone 5 mg, amlodipine 5 mg, aspirin 75 mg, bisoprolol 2.5 mg, co-trimoxazole 480 mg, doxazosin 2 mg, isoniazid 300 mg, omeprazole 20 mg, pyridoxine 25 mg, and gliclazide 120 mg and 80 mg daily. Three months after deceased donor kidney transplantation, on 10th March , she presented with shortness of breath to the emergency room. On initial assessment her oxygen saturations were 60% with heart rate of 105 beats/min and blood pressure of 190/99 mmHg. She was started immediately on CPAP and her oxygen saturations improved to 87%. Auscultation of the chest revealed widespread crepitations and her CXR showed bilateral pulmonary infiltrates (Fig S1A). She was found to be positive for SARS-CoV-2 RNA. Her CMV, adenovirus and other respiratory viral screen along with atypical pneumonia serologies were negative. There was no other positive microbiological diagnosis. She developed features of acute respiratory distress syndrome (ARDS) and AKI (creatinine 242 $\mu\text{mol/L}$, baseline 132 $\mu\text{mol/L}$).

Her respiratory status rapidly deteriorated in the emergency room and she required intubation 8 hours later and continues to be ventilated currently. MMF was stopped on 10th March and

tacrolimus on 16th March. Broad spectrum antibiotics and anti-viral, oseltamivir were administered. She was also empirically treated for pneumocystis with high dose co-trimoxazole. Serum CRP decreased from 329 mg/L on day of admission to 169 mg/L 7 days later. She became anuric and started CVVHF which continues. Her latest chest X-ray showed some resolution of the pulmonary infiltrates (Fig S1B).

4. A 65-year-old wheelchair-bound male, with a history of hypertensive nephrosclerosis and recurrent thromboembolic events developed ESRD in 2014 and received a deceased donor kidney transplant in August 2018. Seventeen months after kidney transplantation, he presented to hospital with shortness of breath and chest pain and was admitted to ITU. He was diagnosed with COVID-19 infection on 15th March. MMF was stopped and he currently continues with tacrolimus and prednisolone. He was discharged from the ITU and is currently admitted to a medical ward still requiring 4-6 L oxygen to maintain saturations. Kidney function remained stable.

5. A 69-year-old woman with long standing diabetes and hypertension, ESKD, on peritoneal dialysis therapy since 2012 and hemodialysis therapy since 2014, received a deceased donor kidney transplantation on 29th February and discharged on 9th of March. Her immunosuppressive treatment included Tacrolimus, MMF and prednisolone. Other medications included insulin, amlodipine 10 mg, ezetimibe 10 mg, levothyroxine 150 mcg, co-trimoxazole 480 mg, as well as doxazosin 4 mg BD and clonazepam 1 mg prn. She presented with shortness of breath, fever (39⁰C), diarrhoea and vomiting on 13th March. Her chest X-ray showed shadowing of left base on 13th March which worsened on 19th March (Fig S2A and Fig S2B). She tested positive for SARS-CoV-2 RNA on 14th March 2020. She was unwell with oxygen saturation of 82%, blood pressure 166/52 mmHg. Oxygen saturation improved to 97% with 4 L oxygen by nasal cannula. Haemoglobin was 74 g/L, serum NT-proBNP 5186 ng/L, and serum fibrinogen 4.2 g/L. Her lymphocyte count decreased on day 3 of admission to $0.3 \times 10^9/L$ and has remained low. Tacrolimus was continued and MMF was held from 14th March. She was treated initially with doxycycline, piperacillin/tazobactam, paracetamol, furosemide and blood

transfusion. She was moved to ITU on 15th March for respiratory support but did not need more than 5 L/min oxygen and transferred back to ward on the 17th March. On 20th March her serum creatinine was 138 $\mu\text{mol/L}$. She remains an in-patient and is being managed in general ward.

6. A 54-year-old man with urate nephropathy and past history of hereditary haemolytic anaemia, received a kidney transplant 7 years ago. He presented on 10th March with cough and fever (38.5°C) and tested positive for SARS-CoV-2 RNA on 13th March. He was adequately hydrated and vitals were stable. He received paracetamol and continued his usual medications including advagraf 3.5 mg OD, MMF 500 mg BD, nifedipine 30 mg OD, atorvastatin 30 mg at night, bisoprolol 10 mg OD, Ramipril 10 mg OD, Doxazosin 8 mg BD, alfacalcidol 1 mcg OD, and penicillin 250 mg OD. He developed AKI with a rise in creatinine from 145 $\mu\text{mol/L}$ to 187 $\mu\text{mol/L}$. Haemoglobin was 141 g/L Blood cell counts are shown in Table 2. He remained symptomatic on 21th March with cough and mild fever. As the symptoms were not resolving, MMF was stopped and he has managed to stay at home.

7. A 45-year-old man with a failing, second kidney transplant from September 2017 presented with fever, flu-like symptoms and cough for 7 days and shortness of breath for 1 day. He had arterial hypertension with no other comorbidities. He was a sensitised recipient Panel Reactive Antibody 90%) and therefore, was maintained on long-term triple immunosuppression: tacrolimus, azathioprine (switched in late 2018 from MMF due to gastrointestinal side effects) and prednisolone 10 mg OD. On admission on 17th March, he was tachypnoeic and hypoxic with oxygen saturation of 90% on room air, corrected to >95% on 4 L/min oxygen through nasal cannula. Nasal and throat swabs were positive for SARS-CoV-2 RNA. He developed AKI with serum creatinine 967 $\mu\text{mol/L}$ and eGFR 5 ml/min/1.73 m² (baseline creatinine 400-450, eGFR 12-16). He was lymphopenic with lymphocyte count of 0.3 $\times 10^9/\text{L}$ (baseline 1-1.2 $\times 10^9/\text{L}$) with normal haemoglobin and white cell count. Liver function tests were normal on admission but ALT went up to 138 U/L on days 4.. Chest X-ray revealed bilateral infiltrates (Fig S3). Azathioprine was stopped on admission, tacrolimus reduced and prednisolone increased to 15 mg OD. He needed so far one hemodialysis session. He is

recovering from respiratory point of view and as of 23st March 2020, the oxygen saturations are >95% on 2 L/min. He remains haemodynamically stable.

Discussion

In this report we discuss our first 7 cases of COVID 19 infection in kidney transplant recipients from south London, UK. Median age of transplant recipients was 54 years (range 45-69y) comprising 4 males, 3 females. Out of 7 patients, 2 were managed on an out-patient basis and stayed at home, with the remaining 5 (71%) requiring hospital admission. Four among the latter required ITU admission and one is being managed in the renal ward. Out of four patients sent to ITU, two needed intubation and ventilation; the other two were managed with oxygen through mask and non-invasive ventilation only. There was one death in this small series of 7 patients (mortality rate of 14%). All 3 patients with severe disease were females and also had diabetes. Two patients presented within 3 months of kidney transplantation (one within 2 weeks) while kidney transplant vintage was 12 months or more in the remaining five cases. The patients were managed in three centres and the total number of prevalent transplant patients in these centres was 2082, with 32 patients transplanted from 15th December 2019 to 15th March 2020 during the developing pandemic.

Transplant patients are at higher risk due to immunosuppression, underlying CKD and other co-morbidities, in particular diabetes and hypertension which are now recognised as significant factors that influence outcomes in patients with COVID 19 infection. (9) Three of our patients had CKD stage 4-5 with one recovering at home and one requiring hospital admission but recovering without needing ITU admission. The remaining 4 patients had CKD stage 3 of which two had severe disease requiring intubation and ventilation and one of them died. Both patients who had severe COVID-19 including the one who died had diabetes mellitus.

Managing immunosuppression in these patients is challenging and this should take in to account age of, severity of COVID 19 infection, associated co-morbidities and time post-transplant. In transplant

patients with mild to moderate infections, usual practice is to continue or make reductions in the dose of immunosuppressive drugs but this approach might favour high mortality in patients admitted to hospital with COVID-19 infection. Whilst we acknowledge that firm recommendations are not possible based on the small sample size of this study, we suggest that anti-proliferative agents (mycophenolate mofetil/azathioprine) should be stopped at the time of admission to hospital, dose of prednisolone either unchanged or increased, and tacrolimus dose reduced. In severe infections (requiring intubation and ventilation), an argument can be made for stopping calcineurin inhibitors completely while maintaining corticosteroid therapy. The role of cytokine storm and inflammation due to anti-viral immune response as a driver of severe respiratory disease and ARDS has been discussed since the outbreak of this disease in December 2019, prompting trials of anti-interleukin 6 monoclonal antibody tocilizumab and case for continuing steroids in infected patients. Similar argument can be made for continuing low dose tacrolimus but more evidence is needed before drawing firm conclusions. An obvious concern is risk of rejection with reduction in immunosuppression but given the high mortality rate of COVID-19 infection in hospitalised patients, clinicians should focus on keeping their patients alive with a careful case by case assessment of risks versus benefits of continuing immunosuppression. With regards to induction treatment, it is likely that lymphocyte depleting antibodies increase the risk and therefore, many centres in the UK have stopped performing transplants requiring induction with either ATG or alemtuzumab. All patients in this series received basiliximab induction therapy at time of transplantations. Five of the 7 patients presented here were receiving triple immunosuppression. Two patients with mild illness who did not require hospital admission and recovered fully at home were on dual immunosuppression (one on azathioprine/ prednisolone and one on tacrolimus/MMF).

With regard to concomitant therapy with ACEI/ARBs, in line with current UK Renal Association and European Society of Cardiology recommendations, these therapies were not discontinued.

One out of our 7 patients died (a mortality rate of 14%) although it is too soon to comment on likely mortality rates in this group of patients. Two of our patients presented within 3 months after transplantation and one presented within 2 weeks. UK NHSBT/ODT have since produced guidelines on COVID-19 screening in deceased donors and the transplant units are risk stratifying donors and recipients before considering kidney transplantation. Transplantation is a high-risk procedure during this pandemic due to the risk of transmitting COVID-19 infection from the donor to the recipient as well as risk of recipient developing severe disease under higher levels of immunosuppression in the first 3 months post transplant. We suggest that apart from carefully selecting donor-recipient pairs, transplantation is not advisable during this pandemic, especially for older recipients with co-morbidities, in particular diabetes. We have stopped performing living donor transplants and are in discussions to suspend deceased donor programme. In addition to significant concerns about the effect of COVID-19 on immunosuppressed patients, increasing worries about access to ITU in the coming weeks and re-distribution of staff to critical care to provide support for increasing number of COVID-19 patients, it is likely that deceased donor programme will be suspended within most of the UK centres soon.

AKI has been described with COVID-19 infections in up to 15% patients, and occurrence of proteinuria /hematuria has been reported. In our series, the observation that 4/7 patients had AKI (57%) may be an early signal that transplant patients are at higher risk of AKI with COVID-19 infection, compared to 29% AKI in critically ill patients of general population in Wuhan, China. (11) ACE2 and dipeptidyl peptidase (DPP4), which are expressed in proximal tubule cells, (12, 13) have been identified as receptors for SARS-CoV and MERS-CoV. Uptake of SARS-CoV-2 virus into the proximal tubular epithelium is a possible explanation for AKI.

With regards to prognostic blood tests including lymphocyte counts and serum levels of D dimer, ferritin and troponin are likely to be valuable. Four out of five patients who required admission had lymphopenia, whereas the two who did not need admission had normal lymphocyte counts. As

many patients on immunosuppression are likely to have baseline lymphopenia, further drop in lymphocyte count is likely to be of prognostic value. In our patient who died, both D dimer and troponin levels were elevated on day 3 post admission with further marked increase (in particular D dimer) later during the course of her illness. In the absence of any obvious thromboembolic events, this suggests microvascular thrombosis or DIC with possible gut ischaemia. Very high ferritin and D dimer levels were also noted in case 7 of our series. We suggest that D dimer, ferritin and troponin should be measured in all patients with severe COVID-19 infection on admission and subsequently in those who are not showing clinical improvement.

In two of our patients, the lung infiltrates showed significant improvement without any specific anti-viral treatment 7-9 days post admission. The patient who died is one of them and was improving from the respiratory point of view. She died of an abdominal complication and the clinical diagnosis was possible bowel infarction or intra-abdominal sepsis. Based on this observation we would like to highlight that the mortality in critically ill patients with COVID-19 infection could be due to extra-pulmonary complications such as myocarditis or bowel involvement.

With regards to specific anti-viral therapies, although a recent trial showed no benefit of lopinavir-ritonavir in hospitalized patients with severe COVID-19, it remains possible that treatment with these drugs as well as hydroxychloroquine will be considered in patients with COVID-19 pneumonia.

(14) The choice of calcineurin inhibitor may also have a role to play. Thus for instance cyclosporin A has been shown to have an inhibitory effect on proliferation of corona viruses and hepatitis C virus in vitro, while this is not the case for tacrolimus. Cyclosporin A is thought to inhibit the replication of a diverse array of coronaviruses through its impact on cyclophilin A and B. (15, 16) Whilst this needs further exploration, we do not think switching to cyclosporine A from tacrolimus can be recommended at this stage for transplant patients with COVID-19 infection.

In conclusion, in this first series of 7 renal transplant patients infected with SARS-CoV-2 virus, one recipient died (14%) and significant AKI was observed. Lymphopenia, very high ferritin and D dimer

levels and raised troponin levels are seen in severe disease and may be of prognostic value. These tests should be part of routine testing in kidney transplant patients requiring hospital admission for COVID-19 infection. We suggest suspending kidney transplantation during the COVID-19 pandemic particularly for high risk older recipients with comorbidities. Rigorous adherence to hand hygiene, recommended isolation procedures and regular virtual/telephonic assessment of transplant patients will help reduce the incidence and facilitate management of mild to moderate cases in the community as we could in two of our 7 patients described.

The COVID-19 UK register has been set up by UK transplant registry held by ODT to record all cases of renal transplant patients presenting with COVID-19 infection and analysis of registry data will help clinicians make informed decisions about management of these complex patients in these uncertain and rapidly evolving times.

Figure 1: Case2: CxR on admission(1A) showing bilateral patchy consolidation and 8 days later showing improvement in lung infiltrates (1B)

Supplementary Material

Supplementary Figure 1: Case3: CxR on admission(S1A) showing bilateral patchy consolidation and 9 days later showing improvement in lung infiltrates (S1B)

Supplementary Figure 2: Case5: CxR on admission(S2A) showing left basal shadow worsening to B/L patchy consolidation 6 days later (S2B)

Supplementary Figure 3: Case7: CxR on admission showing bilateral lung infiltrates

Supplementary information is available on Kidney International's web site.

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Table 1 Clinical characteristics and outcome of seven kidney transplant patients with COVID 19 infection

	Age/ Sex	Tx date	Co- morbidity	Respiratory and renal involvement	Baseline creatinine (eGFR ml/min/1.73 m ²)	Baseline Immunosuppression and treatment	ACEi or ARB	Outcome
1	48/ M	1989	HT	No	350 (15-18)	Aza/Pred- No change	N	Stayed at home, full recovery
2	67/F	03/20 19	T2D/HT	Yes, ARDS + AKI(CVVH)	150(45)	Tac/MMF/Pred- MMF stopped	Y ACEi	Died
3	54/F	12/20 19	PTDM/ CMV	Yes, ARDS+AKI (CVVH)	132(48)	Tac/MMF/Pred- Tac and MMF stopped	N	Alive, Ventilated
4	65/ M	08/20 18	Wheelchair/ HTN	No ARDS	180 (23)	Tac/MMF/Pred MMF stopped	N	Alive, in medical ward
5	69 /F	02/20 20	DM/HT	No ARDS AKI	165 (31)	Tac/MMF/Pred MMF stopped	N	Brief ITU stay, not intubated stepped down to ward

6	54 /M	05/20 13	Haemolytic anaemia/HT	No ARDS	187(47)	Tac/MMF MMF stopped	N	Stayed at home, still has some cough and flu like symptoms
7	45 /M	09/20 17 (2 nd Tx)	HT	No ARDS AKI(HD)	450 (12-16)	Tac/Aza/Aza Aza stopped Tac dose reduced	N	Admitted, managed in the ward. Severe AKI

Legend: Yr=year, M=male, F=female, Y=yes, N=No, Aza=azathioprine, MMF=mycophenolate mofetil, ITU=intensive therapy unit, NIV=non-invasive ventilation, CVVH-continuous veno-venous haemofiltration, AKI-Acute Kidney injury, ARDS-Acute Respiratory Distress Syndrome).

Table 2: Blood parameters during COVID-19 infection

Case	White cell count ($\times 10^9/L$) (3.5-10)	Lymphocyte count ($\times 10^9/L$) (1-3.5)	Serum CRP (mg/L) (<5)	Serum ferritin ($\mu g/L$) (25-200)	Serum D Dimer ($\mu g/L$) (0-500)	Serum LDH (U/L) (100-240)	Serum troponin I (ng/L) (<34)
1							
2	6(D1)	0.8(D1)	83 (D1)		2032 (D3), >6000 (D10)	1226 (D10)	78(D1), 395 (D10)
3	11.25(D1)	0.5(D1)	329 (D1)				
4							
5	9.4(D1)	0.3(D1)					*30 (D4)
6	10(D1)	4.0(D1)					
7	5.5(D1)	0.3(D1)	198 (D1)	6919 (D3)	1907 (D3)	502 (D3)	35 (D7)

CRP- C-reactive protein, LDH- Lactate Dehydrogenase

*Serum troponin T (0-14ng/l)

D=day after admission and D1 is day of admission

