Report into a nosocomial outbreak of coronavirus disease 2019 (COVID-19) at Netcare St. Augustine’s Hospital

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Disclaimer

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Note

This is the final report on the outbreak investigation at St. Augustine’s Hospital. It contains additional and updated information to that in the interim report released on 30 April, in particular updated number of cases and deaths. It has also been corrected for a small number of factual and typographical errors that were in the interim report.
Executive summary

This report presents the findings and recommendations of an investigation into a nosocomial outbreak of coronavirus disease 2019 (COVID-19) at St. Augustine’s Hospital in Durban, South Africa. The investigation began on 4 April after the identification of a number of confirmed COVID-19 cases and three deaths at the hospital. Investigation methods included medical record reviews, ward visits, and interviews with health care workers and management. A detailed timeline of patient cases was constructed to generate hypotheses as to the spread of infection through the hospital. In addition, DNA sequencing of severe acute respiratory syndrome-related coronavirus 2 (SARS-CoV-2) nucleic acid extracted from nasopharyngeal and oropharyngeal swab samples was performed and phylogenetic analysis was conducted.

Between 9 March and 30 April 2020, 119 confirmed cases were identified at St. Augustine’s Hospital (39 patients and 80 staff). The 80 staff represent approximately 5% of all staff tested for SARS-CoV-2. The most plausible hypothesis is that there was a single introduction of SARS-CoV-2 to the hospital on 9 March, most likely as a result of transmission from a patient attending the Emergency Department for investigation of COVID-19 to another patient present in the ED at the same time who was then admitted to the cardiac intensive care unit with a suspected stroke. The infection then spread rapidly through the hospital, involving patients on at least five wards. The spread through the hospital was facilitated by the frequent movement of patients between and within wards. The evidence suggests that indirect contact and fomite transmission were likely to be the predominant modes of patient to patient transmission. We hypothesize that the main outbreak also seeded smaller outbreaks at a local nursing home (four additional cases) and at the National Renal Care outpatient dialysis unit on the hospital campus (nine additional patient cases and eight additional staff cases). Overall, we estimate that up to 135 infections may have been nosocomially acquired as a result of the single introduction of the virus to the hospital, accounting for about 14% of all cases in KwaZulu-Natal reported by 30 April. Phylogenetic analysis supports the main hypothesis of a unique introduction followed by widespread transmission in the hospital. All of the 18 SARS-CoV-2 genomes produced (nine from patients and nine from health care workers) clustered together with limited genetic diversity. All of the sequences belong to the A2a clade associated with infections from Europe.

Fifteen of the 39 patients infected with SARS-CoV-2 in the main outbreak died (case fatality rate 38.5%). Most of the deaths were in elderly patients with multimorbidity. In most cases, a medical decision was taken not to intubate and ventilate because of the comorbidities and poor prognosis. There was no evidence that, once these patients had been infected with SARS-CoV-2, any specific intervention would have prevented their death.

With the benefit of hindsight, there were a number of opportunities where earlier problem recognition and earlier intervention might have limited the extent of the outbreak. The first opportunity was with the unexplained fever of a 81-year-old female on 13 March following a transient ischaemic attack; the second was when a 46-year-old female was readmitted with an acute respiratory illness on 21 March; the third was when the 81-year-old female was readmitted with severe pneumonia on 22 March; and the fourth was with the first confirmed case in a health care worker (a nurse from cardiac ICU), reported on 23 March. Earlier recognition of possible COVID-19 infection in the patients, leading to earlier isolation, tracing of potential sources of infection, and appropriate management of exposed contacts could potentially have averted infections and limited onward transmission. Earlier investigation of the first health care worker case to identify potential sources of infection within the hospital could also have uncovered the problem at an earlier stage.
This outbreak highlights how easily and rapidly SARS-CoV-2 can spread through a hospital, exposing weaknesses in respiratory virus infection prevention and control (IPC). It underlines that personal protective equipment (PPE) is only one component of a comprehensive approach to IPC and does not replace the need for good IPC systems and practices. The extent of the outbreak underlines the potential for nosocomial transmission to be a major amplifier of transmission in South Africa. There is no reason to believe that a similar outbreak cannot and will not happen in other hospitals and institutions in South Africa, in both the private and public sector.

To reduce the risk of similar outbreaks, we need to strengthen infection prevention and control systems and practices throughout our hospitals. Management must promote a culture that IPC is everyone’s responsibility and that everyone has a role to play. Hospitals need to establish separate zones (and separate entry points) for people who might have COVID-19 and people who are unlikely to have COVID-19. There needs to be vigilance throughout the hospital for acute respiratory illness, especially in green zones where patients considered low risk for COVID-19 have been admitted. Training on COVID-19, especially on infection prevention & control, should be mandatory for all staff and implementation of IPC practices should be monitored closely. The importance of hand hygiene needs to be continually emphasised and hand hygiene practices need to be monitored.

Environmental cleaning practices need to be aligned with the national COVID-19 IPC guidelines and the national IPC framework manual. Cleaning should be monitored closely using visual inspection and fluorescent markers. The importance of regular cleaning of surfaces and of medical equipment between patient contacts to reduce fomite transmission should be highlighted to all staff. Physical distancing within the hospital should be promoted through the use of floor markings and prominent signage. Consideration should be given to weekly PCR testing of all frontline staff and the early use of DNA sequencing and phylogenetic analysis to investigate potential nosocomial transmission.

The COVID-19 epidemic is an unprecedented challenge for the health system and the community in South Africa. We hope that lessons learnt from this nosocomial outbreak can be used to highlight areas that can be strengthened across the private and public health system, so as to prevent nosocomial outbreaks becoming a major amplifier of COVID-19 transmission.
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<th>Description</th>
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<tbody>
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<td>ARDS</td>
<td>Acute respiratory distress syndrome</td>
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<tr>
<td>CICU</td>
<td>Cardiac intensive care unit</td>
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<tr>
<td>COVID-19</td>
<td>Coronavirus disease 2019</td>
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<tr>
<td>ED</td>
<td>Emergency department</td>
</tr>
<tr>
<td>HCW</td>
<td>Health care worker</td>
</tr>
<tr>
<td>IALCH</td>
<td>Inkosi Albert Luthuli Central Hospital</td>
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<tr>
<td>IPC</td>
<td>Infection prevention and control</td>
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<tr>
<td>MICU</td>
<td>Medical intensive care unit</td>
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<tr>
<td>MW</td>
<td>Medical ward</td>
</tr>
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<td>National Department of Health</td>
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<td>National Health Laboratory Service</td>
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<td>National Institute for Communicable Diseases</td>
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<td>NRC</td>
<td>National Renal Care</td>
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<tr>
<td>PCR</td>
<td>Polymerase chain reaction</td>
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<tr>
<td>PPE</td>
<td>Personal protective equipment</td>
</tr>
<tr>
<td>PUI</td>
<td>Person under investigation</td>
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<tr>
<td>RNA</td>
<td>Ribonucleic acid</td>
</tr>
<tr>
<td>SARS-CoV-2</td>
<td>Severe acute respiratory syndrome-related coronavirus 2</td>
</tr>
<tr>
<td>SICU</td>
<td>Surgical intensive care unit</td>
</tr>
<tr>
<td>SW</td>
<td>Surgical ward</td>
</tr>
<tr>
<td>UKZN</td>
<td>University of KwaZulu-Natal</td>
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<td>WHO</td>
<td>World Health Organization</td>
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Introduction

On Saturday 4 April, at the request of Dr Anban Pillay (Acting Director General, National Department of Health), Professor Salim Abdool Karim convened a team to investigate an outbreak of coronavirus disease 2019 (COVID-19) at St. Augustine’s Hospital. Professor Abdool Karim is the Director of the Centre for the AIDS Programme of Research in South Africa (CAPRISA) and is currently serving as the Chair of the Ministerial Advisory Committee on COVID-19 in South Africa. The outbreak investigation team consisted of scientists and clinicians from the University of KwaZulu-Natal: Dr Richard Lessells, Infectious Diseases Specialist at the KwaZulu-Natal Research Innovation & Sequencing Platform (KRISP); Professor Tulio de Oliveira, Research Professor and Director of KRISP; and Professor Yunus Moosa, Head of Department of Infectious Diseases.

The investigation was requested following reporting of 13 cases of COVID-19 in inpatients and health care workers at St. Augustine’s Hospital, three of which had resulted in death.

Background

The global epidemic of coronavirus disease

Coronavirus disease 2019 (COVID-19) is an infectious disease caused by the severe acute respiratory syndrome-related coronavirus 2 (SARS-CoV-2). COVID-19 is predominantly a respiratory disease, which presents most commonly with fever and dry cough, and which in more severe cases can progress to viral pneumonia and acute respiratory distress syndrome (ARDS). At present, the case fatality rate is estimated to be 1-3%, although this varies considerably by setting. The disease was first identified in Wuhan, China in December 2019 and has subsequently spread to 188 countries around the world. As of 30 April, there were over 3 million confirmed cases and over 200 000 deaths related to COVID-19.

COVID-19 in South Africa

The first confirmed case in South Africa was reported on 5 March in a 38-year-old male from KwaZulu-Natal Province who had recently arrived back in the country from a holiday in Italy. As of 29 April, there had been 5350 confirmed cases and 103 deaths related to COVID-19 (Figure 1). An initial exponential rise in number of cases, similar to many European countries, has then been followed by a slower growth since 28 March.

![Cumulative and daily COVID-19 data for South Africa](image)

*Figure 1 Cumulative and daily counts of confirmed COVID-19 cases for South Africa (data up to 29 April)*

- 1 -
The majority of infections in South Africa were initially in three provinces, namely Gauteng, KwaZulu-Natal and Western Cape, which contain the main urban populations and the international travel hubs. In KwaZulu-Natal Province, as of 30 April, there were 956 confirmed cases and 32 deaths (Figure 2).

**Transmission of SARS-CoV-2**

SARS-CoV-2 is thought, on the basis of current evidence, to be transmitted between people through respiratory droplets and contact. Droplet transmission occurs when a person is in close contact (within 1 metre) with someone who has respiratory symptoms (e.g. cough) and is therefore at risk of having their mucosae (mouth and nose) or conjunctiva (eyes) exposed to potentially infective respiratory droplets (which are generally considered to be >5-10μm in diameter). Droplet transmission may also occur through fomites in the immediate environment around the infected person. Therefore, transmission of SARS-CoV-2 can occur by direct contact with infected people and indirect contact with surfaces in the immediate environment or with objects used on the infected person (e.g. stethoscope or thermometer). SARS-CoV-2 can be highly stable on certain surfaces, particularly plastic and stainless steel, but is susceptible to standard disinfection measures.

Whilst aerosol transmission may be possible in specific circumstances, particularly in the health care setting with aerosol-generating procedures (i.e. endotracheal intubation, open suctioning, and

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manual ventilation before intubation), there is currently no evidence that aerosol transmission is an important mode of transmission more generally\(^1\).

Based on current evidence, transmission of SARS-CoV-2 is thought to be predominantly driven by symptomatic individuals, who may be most infectious around the time of symptom onset. However, there is increasing evidence that SARS-CoV-2 is detectable in the upper respiratory tract from 1-3 days prior to symptoms\(^1\), and modelling suggests that presymptomatic transmission may be an important contributor to the spread of the infection. Recent estimates have suggested that around 40% of transmission may occur in this presymptomatic phase\(^ii\). Whilst it is recognized that some people have true asymptomatic infection (i.e. never develop symptoms), we still do not know how frequent this is and we do not know to what extent this contributes to transmission.

**Nosocomial transmission of SARS-CoV-2**

In the early phases of the epidemic in China, the risks of nosocomial transmission of SARS-CoV-2 became apparent. In one case series of 138 consecutive hospitalized patients with confirmed COVID-19 at a university hospital in Wuhan, 57 (41%) cases were presumed to have been infected in hospital. This included 17 patients hospitalized for other reasons and 40 health care workers, and there was presumed patient-to-patient transmission as well as patient-to-health care worker transmission\(^iii\). The WHO-China Joint Mission on COVID-19 reported that, as of 20 February 2020 there had been 2055 confirmed cases in health care workers across 476 hospital in China (at that time, this was approximately 3% of all confirmed cases in the country)\(^iv\). The report noted that many of the health care workers may have been infected within their households rather than the health care facility. As a result, they surmised that nosocomial transmission had not been a major amplifier of transmission. In Italy, around one in ten confirmed cases have been in health care workers and entire hospitals have been closed because of the infection circulating among doctors and nurses\(^v\). In a recent report from the United States of America, almost one in five confirmed cases were health care workers\(^vi\).

**Netcare St. Augustine’s Hospital**

Netcare St. Augustine’s Hospital is a 469-bed hospital situated on the Berea in Durban. It has 18 wards, including six intensive care units providing a total of 88 ICU beds. The hospital employs 735 staff (484 nursing and 251 non-nursing), and has another 1247 support staff. The breakdown of the staff complement by group is shown in Table 1. In Feb and March 2020, agency nurses covered 32% of nursing shifts. The hospital has two Infection Prevention & Control (IPC) Practitioners at the hospital with a Regional IPC Manager to cover all Netcare hospitals in the Coastal Region.

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Table 1 Current staff complement at Netcare St. Augustine’s Hospital

<table>
<thead>
<tr>
<th>Staff group</th>
<th>Number</th>
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<tbody>
<tr>
<td><strong>Netcare staff</strong></td>
<td>735</td>
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<tr>
<td>Nursing staff</td>
<td>484</td>
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<tr>
<td>Non-nursing</td>
<td>251</td>
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<tr>
<td><strong>Support staff</strong></td>
<td>1247</td>
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<tr>
<td>Agency nursing staff</td>
<td>281</td>
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<tr>
<td>Cleaning (Tsebo)</td>
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<tr>
<td>Catering (Empact)</td>
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<tr>
<td>Security</td>
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<td>Doctors</td>
<td>139</td>
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<tr>
<td>Physiotherapists</td>
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<tr>
<td>Ampath Laboratories staff</td>
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<tr>
<td>Lancet Laboratories staff</td>
<td>29</td>
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<tr>
<td>Radiology (Lake Smit &amp; Partners)</td>
<td>55</td>
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<tr>
<td>Laundry</td>
<td>21</td>
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<tr>
<td>Other support staff</td>
<td>27</td>
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COVID-19 preparedness

In terms of the response to COVID-19, a facility readiness assessment on 7 February 2020 scored 91%, with a few key gaps identified: lack of a facility preparedness and response plan, lack of policies and procedures for monitoring and managing health care workers with potential for exposure to SARS-CoV-2, need to review plans for visitor access and movement, lack of event-based response system, and need for staff training in the management of specimen packaging and transport.

The hospital delivered face-to-face training on COVID-19 in February 2020 with Netcare staff and support staff (using the standard training materials developed by the NICD and National Department of Health). Records from 19 March show that more than 80% of nursing staff and non-nursing Netcare employees had been trained. The same records show that only 43% of contract or outsourced staff had been trained. It was noted particularly that no doctors had been trained. It is not known whether these staff received training from other sources.

In early March, an Operational Manual was released and a Joint Operations Committee (JOC) was formed on 18 March, including physicians, surgeons, anaesthetists and intensivists. On 6 March (the day after the first confirmed case in South Africa), the hospital initiated entry screening, using the national screening tool, and reduced the number of entry points to the hospital (from 12 to five). On 16 March a repeat facility readiness assessment scored 100%. The facility readiness tool was on both occasions completed by the Deputy Nursing Manager, with input from the IPC practitioner.

Investigation methods

This investigation was conducted in situ at St. Augustine’s Hospital between 4 April and 20 April, with additional data collected in early May. The outbreak investigation team (Dr Lessells, Prof Moosa and Prof de Oliveira) was supported at all times by Liza Sitharam (Regional IPC Manager Coastal Region for Netcare), Nicole Govender and Maryann Maistry (IPC Practitioners, St. Augustine’s Hospital). The investigation involved the following:
- Review of medical files, laboratory records, and radiological images of confirmed cases
- Review of inpatient electronic tracking system to determine the movement of cases within and between wards and to identify the exact location of beds occupied
- Scrutiny of staff records to understand work areas and shift patterns of infected staff
- Walk through the emergency department to understand patient flow and to critically review the COVID-19 isolation/ triage area
- Tour of all affected inpatient wards to understand patient and staff movement and bed distribution within the wards
- Desktop review of Netcare COVID-19 Operational Manual and hospital protocols
- Telephone interview with doctors involved in the care of affected patients
- Discussions with the IPC Practitioners and Manager

To complement the core investigation, we received samples positive for SARS-CoV-2 from the outbreak. The samples arrived in batches at different times. We managed to generate whole SARS-CoV-2 genomes from 18 samples (eight from St. Augustine’s patients, nine from health care workers, and one from a local nursing home). We also sequenced whole viral genomes of five randomly selected samples from eThekwini that were provided to us by the NICD.

In short, this process involves the generation of whole genome sequences of SARS-CoV-2, which contains 29,500 nucleotides. We used the ARTIC network protocol to amplify the genome of the virus1. The genome was sequenced using the Illumina Miseq platform. The ARTIC protocol and the Illumina sequencing platform are considered to be the gold standard of the field, i.e. the most accurate system to generate whole genome sequences of SARS-CoV-2. The sequences were assembled using Genome Detective2 and all of the mutations were carefully evaluated using the bam files (i.e. deep coverage) in Geneious R8 software application3.

Results

Epidemiological curve

Between 9 March and 30 April, there were 119 confirmed COVID-19 cases in patients and staff at St. Augustine’s Hospital, comprising 39 patients and 80 staff members. The patients include three outpatients seen only in the emergency department, 26 hospitalised patients diagnosed at St. Augustine’s, five patients hospitalised at St. Augustine’s in March/April but then readmitted and diagnosed with COVID-19 at other hospitals, and five patients hospitalised at St. Augustine’s in March then diagnosed with COVID-19 during a recall screening & testing process. The epidemiological curve is shown in Figure 3.

The first three cases at the hospital were people who attended the Emergency Department for assessment, triage and sample collection but were not admitted. The first two (sampled on 9 March and 12 March respectively) had recently returned from Europe; the third case sampled on 15 March was a local resident with no travel history and no definite contact with a confirmed case, but who had contact with many international travellers through her work.

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3 Geneious R8 (https://www.geneious.com)
The first inpatient case was an 81-year-old female who was diagnosed with COVID-19 on 25 March following a positive SARS-CoV-2 PCR test on an endotracheal aspirate and an oropharyngeal swab collected on 22 March. This patient was first admitted to the hospital from Arcadia Old Age Home in the Bluff on 9 March with slurred speech, right-sided weakness and sensory disturbance. She was diagnosed with a transient ischaemic attack. She spent time on cardiac ICU then medical ward 1. Magnetic resonance imaging of the brain demonstrated a left perirolandic ischaemic infarct. On 16 March she was discharged to the Bill Buchanan Association for the Aged in Morningside. Six days later (22 March), she was readmitted to the medical intensive care unit (MICU) in respiratory distress, requiring intubation and ventilation. Chest X-ray on admission demonstrated diffuse bilateral airspace opacification with sparing of the lung apices. At that time, the medical team contacted the NICD hotline and were advised not to test for SARS-CoV-2 as this person did not meet the surveillance case definition. The medical team nevertheless sent an oropharyngeal swab for SARS-CoV-2 polymerase chain reaction (PCR), given the presentation of a severe pneumonia of unknown aetiology and the clinical suspicion of COVID-19. An indeterminate result (detection of E gene only) was communicated to the physician on the evening of 22 March; the swab sample was sent to the reference laboratory for confirmatory testing and at the same time an endotracheal aspirate sample was processed for SARS-CoV-2 PCR testing at the local laboratory. Both tests were positive and results were reported on 25 March.

**Timeline of inpatient cases**

Figure 4 displays the timeline of 34 patient cases, including the three outpatients (P1, P2, P18), the 26 people who were hospitalised at St. Augustine’s Hospital only (P3-P17, P19-P29), and the five people who were discharged from St. Augustine’s Hospital and then readmitted and diagnosed with COVID-19 at another hospital (X1 – X5). The timeline displays the movement and time spent on the different wards and pinpoints the date of symptom onset (for the symptomatic cases) and date of sample collection. This timeline is truncated at 14 April. The full timeline up to 6 May is shown in Appendix A.

This timeline was used to observe overlap of time spent in the same ward between symptomatic cases and susceptible patients who later became infected. From this exercise, we generated a hypothesis about how the virus might have spread through the facility (Figure 5).

Under this hypothesis, SARS-CoV-2 was most likely introduced to the hospital by the first confirmed case seen in the Emergency Department on 9 March. After its introduction, the virus spread widely through the hospital. Patients were exposed to the virus in six main waves of transmission involving...
five hospital wards. In chronological order, these were medical ward 1, neurology ward, medical ICU, medical ward 1 (a second wave involving a new cluster of infections), surgical ICU, and surgical ward 1. A close correlation was found between the sites where intense transmission occurred amongst patients and the wards where high levels of health care worker infections were observed. We also hypothesise that the main St. Augustine’s outbreak seeded smaller outbreaks in a local nursing home and in the National Renal Care (NRC) Berea outpatient dialysis unit on the St. Augustine’s site.

Figure 4 Timeline of COVID-19 cases at St. Augustine’s Hospital (up to 14 April) showing patient location over time
Figure 5 Hypothesis showing putative waves of COVID-19 transmission centred on different wards and showing spread to nursing home and dialysis unit.
Postulated introduction of SARS-CoV-2 to the hospital (transmission P1 to P3)

On review of the timeline, it was noticed that the first outpatient case (P1) and the person who would become the first inpatient case (P3) were both in the Emergency Department on 9 March. A more detailed investigation of events of that day uncovered that they were in the ED at overlapping times, were in close proximity to one another, and were attended to by the same medical officer. P1 was located in the isolation/triage area - it was noted that, although this was a separate room off the main resuscitation bay, entry and exit to this room required movement through the main resuscitation bay. It was notable that P3 was located in the trolley almost directly opposite the entrance to the isolation/triage room. Figure 6 shows the layout of the ED and describes the movements and timings of the two patients on the evening of 9 March.

![Figure 6 Summary of events in emergency department, St. Augustine’s Hospital, 9 March](image)

**Figure 6 Summary of events in emergency department, St. Augustine’s Hospital, 9 March**

Patient journey for P3

Patient P3 was admitted to the Cardiac Intensive Care Unit (CICU) from the ED on the evening of 9 March and stayed there 4 nights until 13 March. She was then transferred to Medical Ward 1 (MW1) and was there until 16 March when she was discharged to the Bill Buchanan Association for the Aged in Morningside. On review of the patient file, we noted a significant fever (38.9°C) on the 13 March on MW1. No respiratory symptoms were noted in the patient file, or by the treating physician, and the matron at the nursing home reported that she did not have respiratory symptoms on arrival at the home on 16 March. According to the matron, her condition deteriorated on 19 March with hypotension and dizziness, followed by fever, dyspnoea and wheeze on 21 March. In retrospect the fever might have been the first symptom of COVID-19, suggesting that P3 was symptomatic and infectious during the first admission, and this supports the hypothesis that she may have been infected on 9 March in the ED with an incubation period of 4 days.

Further support for this is provided by the details and timing of the first confirmed staff case. The first staff case was a 62-year-old female nurse working on CICU. She developed cough and sore throat on 17 March and visited her GP on 18 March – swabs collected on that day were positive on
PCR for SARS-CoV-2 (result received on 23 March). On review of her shift patterns and patient allocation, it was noted that she was directly responsible for the care of P3 in CICU on the night shift of 12 Mar-13 March. Based on the understanding that people can be infectious 1-3 days prior to symptom onset, this link provides strong circumstantial evidence that P3 was infectious at this time and transmitted to the CICU nurse.

Chains of transmissions

Medical ward 1
On the assumption that P3 was infectious throughout her time on MW1 (13-16 Mar), there were five other cases who may have been exposed during that time period. This includes a 46-year-old female (P4) who was in the bed directly opposite (in room 12), and four male patients (P6, P7, P14, X4) who were co-located in a room down the corridor (room 15). Figure 7 includes an extract of the timeline specific to this period on this ward and a detailed ward layout showing the location of the symptomatic individual (P3) and the other patients who would subsequently be diagnosed with COVID-19.

![Diagram showing patient locations and timeline for Medical Ward 1](image)

Figure 7 Specific timeline and ward layout showing infectious case and exposed individuals on medical ward 1 from 13 March onwards (red outline shows bed occupied by infectious case, green outlines show beds occupied by exposed individuals)

Neurology ward
On 21 March (five days after discharge), P4 was readmitted to MW1 with cough, dyspnoea and vomiting (similar presentation to the previous admission 7-16 Mar). On 21 March, as part of COVID-19 preparedness, MW1 was closed in order to prepare it as a COVID-19 ward. As a result, P4 was decanted to the neurology ward. She became febrile on 23 March whilst on that ward. As can be seen in Figure 8 there were two other patients (P11, P15) who subsequently became infected – both
were tested during the systematic testing of all inpatients on 3–4 April. Of note, these two patients were located in different parts of the ward so direct patient-to-patient transmission is unlikely. A professional nurse on this ward (HW4) became symptomatic with fever and flu-like symptoms at the end of her shift on 23 March and subsequently tested positive on 29 March. This nurse worked directly with P4 on 23 March, when P4 was coughing, and performed tasks including nebulisation. She was signed off work until 27 March and when she returned to work on 27 March she was still symptomatic with cough, fever and sore throat, but she continued to work day shifts on 27 & 28 March. On those days she worked directly with P11 and P15. This chain of events suggests that on this ward there was probably patient to health care worker transmission followed by health care worker to patient transmission.

Medical Intensive Care Unit
Two patients (P3 and P4) were both symptomatic with acute respiratory illness on MICU at overlapping times between 22 – 27 March. Both were initially managed in an open shared area of the ward before being moved to single cubicles within 24 hours of admission. There were nine other patients on the ward during that period or just after that period that subsequently became infected. Figure 9 includes the timeline relevant to this period and a detailed ward layout showing the locations of the symptomatic individuals (P3 and P4) and the patients who would later develop COVID-19. It is worth noting that all areas of this ward were involved in the spread of infection.
Medical ward 1 (second wave of infections)

Medical ward 1 was reopened on 25 March (four days after closure and after deep cleaning and disinfection). Patients were moved to the reopened ward from medical ward 2 and cardiothoracic ward, which was then being converted into the COVID-19 ward. One of the patients moved from MW2 on 27 March was patient P7, an 86-year-old male with multiple myeloma and hypertension who was admitted on 26 March with bronchopneumonia. He was previously admitted to MW1 between 11-20 March when he most probably acquired COVID-19 in room 15 (Figure 7). On readmission, he was not considered to be a PUI, perhaps because his presentation with bronchopneumonia was the same as his prior admission. During his stay on MW1, he overlapped with eight other individuals who were subsequently diagnosed with COVID-19 (Figure 10). Two of these (X1 and X3) were located in the same room as P7, whereas the others were widely distributed around the ward (in rooms 1, 4 and 7).
Surgical ward 1
Patient P6, an 80-year-old male with dementia and stroke was decanted from MW1 to surgical ward 1 (SW1) on closure of MW1 on 21 March. Based on our hypothesis of the chains of transmission, he was probably exposed to SARS-CoV-2 in room 15 of MW1 during the first wave of transmission (Figure 7). On 31 March he was noted to be ‘chesty’ and hypoxic with SaO2 of 84% on room air and he died on 1 April. Two other patients on that ward at the same time (one in the same room and the other in a separate room) were subsequently diagnosed with COVID-19 during the systematic testing on 4 April (Figure 11). The one patient in the separate room (P17) is probably more likely to have acquired infection earlier on the medical ICU (Figure 9).

Surgical Intensive Care Unit
Medical ICU was closed on 31 March after recognition of the outbreak in the hospital, and apparent localisation to that ward. The patients in MICU at that time were moved to surgical ICU or cardiac ICU. One of the patients moved to SICU was P12, a 56-year-old male who had been in hospital since 2 March and on MICU since 19 March, where he was almost certainly exposed to SARS-CoV-2. He became symptomatic on 1 April shortly after transfer to SICU, but was not identified as a PUI until 3 April, following which he was moved to the cardiothoracic PUI ward. Eight other patients that overlapped with P12’s stay on SICU between 31 March and 3 April were subsequently diagnosed with COVID-19 (Figure 11). Three of those patients shared a room with P12 whereas five others were in other shared rooms or single cubicles.
Recalled patients

As part of the process of dealing with the outbreak, patients who had admissions during March and had been discharged were contacted. Symptom screening was done by phone and anyone reporting symptoms compatible with COVID-19 was asked to attend the hospital for SARS-CoV-2 testing. A total of 1892 individuals were contacted, 191 were recalled for SARS-CoV-2 testing and seven tested positive. One of these individuals was part of the NRC Berea outbreak, and another individual was the father of one of the confirmed cases (P19). He had only a short ED visit on 31 March and we believe he was more likely to have acquired infection from direct contact with his daughter. The other five cases are shown in Table 2. Three of these cases were on key wards involved in the outbreak (medical ward 1 and surgical ICU) at a time when transmission was occurring. The other two cases were both on the maternity ward in late March/early April. There were no documented cases on the maternity ward in the main outbreak. There was one staff case - a deputy nurse manager working on the maternity ward who tested positive in the systematic staff testing on 5 April. She was asymptomatic at the time of testing then became symptomatic on 8 April and was admitted that day to the COVID-19 ward. Given the timing of her positive swab and symptom onset, it is unlikely but not impossible that she was infectious at the time these two women were on the maternity ward. Based on our information, we don’t have another explanation for how these two women acquired infection on the maternity ward. It is possible that they acquired infection in the community.

Table 2 Admission details for recalled patients testing positive for SARS-CoV-2

<table>
<thead>
<tr>
<th>Age/sex</th>
<th>Dates of admission</th>
<th>Wards</th>
<th>Date of sample collection</th>
</tr>
</thead>
<tbody>
<tr>
<td>38y♂</td>
<td>23-30 March</td>
<td>SICU, MW1</td>
<td>6 April</td>
</tr>
<tr>
<td>72y♀</td>
<td>29 March-2 April</td>
<td>SICU</td>
<td>7 April</td>
</tr>
<tr>
<td>35y♀</td>
<td>25-30 March</td>
<td>MW2, MW1</td>
<td>9 April</td>
</tr>
<tr>
<td>36y♀</td>
<td>27-29 March &amp; 30 March-1 April</td>
<td>Maternity</td>
<td>11 April</td>
</tr>
<tr>
<td>36y♀</td>
<td>30 March-3 April</td>
<td>Maternity</td>
<td>13 April</td>
</tr>
</tbody>
</table>

Deaths

As of 12 May, 15 of the 39 COVID-19 patient cases had died, including one who died following discharge and readmission to Parklands Hospital and another that died following transfer to Umhlanga Hospital. The case fatality rate amongst patients was 38.5%. The median age of those who died was 79 years (IQR 69-84); nine (60%) were female. Seven died on the intensive care unit and six died on the main COVID-19 ward. To the best of our knowledge, none had post mortem examinations. Details of the cases resulting in death are shown in Table 3.

All the patients who died had significant comorbidities and most had multimorbidity (the coexistence of two or more chronic conditions). Eleven patients had hypertension, seven had diabetes mellitus, and two had cancer. None was known to be HIV positive. Four patients received antiviral therapy: P3 was treated with chloroquine, azithromycin, and oseltamivir; P4 received the same therapies but was also treated with lopinavir/ritonavir; P8 and P24 received only chloroquine. Three patients (P3, P4 and P8) were ventilated for COVID-19 disease; one other patient (P25) was on a ventilator following cardiac surgery for her atrial myxoma on 2 April until her death. In all other cases, a medical decision was taken not to intubate and ventilate because of the background comorbidities and poor prognosis.
<table>
<thead>
<tr>
<th>Code</th>
<th>Sex</th>
<th>Age</th>
<th>Admission date</th>
<th>Admission diagnosis</th>
<th>Comorbidities</th>
<th>COVID symptoms</th>
<th>Symptom onset</th>
<th>Date of death</th>
<th>Ventilated</th>
<th>Antiviral treatment</th>
</tr>
</thead>
<tbody>
<tr>
<td>P3</td>
<td>Female</td>
<td>81</td>
<td>22-Mar-20</td>
<td>Heart failure &amp; chest infection</td>
<td>DM, hypertension, stroke, depression</td>
<td>Yes</td>
<td>13-Mar-20</td>
<td>02-Apr-20</td>
<td>Yes</td>
<td>Yes⁶</td>
</tr>
<tr>
<td>P4</td>
<td>Female</td>
<td>46</td>
<td>21-Mar-20</td>
<td>Asthma</td>
<td>Obesity, hypertension</td>
<td>Yes</td>
<td>23-Mar-20</td>
<td>31-Mar-20</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>P6</td>
<td>Male</td>
<td>80</td>
<td>14-Mar-20</td>
<td>Stroke</td>
<td>DM, hypertension, hyperlipidaemia, Parkinson’s Disease, dementia</td>
<td>Yes</td>
<td>31-Mar-20</td>
<td>01-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P7</td>
<td>Male</td>
<td>86</td>
<td>26-Mar-20</td>
<td>Pneumonia</td>
<td>Multiple myeloma, hypertension, arthritis</td>
<td>Yes</td>
<td>25-Mar-20</td>
<td>03-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>X1</td>
<td>Male</td>
<td>60</td>
<td>25-Mar-20</td>
<td>Anaemia</td>
<td>Prostate cancer, hypertension</td>
<td>Yes</td>
<td>02-Apr-20</td>
<td>06-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P13</td>
<td>Male</td>
<td>70</td>
<td>29-Jan-20</td>
<td>Elective admission for spinal surgery</td>
<td>Cardiac disease, permanent pacemaker</td>
<td>Possibly⁵</td>
<td>07-Apr-20</td>
<td>07-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P29</td>
<td>Female</td>
<td>71</td>
<td>06-Apr-20</td>
<td>Pneumonia</td>
<td>End-stage renal disease, hypertension, DM, obesity</td>
<td>Yes</td>
<td>05-Apr-20</td>
<td>10-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P14</td>
<td>Male</td>
<td>91</td>
<td>14-Mar-20</td>
<td>Respiratory tract infection</td>
<td>Dementia, Parkinson’s disease, DM, hydrocephalus (VP shunt), Stokes-Adams syndrome</td>
<td>Possibly⁵</td>
<td>10-Apr-20</td>
<td>12-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P25</td>
<td>Female</td>
<td>72</td>
<td>20-Mar-20</td>
<td>Atrial myxoma</td>
<td>Asthma, hypertension, hyperlipidaemia</td>
<td>Possibly⁵</td>
<td>10-Apr-20</td>
<td>15-Apr-20</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>P13</td>
<td>Female</td>
<td>79</td>
<td>23-Mar-20</td>
<td>Cellulitis right leg</td>
<td>DM, hypertension, hyperlipidaemia, gastric ulcer, cardiac disease</td>
<td>Yes</td>
<td>03-Apr-20</td>
<td>13-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P16</td>
<td>Female</td>
<td>86</td>
<td>12-Mar-20</td>
<td>Posterior circulation stroke</td>
<td>DM, hypertension, IHD, CHF, AF</td>
<td>Possibly⁵</td>
<td>06-Apr-20</td>
<td>11-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P24</td>
<td>Male</td>
<td>79</td>
<td>30-Mar-20</td>
<td>Colon cancer (for elective colectomy)</td>
<td>Hypertension</td>
<td>Yes</td>
<td>05-Apr-20</td>
<td>17-Apr-20</td>
<td>No</td>
<td>Yes⁵</td>
</tr>
<tr>
<td>P25</td>
<td>Female</td>
<td>89</td>
<td>13-Dec-19</td>
<td>Sacral decubitus ulcer</td>
<td>Dementia</td>
<td>Yes</td>
<td>14-Apr-20</td>
<td>20-Apr-20</td>
<td>No</td>
<td>No</td>
</tr>
<tr>
<td>P8</td>
<td>Female</td>
<td>67</td>
<td>01-Apr-20</td>
<td>Pneumonia</td>
<td>Hypertension, myasthenia gravis</td>
<td>Yes</td>
<td>01-Apr-20</td>
<td>21-Apr-20</td>
<td>Yes</td>
<td>Yes⁵</td>
</tr>
<tr>
<td>P17</td>
<td>Female</td>
<td>48</td>
<td>12-Jan-20</td>
<td>Pneumonia</td>
<td>DM, end-stage renal disease</td>
<td>Yes</td>
<td>14-Apr-20</td>
<td>05-May-20</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

AF, atrial fibrillation; CHF, chronic heart failure; DM, diabetes mellitus; IHD, Ischaemic heart disease

⁶ Noted to be asymptomatic 14-Apr morning ward round with normal respiratory rate, saturation, LUNG function normal. Connection with COVID-19 unclear

⁵ Symptomology unclear: hypothermic 9-Apr (T 33°C); desaturates (SaO₂ 66% room air) and hypotensive on 10-Apr; decision taken not to escalate care. Relationship between COVID-19 and death unclear


⁵ Admitted from nursing home unresponsive; on transfer to COVID-19 ward 6-Apr GCS 7-8, noted to be hypoxic (SaO₂ 82% room air), placed on oxygen, decision taken not to intubate/ventilate

⁶ Chloroquine, azithromycin, oseltamivir

⁷ Chloroquine, azithromycin, oseltamivir, lopinavir/ritonavir

⁸ Chloroquine
Health care worker infections
As of 12 May, 1711 staff had been tested at least once with a SARS-CoV-2 PCR (approximately 86% of all staff). A total of 80 HCWs had a positive test (~5% of all staff tested). Most staff with COVID-19 (89%) were female and the median age was 39 years (IQR 33-46). Most (78%) were nurses or nursing students (Table 4); of the nurses, 47 were Netcare employees and nine were agency nurses. The nurses and nursing students were predominantly from the wards most affected by the outbreak, in particular MICU and MW1 (Table 5). It is noteworthy that no HCW infections were detected from the COVID-19 ICU (surgical ICU 2), which might be considered the highest risk area of the hospital. This could reflect that patients may be less infectious by the time they are admitted to ICU with COVID-19 or suggests that where the risk of transmission is recognised, appropriate care, attention and use of PPE can limit infections in health care workers.

Table 4 Cadre of staff testing positive for SARS-CoV-2

<table>
<thead>
<tr>
<th>Staff category</th>
<th>n</th>
<th>%</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nurse</td>
<td>56</td>
<td>70.0</td>
</tr>
<tr>
<td>Student nurse</td>
<td>6</td>
<td>7.5</td>
</tr>
<tr>
<td>Cleaner (Tsebo)</td>
<td>4</td>
<td>5.0</td>
</tr>
<tr>
<td>Physiotherapist</td>
<td>3</td>
<td>3.8</td>
</tr>
<tr>
<td>Kitchen staff</td>
<td>3</td>
<td>3.8</td>
</tr>
<tr>
<td>Doctor</td>
<td>2</td>
<td>2.5</td>
</tr>
<tr>
<td>Others</td>
<td>6</td>
<td>7.5</td>
</tr>
</tbody>
</table>

Table 5 Primary ward for nursing staff (and nursing students) testing positive for SARS-CoV-2

<table>
<thead>
<tr>
<th>Ward</th>
<th>n</th>
<th>%</th>
<th>Earliest case</th>
</tr>
</thead>
<tbody>
<tr>
<td>Medical ICU</td>
<td>18</td>
<td>29.0</td>
<td>27 March</td>
</tr>
<tr>
<td>Medical ward 1</td>
<td>9</td>
<td>14.5</td>
<td>31 March</td>
</tr>
<tr>
<td>Neurology</td>
<td>6</td>
<td>9.7</td>
<td>29 March</td>
</tr>
<tr>
<td>Surgical ICU</td>
<td>6</td>
<td>9.7</td>
<td>03 April</td>
</tr>
<tr>
<td>Cardiothoracic</td>
<td>4</td>
<td>6.5</td>
<td>03 April</td>
</tr>
<tr>
<td>Neonatal ICU</td>
<td>3</td>
<td>4.8</td>
<td>16 April</td>
</tr>
<tr>
<td>Cardiac ICU</td>
<td>2</td>
<td>3.2</td>
<td>18 March</td>
</tr>
<tr>
<td>Medical ward 2</td>
<td>1</td>
<td>1.6</td>
<td>03 April</td>
</tr>
<tr>
<td>Surgical ward 2</td>
<td>1</td>
<td>1.6</td>
<td>20 April</td>
</tr>
<tr>
<td>Surgical ward 3</td>
<td>1</td>
<td>1.6</td>
<td>04 April</td>
</tr>
<tr>
<td>Surgical ward 4</td>
<td>1</td>
<td>1.6</td>
<td>31 March</td>
</tr>
<tr>
<td>Other units</td>
<td>9</td>
<td>14.5</td>
<td></td>
</tr>
</tbody>
</table>

Fourteen of the infected health care workers were admitted to the COVID-19 ward at St. Augustine’s Hospital for clinical care. None was admitted to the intensive care unit. All have subsequently recovered. No death has been recorded in a health care worker.

Linked outbreaks in other institutions
Bill Buchanan Association for the Aged
On 16 April, patient P3 was discharged from St. Augustine’s Hospital to the Bill Buchanan Association for the Aged in Morningside, a nursing home with 210 residents. She was there until her readmission to St. Augustine’s on 22 March. By 31 March, we understand that four other residents at the home were diagnosed with COVID-19, including three women who had shared the sick bay with P3 and
one woman who stayed in a separate unit and only visited P3 (as they were friends from their residential home). One of the cases from the sick bay was admitted to St. Augustine’s on 31 March (patient P10) - she completed 14 days of isolation, had a negative follow-up SARS-CoV-2 PCR and was discharged back to the home on 15 April. The nursing home managed to isolate the other cases in a separate building and no staff members at the home tested positive. We hypothesise that P3 introduced the infection into the nursing home and the virus was transmitted to the four other residents.

**National Renal Care dialysis unit**

National Renal Care operates a 39-bed outpatient dialysis unit (NRC Berea) on the St. Augustine’s Hospital campus. NRC also through a separate team provides inpatient dialysis services for St. Augustine’s Hospital. A total of 19 confirmed cases (11 patients and eight staff) have been identified within the NRC outpatient dialysis services. We hypothesise that two patients (P29, X4) acquired COVID-19 infection whilst they were inpatients on MICU in late March (Figure 9) and then, on resuming outpatient dialysis care, introduced the infection into the outpatient dialysis unit on either 1 April or more likely 3 April. An epidemiological curve specifically for the NRC dialysis unit is shown in Figure 13.

![Epidemiological curve for outbreak of COVID-19 in National Renal Care Berea dialysis unit April 2020](image)

**Figure 13 Epidemiological curve for outbreak of COVID-19 in National Renal Care Berea dialysis unit April 2020**

P29, a 72-year-old female with diabetes mellitus, hypertension and end-stage renal failure was admitted to MICU from 25 - 28 March, at a time of active SARS-CoV-2 transmission (Figure 9) and then resumed outpatient dialysis on 1 April. She developed symptoms of COVID-19 on 5 April and tested positive on 6 April. X4, a 60-year-old male on chronic haemodialysis was admitted to MICU from 29 - 31 March and then resumed outpatient dialysis on 3 April. He presented as an emergency to Kingsway Hospital with fever and dyspnoea on 5 April and tested positive for SARS-CoV-2.

One other patient (P30) from the NRC dialysis unit had been an inpatient at St. Augustine’s in late March. He was admitted through the emergency department to the COVID-19 PU1 ward on 27 March with fever and a ruptured arteriovenous fistula. His SARS-CoV-2 PCR test was negative, he was discharged on 30 March, and resumed chronic haemodialysis in the outpatient unit on 1 April. On 7 April he developed fever and dyspnoea, tested positive for COVID-19 on 8 April and was readmitted to St. Augustine’s on 9 April. Although it’s possible he acquired infection during his inpatient admission, we believe it’s more likely he acquired infection in the dialysis unit.

In response to these three cases, all patients (n=133) and staff (n=36) linked to the unit were tested for SARS-CoV-2. Eight other patients tested positive, six of who were from a group of 32 who
underwent routine haemodialysis at the same sessions as P29 on 1 April and P29 & X4 on 3 April. Four of these were admitted to hospital with COVID-19 symptoms. As of 12 May, five of the eleven patient cases linked to the dialysis unit had died (case fatality rate 45.5%).

Eight staff members tested positive for SARS-CoV-2 (22% of those tested). All but one of the staff cases had symptoms at the time of testing or developed symptoms after testing. Two were admitted to hospital. None has died.

DNA sequencing and phylogenetic analysis

As of 20 April, we have sequenced SARS-CoV-2 RNA extracted from 18 samples retrieved from Ampath Laboratories and the IALCH NHLS Laboratory, using the Illumina platform. This includes eight samples from inpatients (P3, P5, P6, P7, P11, P12, P13 & P14), one sample from one of the nursing home residents (NH1) and nine from health care workers (HW4, HW5, HW9, HW11, HW13, HW30, HW32, HW33 & HW49). Health care workers’ codes were based on a numerical sequence according to the date of sample collection. In addition, we sequenced SARS-CoV-2 RNA from five other samples from confirmed COVID-19 cases in eThekwini not known to be linked to the St. Augustine’s outbreak.

The 23 SARS-CoV-2 genome sequences were analysed in a phylogenetic framework on the NextStrain platform (https://nextstrain.org), to ascertain their genetic relatedness with one another and with other sequences from around the world. In short, NextStrain allows for the analysis of pathogen whole genome sequences – like SARS-CoV-2 – along with available clinical, demographic and geographic data. The underlying methods applied include: 1) sequence alignment, 2) tree and molecular clock inference, 3) ancestral state reconstruction and 4) tree annotation. We selected 32 reference sequences from the GISAID SARS-CoV-2 sequence repository (https://www.gisaid.org) to analyse alongside our sequences. These reference sequences includes the two genomes derived from the earliest samples in Wuhan, China, as well as 30 sequences representing the major lineages of SARS-CoV-2.

A phylogenetic tree is shown in Figure 14. All of the 18 sequences of the patients and health care workers clustered closely together in the A2a clade, which has been identified in cases from Europe or people that have travelled from Europe. These sequences were highly identical, i.e. >99.99%. Six sequences (including the sequence from P3) were basal in the phylogenetic tree, consistent with those patients acquiring infection early in the outbreak. There were one or two additional mutations in the sequences from the other four patients and eight health care workers, consistent with them being in the later wave of infections on MICU and MW1. The appearance of one to two mutations is consistent with a month-long nosocomial outbreak as the virus mutation rate in the population is approximately two mutations per month (1.8 x 10³ substitutions per site in the genome per year)\(^1\).

The phylogenetic tree also shows that the all of the five control sequences from eThekwini (KRISP_002, KRISP_004, KRISP_006 and KRISP_012 & KRISP_045) did not cluster with the hospital infections. For example, the KRISP_004, KRISP_012 and KRISP_045 sequences clustered with other European sequences in the A2a European clade, suggesting a distinct introduction of SARS-CoV-2 to eThekwini from Europe. The KRISP_002 sequence clustered with a sequence from New York and the KRISP_006 sequence clustered with two Australian sequences. This suggests that there were

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multiple introductions from overseas to eThekwini, but that a unique introduction to the hospital was most likely to have been responsible for the nosocomial outbreak.

![Figure 14 Phylogenetic tree showing 18 closely related SARS-CoV-2 sequences from St. Augustine’s outbreak and five other sequences from eThekwini](image)

**Discussion**

**Main hypothesis for introduction and spread of SARS-CoV-2**

We present evidence that suggests there was a single introduction of SARS-CoV-2 into St. Augustine’s Hospital followed by widespread transmission to patients and health care workers, with several consecutive waves of infections localised to specific wards. Our hypothesis is supported by phylogenetic analysis. We provide a plausible explanation for how the virus was introduced into the hospital, by transmission between a symptomatic case and a susceptible patient in the Emergency Department on 9 March, but as we haven’t obtained the sample from the ED case for sequencing, we can’t exclude other routes of introduction.

Only one inpatient case cannot be explained by the hypothesis presented here and does not seem to be a nosocomially-acquired infection. Patient P9 is a 44-year-old male living with HIV who was admitted with a three month history of cough. He was diagnosed with pulmonary TB on the basis of a positive Xpert MTB/Rif Ultra test. He tested positive for SARS-CoV-2 on admission on 1 April. He had not travelled recently, had no known exposure to a COVID-19 case and had no other recent hospitalisation. He clinically improved with anti-TB treatment. Two repeat swabs (on 3 April and 5 April) were both negative on PCR for SARS-CoV-2. Whilst it is possible that he had community-acquired COVID-19, we believe it is more likely that the initial SARS-CoV-2 PCR result was a false positive.
Other than that case and P10, who we believe acquired infection in the nursing home, there is strong evidence that all other cases in the St. Augustine’s Hospital inpatients in March and April were nosocomially acquired infections. Excluding P9, P10 and the three ED cases, the number of nosocomially acquired infections in hospitalised individuals would therefore be 34. With the four cases acquired in the nursing home and nine cases acquired in the outpatient dialysis unit, 47 cases in total were linked to the nosocomial outbreak. In addition, up to 88 infections in staff (80 St. Augustine’s staff and eight NRC Berea staff) may have been nosocomially acquired during this outbreak. This total of up to 135 cases associated with the outbreak would represent approximately 14% of all cases reported in KwaZulu-Natal up to 30 April.

We provide a plausible explanation for how the infection entered the hospital, by transmission between two patients (P1 and P3) in the Emergency Department. We cannot exclude the possibility that patient P3 acquired COVID-19 in the community prior to the initial admission on 9 March. We found no evidence that she had been in contact with anyone with COVID-19 or with anyone that had recently travelled. All three confirmed cases in the country prior to this date were imported infections; in fact, the first case of local transmission in South Africa was not reported until 13 March, four days after her admission. Therefore, we consider the likelihood of her having been exposed to SARS-CoV-2 prior to her admission on 9 March to be extremely low.

We considered whether the CICU nurse (first staff case) could have been the source of infection for P3. However, the nurse’s symptoms started five days after last contact with P3 (and four days after the onset of fever in P3). The timings of symptom onset therefore make this direction of transmission implausible.

We considered whether P3 could have acquired COVID-19 in the Bill Buchanan Association for the Aged in the period between the first and second admission (16-22 March). However, if that were the case, we have no plausible explanation for the first health care worker infection (CICU nurse), no other clear source of the first wave of infections on MW1, and the timing would mean an extremely rapid incubation period and disease progression (maximum six days from exposure until severe pneumonia requiring intubation and ventilation). Putting all those together, we therefore believe this explanation to be extremely unlikely.

We also considered the possibility that patient P4 acquired COVID-19 in the community and brought the infection into the hospital on her first admission on 7 March, and that the virus was then transmitted to P3 and to the other patients on MW1. Again, however, we believe this to be implausible given that prior to 7 March there were only two reported cases in the country and there was no documented local transmission of SARS-CoV-2. This would also leave the source of the first health care worker’s infection (CICU nurse) unexplained.

Likely mode of transmission and factors contributing to outbreak

According to current evidence, SARS-CoV-2 is transmitted between people through respiratory droplets and contact routes. Droplet transmission may also occur through fomites so transmission of the virus can occur by direct contact with an infected person or indirect contact with surfaces in the immediate environment of that person or with objects used on the infected person (e.g. stethoscope or thermometer). The spatial distribution of cases and exposed individuals who became infected on the wards suggests that indirect contact via health care workers or fomite transmission were the predominant modes of transmission between patients in this outbreak. Direct droplet or

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contact transmission would be plausible where the people that were exposed were located in close proximity to an infectious case, e.g. P4 in the bed directly opposite P3 on MW1 between 13 - 16 March (Figure 7); or X1 and X3 sharing a four-bedded bay with P7 on MW1 between 27 March - 2 April (Figure 10). However, in other examples the exposed individuals were located in different rooms and different areas of the ward, making indirect contact via health care workers or fomite transmission more plausible. We also present evidence suggestive of direct droplet transmission from a symptomatic health care worker to two patients on the neurology ward.

There continues to be debate around whether aerosol transmission is important in the spread of SARS-CoV-2. Aerosol transmission may be possible in specific circumstances and settings in which aerosol-generating procedures are performed (e.g. endotracheal intubation and manual ventilation before intubation). There is no evidence to suggest aerosol transmission contributed to the outbreak at St. Augustine’s Hospital. In fact we were struck by the observation that a rushed intubation and ventilation of P3 on 22 March where several health care workers were not wearing appropriate PPE, and therefore a high risk exposure, did not result in any health care worker infections (all HCWs involved did not develop symptoms and had at least one negative SARS-CoV-2 PCR). We therefore believe that the current PPE recommendations for health care workers caring for people with COVID-19 are appropriate.

Under our hypothesis, there may have been two transmission events suggestive of presymptomatic transmission. The transmission from P3 to HCW1 occurred on 12 March in CICU the day before the onset of fever in P3. Similarly the transmission from P29 and/or X4 in the outpatient dialysis unit seemed to occur prior to the onset of symptoms in either case. Whilst all the other patient infections can be explained by exposure to a symptomatic individual, we cannot exclude the possibility that other transmissions occurred in the presymptomatic phase.

One of the striking observations when reviewing the timeline of cases was the frequent movement of patients between and within wards. It seems this partly reflects the timing as the hospital was preparing for COVID-19 (i.e. emptying wards to repurpose them as COVID-19 wards) and then responding to the outbreak as it evolved. It seems likely that this movement of patients around the hospital facilitated the spread of the virus by bringing unsuspected cases into contact with new groups of susceptible patients and health care workers. One such example was the closure of MW1 on 21 March to prepare that ward as a COVID PUI ward. On closure, P4 was decanted to the neurology ward and may have exposed two other people on that ward whilst she was symptomatic and infectious. Another example was the closure of MICU on 31 March in response to the outbreak, where P12 was moved to SICU, developed symptoms and may have exposed up to eight other people on that ward.

Of all the infections in nursing staff (including nursing students), 63% (39/62) were working on medical ICU, medical ward 1, neurology ward or surgical ICU. These four wards were the wards where unsuspected cases were managed and where the majority of patients acquired infection. On those general wards at those times standard precautions were being used. This was consistent with guidelines from National Department of Health and NICD at the time, and remains consistent with current guidelines1. As patients such as P3 and P4 were not initially recognised as high risk for COVID-

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19, it seems that isolation was delayed and droplet and contact precautions were not instituted until there may have been significant numbers of exposures amongst patients and health care workers.

Factors associated with patient deaths
A detailed analysis of the factors contributing to death is beyond the scope of this report. It is clear that deaths occurred predominantly in elderly inpatients with significant comorbidities. Some patients had complex medical problems and had been in hospital for a prolonged period of time before acquiring SARS-CoV-2 infection. It was noted that experimental therapies including chloroquine, azithromycin and lopinavir/ritonavir were given to some cases but no conclusions can be drawn as to whether these had any effect on the outcomes. It is worth noting that there is currently no evidence to support any specific therapy for COVID-19⁹. Based on review of the medical records, the decisions not to intubate and ventilate some of the affected individuals was appropriate.

Missed opportunities for earlier action
By the time the first inpatient COVID-19 infection was confirmed in P3 on 25 March, the outbreak was already at an advanced stage with up to 16 patients already exposed to SARS-CoV-2. With the benefit of hindsight, there are a number of points at which different actions could potentially have influenced the progression of the outbreak.

1. On the 13 March, patient P3 developed a fever that in retrospect may have been the first symptom of COVID-19. Our understanding from review of the medical records and discussion with the physician is that no specific cause for that fever was identified. If the possibility of COVID-19 had been considered at that time, then investigation might have uncovered the potential exposure to P1 in the ED on 9 March and might have led to SARS-CoV-2 PCR testing. However, the fever was present only on 13 March and did not persist on 14-16 March, and no respiratory symptoms were documented at that time. The patient would not have met the criteria for PUI given the apparent lack of contact history. It should also be noted that fever post-stroke is common and can have many causes.

2. On 21 March P4 was readmitted to MW1 with an acute respiratory illness five days after discharge from the same ward. It seems that the presentation on both admissions was similar with cough and dyspnoea and this was attributed on both admissions to an acute exacerbation of asthma. Fever was not present on admission and developed later on 23 March. It seems that a chest X-ray was not performed immediately on readmission and actually was not done until 25 March, at which time it showed diffuse bilateral air space opacification. If chest X-ray had been done at the time of readmission and had been abnormal then there might have been a higher index of suspicion of COVID-19. This case could then have been confirmed and isolated earlier and investigation of the potential sources of the infection and tracing of potentially exposed patients and staff members could have begun sooner.

3. On 22 March P3 was readmitted from the nursing home to medical ICU acutely unwell with clinical and radiological signs of severe pneumonia. There was then a high index of suspicion of COVID-19 and swabs were appropriately sent for SARS-CoV-2 PCR testing (despite the advice from the NICD hotline being that she did not meet the criteria for investigation and should not be tested). Due to the initial indeterminate PCR result (on oropharyngeal swab)

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and the need for the sample to be sent for confirmatory testing and for an endotracheal aspirate sample to be processed for testing, it was three days until the positive result was received. In those three days, although she was considered highly likely to have COVID-19 and was managed accordingly, there was no thorough investigation to review the first admission, to identify potential sources of infection, and to identify people who might have been exposed to her whilst she was infectious. This action did not happen until after the positive result was received on 25 March.

4. On 23 March the first staff case was confirmed (symptom onset 17 March, swabs collected 18 March). This staff member had accessed testing through their GP, but reported the positive test result appropriately to her unit manager on 23 March. The unit manager then communicated the result to the occupational health nurse and to the nursing manager on the same day (23 March). It seems that this first documented infection in a staff member did not trigger any specific investigation or action. If that case had been recognised as important at the time then a thorough investigation could have been performed to uncover possible sources of infection within the hospital. This would have uncovered the link to P3 on the night shift of 12-13 March and, given that by 23 March P3 had been readmitted with a severe pneumonia this could have raised the suspicion of nosocomial SARS-CoV-2 transmission.

The main underlying issue that runs through all these points is that the first inpatient cases of COVID-19 were not suspected to have COVID-19 as they did not fit the typical profile of the cases being detected in the country at that time, and had no clear risk factors for community-acquired infection. As there were no COVID-19 patients being treated in the hospital, the risk of nosocomial acquisition of COVID-19 would understandably have been considered to be very low. Consequently the index of suspicion for COVID-19 in these first cases was low and this contributed to the delayed diagnosis and delayed recognition of the outbreak.

In terms of the NRC outpatient dialysis unit, the outbreak there highlights the importance of being proactive rather than reactive when the risk of COVID-19 being introduced is apparent. By 1 April when P29 resumed attendance for outpatient dialysis, and certainly by 3 April when X4 also resumed outpatient dialysis, the outbreak in the main hospital was well established and it was particularly apparent that MICU had been a hotspot of transmission. Both patients should therefore have been considered to have had high risk exposures and strategies could have been put in place to minimise the risk of transmission within the dialysis unit. This could have involved physical separation of P29 and X4 from other patients (possibly using temporary home dialysis care), and appropriate use of PPE by health care workers attending to those patients.

Limitations

We have presented basic information on the infections in health care workers and other staff, and have shown that the spatial distribution of HCW infections correlates with the wards where most patient infections were acquired. We obtained detailed information on some of the first staff cases to help us understand the chains of transmission. However, we have not been able to interrogate all the health care worker data fully to understand where precisely people worked on specific wards, whether there was any direct contact with cases, and whether people were symptomatic (or presymptomatic) whilst working. We also don’t know exactly how much movement there was of health care workers between different wards during this period. This means that at present we don’t know to what extent direct health care worker to patient transmission contributed to the outbreak and to what extent redeployment of HCWs to different wards might have facilitated the spread through the hospital. Similarly we don’t know to what extent transmission occurred between staff,
either in the workplace or outside in shared transport and accommodation. We don’t know if some of the infections in staff may have been acquired in the household or community. Although this is less likely for the earlier infections, it would be plausible for some of the later infections that were acquired once there was established community transmission in the local area.

We haven’t followed up the health care worker testing information to explore the outcomes of contact tracing and identify how many secondary infections can be linked to the hospital outbreak, as this was not within our remit. The contact tracing has been coordinated by the Communicable Disease Control Programme of the KwaZulu-Natal Department of Health. Clearly this information is important to understand to what extent the nosocomial transmission may have been an amplifier of transmission in the province. The other specific issue that there is real concern about in terms of contact tracing is the issue of nurses employed in the Department of Health who do agency work at St. Augustine’s Hospital. The concern relates to the possibility that they become infected and then could transmit the virus to patients and colleagues at the Department of Health facility. Although eight agency nurses tested positive, we don’t have full details of those cases and have not been able to ascertain whether there has been any risk of spreading the infection to other health facilities.

We did not have the opportunity to review the results of PCR testing of environmental samples from the hospital. Environmental sampling was done late in the outbreak and was done in a limited number of areas and was not systematic so it’s not clear how helpful this information would have been. We also haven’t fully interrogated the cleaning and disinfection records to understand whether there were gaps and weaknesses in environmental cleaning that may have contributed to transmission on specific wards.

We have not yet obtained sequences from all the samples related to the outbreak. We have received additional samples from Ampath and IALCH NHLS Laboratory and further sequencing will be done.

Action taken in response to the outbreak
Since recognising the outbreak, the hospital implemented a number of measures to contain the outbreak, limit the spread of infection into the community and get the hospital prepared for reopening. Some of the key steps taken are detailed below:

- EThekwini Department of Health were notified of the first confirmed case on 25 March. The Chief Director visited the hospital on the evening of 25 March and then again with a delegation on 27 March. A process of contact tracing and staff screening and testing began on 27 March, initially focused on medical ICU
- On confirmation of the second case on 26 March, the Nursing Manager contacted the Netcare Group Medical Director and Netcare’s National Joint Operations Command (JOC) in Johannesburg. The Group Medical Director immediately informed the NICD and sought the advice and guidance of Dr Kerrigan McCarthy, Pathologist: Division of Public Health Surveillance and Response at the NICD. The advice received was:
  - The patients who had shared a room with the COVID-19 positive patients were not considered contacts as they were more than 1m away from the patients in question
  - Nurses and doctors who performed aerosolizing procedures but who had worn adequate PPE were required to self-monitor and only those who had not worn adequate PPE were required to home quarantine. All exposed healthcare workers were advised to only be tested should symptoms develop
  - The ED visit of both confirmed cases had to be scrutinized to understand the origin of the COVID-19 inoculation into the hospital
On 27 March, the cardiothoracic ward was repurposed as a COVID-19 ward, with separate sections for PUIs and confirmed cases. At the same time, the surgical ICU 2 was designated as the COVID-19 ICU.

The hospitals’ emergency department was closed on the night of 2 April and from 3 April at midday all doctors’ rooms were closed and no further admissions were taken to the hospital. Theatre cases were cancelled as of 3 April.

Screening of all people (including staff) entering the facility on a daily basis was instituted, and any person reporting symptoms is sent immediately for testing.

All persons entering the facility must wear a surgical mask.

Testing of all staff (both Netcare and support staff) regardless of symptoms began on 2 April. As of 12 May, 1711 staff members had been tested at least once.

All staff testing positive and requiring admission were admitted to the COVID-19 ward at the hospital. Those not requiring admission were allowed to self-isolate at home but if their home circumstances were not suitable for self-isolation they were admitted to dedicated isolation facilities.

A process to completely decontaminate and disinfect the entire hospital through deep cleaning began on 2 April. As an additional precaution terminal cleaning using a high dosage of chlorine, followed by disinfection with the aid of ultraviolet-C (UV-C) disinfection robots was performed.

All patients who were treated at the emergency department or admitted into the hospital as from 1 March were contacted and screened for symptoms – anyone reporting symptoms was recalled for SARS-CoV-2 PCR testing. Attempts were made to contact 3516 individuals and 1892 (54%) were successfully contacted. Of those, 191 were recalled for testing and seven tested positive.

The hospital has been split into red zones (patients with confirmed COVID-19), yellow zones (persons under investigation) and green zones (low risk patients).

A new plan has been developed for a separate triage and admission area for the red and yellow zones. These individuals will be directed to a separate hospital entrance and PUI will be triaged in an appropriate isolation area.

As of 30 April, the hospital was completely closed. All remaining inpatients (including COVID-19 cases) were transferred to other hospitals.

The NRC outpatient dialysis unit has also taken specific measures in response to the outbreak. As soon as it was realised that the dialysis unit had become involved in the hospital outbreak, the chronic haemodialysis patients were cohort into three groups:

- Group 1 - All patients on chronic HD that were admitted to St. Augustine’s at any time from 1 - 31 March. These were considered patients with potential exposure.
- Group 2 - All patients dialyzed at the same sessions as P29 and X4 on 1 and 3 April
- Group 3 - All patients with confirmed COVID-19
- Group 4 - All other patients that don’t belong to group 1, 2 or 3

Each group is now being dialyzed at different times with appropriate environmental cleaning between dialysis shifts. Patients in group 3 have either been admitted to hospital to receive inpatient dialysis services or are self-isolating at home and receiving home dialysis care.
Lessons learnt
The primary lesson learnt from this outbreak is that SARS-CoV-2 can spread very rapidly in the hospital environment, highlighting the need for strong infection prevention and control systems and practices throughout the hospital. Presymptomatic transmission of SARS-CoV-2 presents a particular challenge in the hospital setting and means that the safest approach is for health care workers to assume that all patients are potentially infectious with COVID-19 and to take appropriate precautions on that basis.

Early recognition and isolation of patients with suspected COVID-19 is essential to prevent or limit transmission in healthcare settings. This outbreak highlights the consequences of delayed recognition in hospital inpatients. All health care workers should have a high index of suspicion and now that there is more widespread community transmission and the PUI criteria have been simplified, all patients with an acute respiratory illness should be suspected to have COVID-19 and investigated accordingly. Health care workers should be alert to symptoms developing in people already in hospital, and should be particularly vigilant in green zones where people thought to be at low risk of COVID-19 are.

The different cadres of staff who were infected highlights that all staff working in hospitals are at risk of infection, even if not involved in frontline patient care. Whilst we cannot be sure that all staff were infected in the hospital and not in the community, this emphasises the importance of general measures such as hand hygiene and physical distancing for all staff in the hospital environment.

The hypothesis around the initial transmission event in the emergency department emphasises the need for the COVID-19 isolation and triage area to be completely separate from other patient care areas (with a separate entrance and exit) to limit the potential for transmission. This also allows for a rethink around the organisation of the hospital and an opportunity to follow best practice from other settings1.

There is no evidence that aerosol transmission contributed to this nosocomial outbreak. Current PPE guidance therefore remains appropriate, particularly around the use of masks8. Health care workers performing an aerosol-generating procedure on a COVID-19 suspect or case should wear N95 masks. The focus on type of masks provided to health care workers and the anxiety around mask use detracts from the importance of good mask etiquette, particularly not touching the front of the mask, and from all other components of IPC.

Hand hygiene remains the most important intervention to prevent transmission of SARS-CoV-2 inside and outside hospitals. The pattern of spread within units in this outbreak suggest that contact and fomite transmission may have been the most common modes of transmission. It has to be emphasised at all times that hand hygiene is more important than wearing gloves.

Environmental hygiene is also critical. The evidence suggests that the SARS-CoV-2 can survive for hours or even days on different surfaces, so it’s very likely that the environment around a patient with COVID-19 will be contaminated. This emphasises the need for regular cleaning and disinfection, especially of high contact surfaces. If cleaning is not appropriately checked and validated by


supervisors then there is a high likelihood that cleaning efforts will be insufficient, which will then allow continued virus transmission. An additional concern, although we have no evidence to support this, is that the over reliance on the UV-C robots could reduce the quality of the initial cleaning and disinfection.

Given the potential role of fomite transmission in hospitals, particular attention needs to be paid to cleaning and disinfection of medical equipment such as stethoscopes, thermometers and other patient care items1.

**Recommendations**

We have a number of recommendations for the hospital and for Netcare to consider in preparation for reopening of the hospital and to reduce the risk of further outbreaks of COVID-19 or of other respiratory virus infections. Many of the recommendations come back to good infection prevention and control practiceii,iii, and some have already been put in place or are currently in development. These recommendations also have broader applicability to other private and public health facilities in South Africa:

- The management should promote a culture where infection prevention and control is everyone’s responsibility and make clear that everyone has an important role to play. This must include all Netcare employees, doctors and other support staff working in the facility
- Training or re-training on COVID-19, with a specific focus on infection prevention and control, should be mandatory for all staff, both Netcare employees, doctors and support staff working in the facility. There should be documentation that all staff have completed training before reopening of the hospital.
- Outdoor screening stations should continue to operate at all entry points to the hospital
- Continue with the practice of all persons inside the hospital building wearing a surgical mask. Ensure daily reminders to all staff and patients around mask etiquette
- The hospital should be separated into red zones (for confirmed COVID-19 cases), yellow zones (for persons under investigation) and green zones (for people at low risk of COVID-19). These zones must be clearly marked with signage. Each transition point between zones must also be clearly delineated using signage, doors and floor markings. Each transition zone should include prominently posted descriptions of the steps to be taken when in that zoneiv.
- If at all possible, separate routes through the hospital to the red and yellow zones need to be delineated so as to avoid contact with the green zones
- Separate Emergency Department entrances leading to i) the green zones and ii) the red and yellow zones need to be constructed, with easy access to appropriate assessment and triage areas

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• Reorganise shared rooms in green zones to ensure physical distancing – beds should be more than 2 metres apart and consider installing partitions
• Limit movement of patients between different wards for non-clinical reasons
• Limit non-essential movement of staff between different units
• Systematic daily symptom screening of inpatients in green zones should be instituted – any patient with documented fever (>38.0°C) or reporting cough, sore throat, or dyspnoea should be tested for SARS-CoV-2.
• If rapid PCR testing is provided on site (e.g. Xpert Xpress SARS-CoV-2), samples from inpatients on green zones should be prioritised for testing with rapid turnaround
• Consider establishing routine weekly SARS-CoV-2 PCR testing of staff, regardless of symptoms
• Promote physical distancing for patients and staff throughout the hospital. This can be promoted by floor markings, markings on seats, reorganisation of common areas etc.
• Encourage physical distancing of staff outside work – consider providing or incentivising safe means of travel to and from work to limit time in overcrowded transport
• Continually reinforce messages around good hand hygiene based on the WHO ‘My 5 Moments for Hand Hygiene’
• Disseminate clear, simple messages about infection prevention and control to all staff through multiple channels (e.g. posters on wards, daily text message reminders)
• Each ward should appoint a hand hygiene champion, who should be the role model and should monitor hand hygiene practices on that ward
• Encourage all staff to monitor each other for improper use of PPE, hand hygiene etc.
• All frontline staff should be reminded of the potential role of fomite transmission and should be reminded to clean and disinfect surfaces regularly, and to clean and disinfect medical equipment and patient care items between patients
• Engage with management of contract cleaning company to ensure that environmental cleaning practices align with COVID-19 IPC guidelines and national IPC strategy manual
• Institute regular systematic checking and validation of cleaning through visual inspection and fluorescent marker

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3 World Health Organization. WHO guidelines on hand hygiene in health care; 2009. https://apps.who.int/iris/bitstream/handle/10665/44102/9789241597906_eng.pdf?sequence=1&isAllowed=y &ua=1
In terms of the NRC outpatient dialysis unit, we recommend that patients be reorganised into three groups, although this will need to be reviewed if there is evidence to suggest ongoing transmission in the unit:

1. **Red:** Confirmed COVID-19 cases: All patients currently in group 3 are red. These patients should be managed separately, and either receive dialysis as an inpatient or dialysis at home. The criteria to move out of the red category are as follows: If the patient has had mild disease then such patients are considered non-infectious two weeks following onset of symptoms and can then be moved into the green group. If the patient had severe disease then the patient can be considered non-infectious two weeks following stabilization of clinical condition (e.g. no requirement for oxygen) and then can be moved into the green group.

2. **Yellow:** Possible exposure to COVID-19: Patient had a negative SARS-CoV-2 PCR, asymptomatic, but last had contact with a potentially positive patient less than 14 days ago. Patients currently in group 2 are yellow. This group was last exposed to a confirmed COVID-19 case on 14 April. This group will need to be monitored for a further seven days and if no new infected patients are uncovered then they can be transferred to the green group. However, if a case is uncovered then that case is moved to red group and the rest of the yellow group needs to be monitored for a further 14 days before they can be transferred to green.

3. **Green:** Low risk of COVID-19: Patient had at least one negative SARS-CoV-2 PCR, last had contact with a confirmed or suspected case more than 14 days ago, and asymptomatic for the preceding two weeks. At this point in time all of patients in group 1 and group 4 can be considered green- all have tested negative, none have symptoms and their last encounter with a potential case of COVID was more than 14 days ago (31 March).
Appendix A. Full patient timeline (up to 6 May)