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Case report

Clinical course of a 66-year-old man with an acute ischaemic stroke in the setting of a COVID-19 infection

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SUMMARY

A 66-year-old man was admitted to hospital with a right frontal cerebral infarct producing left-sided weakness and a deterioration in his speech pattern. The cerebral infarct was confirmed with CT imaging. The only evidence of respiratory symptoms on admission was a 2 L oxygen requirement, maintaining oxygen saturations between 88% and 92%. In a matter of hours this patient developed a greater oxygen requirement, alongside reduced levels of consciousness. A positive COVID-19 throat swab, in addition to bilateral pneumonia on chest X-ray and lymphopaenia in his blood tests, confirmed a diagnosis of COVID-19 pneumonia. A proactive decision was made involving the patients' family, ward and intensive care healthcare staff, to not escalate care above a ward-based ceiling of care. The patient died 5 days following admission under the palliative care provided by the medical team.

BACKGROUND

SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) is a new strain of coronavirus that is thought to have originated in December 2019 in Wuhan, China. In a matter of months, it has erupted from non-existence to perhaps the greatest challenge to healthcare in modern times, grinding most societies globally to a sudden halt. Consequently, the study and research into SARS-CoV-2 is invaluable. Although coronaviruses are common, SARS-CoV-2 appears to be considerably more contagious. The WHO figures into the 2003 SARS-CoV-1 outbreak, from November 2002 to July 2003, indicate a total of 8439 confirmed cases globally.¹ In comparison, during a period of 4 months from December 2019 to July 2020, the number of global cases of COVID-19 reached 10 357 662, increasing exponentially, illustrating how much more contagious SARS-CoV-2 has been.²

Previous literature has indicated infections, and influenza-like illness have been associated with an overall increase in the odds of stroke development.³ There appears to be a growing correlation between COVID-19 positive patients presenting to hospital with ischaemic stroke; however, studies investigating this are in progress, with new data emerging daily. This patient report comments on and further characterises the link between COVID-19 pneumonia and the development of ischaemic stroke. At the time of this patients' admission, there were 95

positive cases from 604 COVID-19 tests conducted in the local community, with a predicted population of 108 000.⁴ Only 4 days later, when this patient died, the figure increased to 172 positive cases (81% increase), illustrating the rapid escalation towards the peak of the pandemic, and widespread transmission within the local community (figure 1). As more cases of ischaemic stroke in COVID-19 pneumonia patients arise, the recognition and understanding of its presentation and aetiology can be deciphered. Considering the virulence of SARS-CoV-2 it is crucial as a global healthcare community, we develop this understanding, in order to intervene and reduce significant morbidity and mortality in stroke patients.

CASE PRESENTATION

A 66-year-old man presented to the hospital with signs of left-sided weakness. The patient had a background of chronic obstructive pulmonary disease (COPD), atrial fibrillation and had one previous ischaemic stroke, producing left-sided haemiparesis, which had completely resolved. He was a non-smoker and lived in a house. The patient was found slumped over on the sofa at home on 1 April 2020, by a relative at approximately 01:00, having been seen to have no acute medical illness at 22:00. The patients' relative initially described disorientation and agitation with weakness noted in the left upper limb and dysarthria. At the time of presentation, neither the patient nor his relative identified any history of fever, cough, shortness of breath, loss of taste, smell or any other symptoms; however, the patient did have a prior admission 9 days earlier with shortness of breath.

The vague nature of symptoms, entwined with considerable concern over approaching the hospital, due to the risk of contracting COVID-19, created a delay in the patients' attendance to the accident and emergency department. His primary survey conducted at 09:20 on 1 April 2020 demonstrated a patent airway, with spontaneous breathing and good perfusion. His Glasgow Coma Scale (GCS) score was 15 (a score of 15 is the highest level of consciousness), his blood glucose was 7.2, and he did not exhibit any signs of trauma. His abbreviated mental test score was 7 out of 10, indicating a degree of altered cognition. An ECG demonstrated atrial fibrillation with a normal heart rate. His admission weight measured 107 kg. At 09:57



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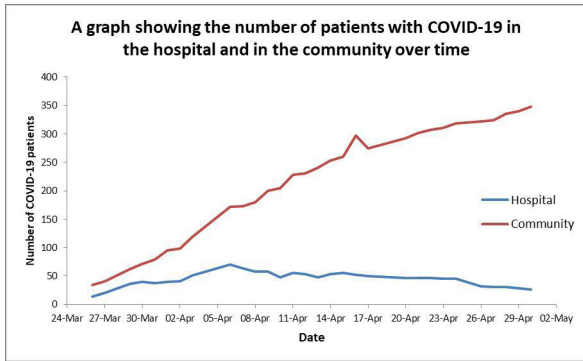


Figure 1 A graph showing the number of patients with COVID-19 in the hospital and in the community over time.

the patient required 2 L of nasal cannula oxygen to maintain his oxygen saturations between 88% and 92%. He started to develop agitation associated with an increased respiratory rate at 36 breaths per minute. On auscultation of his chest, he demonstrated widespread coarse crepitation and bilateral wheeze. Throughout he was haemodynamically stable, with a systolic blood pressure between 143 mm Hg and 144 mm Hg and heart rate between 86 beats/min and 95 beats/min. From a neurological standpoint, he had a mild left facial droop, 2/5 power in both lower limbs, 2/5 power in his left upper limb and 5/5 power in his right upper limb. Tone in his left upper limb had increased. This patient was suspected of having COVID-19 pneumonia alongside an ischaemic stroke.

INVESTIGATIONS

A CT of his brain conducted at 11:38 on 1 April 2020 (figure 2) illustrated an ill-defined hypodensity in the right frontal lobe medially, with sulcal effacement and loss of grey-white matter. This was highly likely to represent acute anterior cerebral artery territory infarction. Furthermore an oval low-density area in the right cerebellar hemisphere, that was also suspicious of an acute infarction. These vascular territories did not entirely correlate with his clinical picture, as limb weakness is not as prominent in anterior cerebral artery territory ischaemia. Therefore this left-sided weakness may have been an amalgamation of residual weakness from his previous stroke, in addition to his

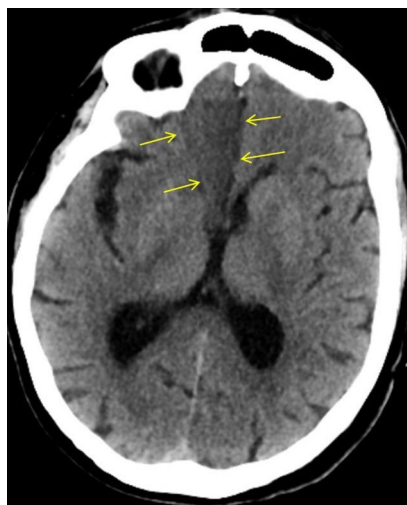


Figure 2 CT imaging of this patients' brain demonstrating a wedge-shaped infarction of the anterior cerebral artery territory.

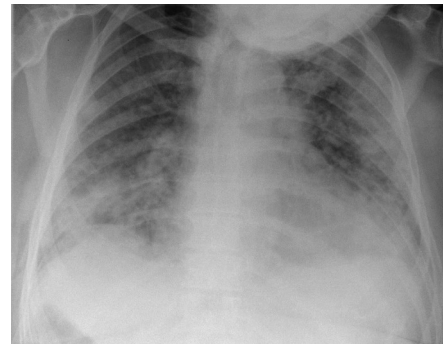


Figure 3 Chest X-ray demonstrating the bilateral COVID-19 pneumonia of this patient on admission.

acute cerebral infarction. An erect AP chest X-ray with portable equipment (figure 3) conducted on the same day demonstrated patchy peripheral consolidation bilaterally, with no evidence of significant pleural effusion. The pattern of lung involvement raised suspicion of COVID-19 infection, which at this stage was thought to have provoked the acute cerebral infarct. Clinically significant blood results from 1 April 2020 demonstrated a raised C-reactive protein (CRP) at 215 mg/L (normal 0–5 mg/L) and lymphopaenia at 0.5×10^9 (normal 1×10^9 to 3×10^9). Other routine blood results are provided in table 1.

Interestingly the patient, in this case, was clinically assessed in the accident and emergency department on 23 March 2020, 9 days prior to admission, with symptoms of shortness of breath. His blood results from this day showed a CRP of 22 mg/L and a greater lymphopaenia at 0.3×10^9 . He had a chest X-ray (figure 4), which indicated mild radiopacification in the left mid zone. He was initially treated with intravenous co-amoxiclav and ciprofloxacin. The following day he had minimal symptoms (CURB 65 score 1 for being over 65 years). Given improving blood results (declining CRP), he was discharged home with a course of oral amoxicillin and clarithromycin. As national governmental restrictions due to COVID-19 had not

Table 1 Clinical biochemistry and haematology blood results of the patient

Hb	157 g/L
WCC	5.8
Platelet count	195
MCV	86
Neutrophils	4.9
Lymphocytes	0.5
INR	1.3
PT	16.7
APTT	28
Sodium	137
Potassium	Sample haemolysed
Urea	9.5
Creatinine	84
eGFR	83
Bilirubin	26
Albumin	29
CRP	215
Total protein	69

APTT, activated partial thromboplastin time; CRP, C-reactive protein; eGFR, estimated glomerular filtration rate; Hb, haemoglobin; INR, international normalised ratio; MCV, mean corpuscular volume; PT, prothrombin time; WCC, white cell count.

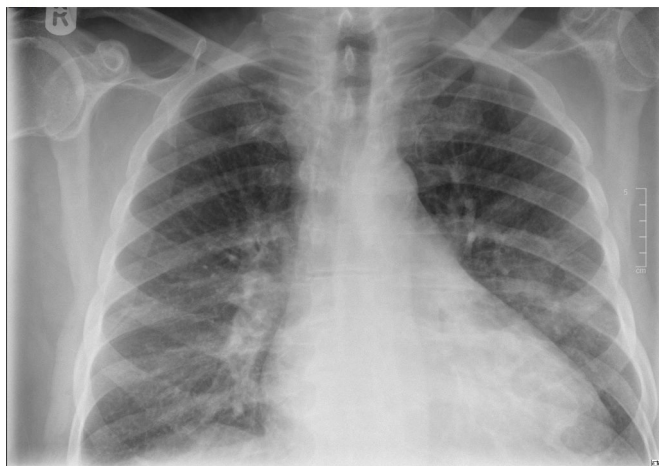


Figure 4 Chest X-ray conducted on prior admission illustrating mild radiopacification in the left mid zone.

been formally announced until 23 March 2020, and inconsistencies regarding personal protective equipment training and usage existed during the earlier stages of this rapidly evolving pandemic, it is possible that this patient contracted COVID-19 within the local community, or during his prior hospital admission. It could be argued that the patient had early COVID-19 signs and symptoms, having presented with shortness of breath, lymphopaenia, and having had subtle infective chest X-ray changes. The patient explained he developed a stagnant productive cough, which began 5 days prior to his attendance to hospital on 23 March 2020. He responded to antibiotics, making a full recovery following 7 days of treatment. This information does not assimilate with the typical features of a COVID-19 infection. A diagnosis of community-acquired pneumonia or infective exacerbation of COPD seem more likely. However, given the high incidence of COVID-19 infections during this patients' illness, an exposure and early COVID-19 illness, prior to the 23 March 2020, cannot be completely ruled out.

TREATMENT

On the current admission, this patient was managed with nasal cannula oxygen at 2 L. By the end of the day, this had progressed to a venturi mask, requiring 8 L of oxygen to maintain oxygen saturation. He had also become increasingly drowsy and confused, his GCS declined from 15 to 12. However, the patient was still haemodynamically stable, as he had been in the morning. An arterial blood gas demonstrated a respiratory alkalosis (pH 7.55, $p\text{CO}_2$ 3.1, $p\text{O}_2$ 6.7 and HCO_3 24.9, lactate 1.8, base excess 0.5). He was commenced on intravenous co-amoxiclav and ciprofloxacin, to treat a potential exacerbation of COPD. This patient had a COVID-19 throat swab on 1 April 2020. Before the result of this swab, an early discussion was held with the intensive care unit staff, who decided at 17:00 on 1 April 2020 that given the patients presentation, rapid deterioration, comorbidities and likely COVID-19 diagnosis he would not be for escalation to the intensive care unit, and if he were to deteriorate further the end of life pathway would be most appropriate. The discussion was reiterated to the patients' family, who were in agreement with this. Although he had evidence of an ischaemic stroke on CT of his brain, it was agreed by all clinicians that intervention for this was not as much of a priority as providing optimal palliative care, therefore, a minimally invasive method of treatment was

advocated by the stroke team. The patient was given 300 mg of aspirin and was not a candidate for fibrinolysis.

OUTCOME AND FOLLOW-UP

The following day, before the throat swab result, had appeared the patient deteriorated further, requiring 15 L of oxygen through a non-rebreather face mask at 60% FiO_2 to maintain his oxygen saturation, at a maximum of 88% overnight. At this point, he was unresponsive to voice, with a GCS of 5. Although, he was still haemodynamically stable, with a blood pressure of 126/74 mm Hg and a heart rate of 98 beats/min. His respiratory rate was 30 breaths/min. His worsening respiratory condition, combined with his declining level of consciousness made it impossible to clinically assess progression of the neurological deficit generated by his cerebral infarction. Moreover, the patient was declining sharply while receiving the maximal ward-based treatment available. The senior respiratory physician overseeing the patients' care decided that a palliative approach was in this his best interest, which was agreed on by all parties. The respiratory team completed the 'recognising dying' documentation, which signified that priorities of care had shifted from curative treatment to palliative care. Although the palliative team was not formally involved in the care of the patient, the patient received comfort measures without further attempts at supporting oxygenation, or conduction of regular clinical observations. The COVID-19 throat swab confirmed a positive result on 2 April 2020. The patient was treated by the medical team under jurisdiction of the hospital palliative care team. This included the prescribing of anticipatory medications and a syringe driver, which was established on 3 April 2020. His antibiotic treatment, non-essential medication and intravenous fluid treatment were discontinued. His comatose condition persisted throughout the admission. Once the patients' GCS was 5, it did not improve. The patient was pronounced dead by doctors at 08:40 on 5 April 2020.

DISCUSSION

SARS-CoV-2 is a type of coronavirus that was first reported to have caused pneumonia-like infection in humans on 3 December 2019.⁵ As a group, coronaviruses are a common cause of upper and lower respiratory tract infections (especially in children) and have been researched extensively since they were first characterised in the 1960s.⁶ To date, there are seven coronaviruses that are known to cause infection in humans, including SARS-CoV-1, the first known zoonotic coronavirus outbreak in November 2002.⁷ Coronavirus infections pass through communities during the winter months, causing small outbreaks in local communities, that do not cause significant mortality or morbidity.

SARS-CoV-2 strain of coronavirus is classed as a zoonotic coronavirus, meaning the virus pathogen is transmitted from non-humans to cause disease in humans. However the rapid spread of SARS-CoV-2 indicates human to human transmission is present. From previous research on the transmission of coronaviruses and that of SARS-CoV-2 it can be inferred that SARS-CoV-2 spreads via respiratory droplets, either from direct inhalation, or indirectly touching surfaces with the virus and exposing the eyes, nose or mouth.⁸ Common signs and symptoms of the COVID-19 infection identified in patients include high fevers, severe fatigue, dry cough, acute breathing difficulties, bilateral pneumonia on radiological imaging and lymphopaenia.⁹ Most of these features were identified in this case study. The significance of COVID-19 is illustrated by the speed of its global spread and the potential to cause severe clinical presentations, which as of April 2020 can only be treated symptomatically. In Italy, as of mid-March 2020,

it was reported that 12% of the entire COVID-19 positive population and 16% of all hospitalised patients had an admission to the intensive care unit.¹⁰

The patient, in this case, illustrates the clinical relevance of understanding COVID-19, as he presented with an ischaemic stroke underlined by minimal respiratory symptoms, which progressed expeditiously, resulting in acute respiratory distress syndrome and subsequent death.

Our case is an example of a new and ever-evolving clinical correlation, between patients who present with a radiological confirmed ischaemic stroke and severe COVID-19 pneumonia. As of April 2020, no comprehensive data of the relationship between ischaemic stroke and COVID-19 has been published, however early retrospective case series from three hospitals in Wuhan, China have indicated that up to 36% of COVID-19 patients had neurological manifestations, including stroke.¹¹ These studies have not yet undergone peer review, but they tell us a great deal about the relationship between COVID-19 and ischaemic stroke, and have been used to influence the American Heart Association's 'Temporary Emergency Guidance to US Stroke Centres During the COVID-19 Pandemic'.¹²

The relationship between similar coronaviruses and other viruses, such as influenza in the development of ischaemic stroke has previously been researched and provide a basis for further investigation, into the prominence of COVID-19 and its relation to ischaemic stroke.³ Studies of SARS-CoV-2 indicate its receptor-binding region for entry into the host cell is the same as ACE2, which is present on endothelial cells throughout the body. It may be the case that SARS-CoV-2 alters the conventional ability of ACE2 to protect endothelial function in blood vessels, promoting atherosclerotic plaque displacement by producing an inflammatory response, thus increasing the risk of ischaemic stroke development.¹³

Other hypothesised reasons for stroke development in COVID-19 patients are the development of hypercoagulability, as a result of critical illness or new onset of arrhythmias, caused by severe infection. Some case studies in Wuhan described immense inflammatory responses to COVID-19, including elevated acute phase reactants, such as CRP and D-dimer. Raised D-dimers are a non-specific marker of a prothrombotic state and have been associated with greater morbidity and mortality relating to stroke and other neurological features.¹⁴

Arrhythmias such as atrial fibrillation had been identified in 17% of 138 COVID-19 patients, in a study conducted in Wuhan, China.¹⁵ In this report, the patient was known to have atrial fibrillation and was treated with rivaroxaban. The acute inflammatory state COVID-19 is known to produce had the potential to create a prothrombotic environment, culminating in an ischaemic stroke.

Some early case studies produced in Wuhan describe patients in the sixth decade of life that had not been previously noted to have antiphospholipid antibodies, contain the antibodies in blood results. They are antibodies signify antiphospholipid syndrome; a prothrombotic condition.¹⁶ This raises the hypothesis concerning the ability of COVID-19 to evoke the creation of these antibodies and potentiate thrombotic events, such as ischaemic stroke.

No peer-reviewed studies on the effects of COVID-19 and mechanism of stroke are published as of April 2020; therefore, it is difficult to evidence a specific reason as to why COVID-19 patients are developing neurological signs. It is suspected that a mixture of the factors mentioned above influence the development of ischaemic stroke.

If we delve further into this patients' comorbid state exclusive to COVID-19 infection, it can be argued that this patient was already at a relatively higher risk of stroke development compared with the general population. The fact this patient had previously had an ischaemic stroke illustrates a prior susceptibility. This patient had a known background of hypertension and atrial fibrillation, which as mentioned previously, can influence blood clot or plaque propagation in the development of an acute ischaemic event.¹⁵ Although the patient was prescribed rivaroxaban as an anticoagulant, true consistent compliance to rivaroxaban or other medications such as amlodipine, clopidogrel, candesartan and atorvastatin cannot be confirmed; all of which can contribute to the reduction of influential factors in the development of ischaemic stroke. Furthermore, the fear of contracting COVID-19, in addition to his vague symptoms, unlike his prior ischaemic stroke, which demonstrated dense left-sided haemiparesis, led to a delay in presentation to hospital. This made treatment options like fibrinolysis unachievable, although it can be argued that if he was already infected with COVID-19, he would have still developed life-threatening COVID-19 pneumonia, regardless of whether he underwent fibrinolysis. It is therefore important to consider that if this patient did not contract COVID-19 pneumonia, he still had many risk factors that made him prone to ischaemic stroke formation. Thus, we must consider whether similar patients would suffer from ischaemic stroke, regardless of COVID-19 infection and whether COVID-19 impacts on the severity of the stroke as an entity.

Having said this, the management of these patients is dependent on the likelihood of a positive outcome from the COVID-19 infection. Establishing the ceiling of care is crucial, as it prevents incredibly unwell or unfit patients' from going through futile treatments, ensuring respect and dignity in death, if this is the likely outcome. It also allows for the provision of limited or intensive resources, such as intensive care beds or endotracheal intubation during the COVID-19 pandemic, to those who are assessed by the multidisciplinary team to benefit the most from their use. The way to establish this ceiling of care is through an early multidisciplinary discussion. In this case, the patient did not convey his wishes regarding his care to the medical team or his family; therefore it was decided among intensive care specialists, respiratory physicians, stroke physicians and the patients' relatives. The patient was discussed with the intensive care team, who decided that as the patient sustained two acute life-threatening illnesses simultaneously and had rapidly deteriorated, ward-based care with a view to palliate if the further deterioration was in the patients' best interests. These decisions were not easy to make, especially as it was on the first day of presentation. This decision was made in the context of the patients' comorbidities, including COPD, the patients' age, and the availability of intensive care beds during the steep rise in intensive care admissions, in the midst of the COVID-19 pandemic (figure 1). Furthermore, the patients' rapid and permanent decline in GCS, entwined with the severe stroke on CT imaging of the brain made it more unlikely that significant and permanent recovery could be achieved from mechanical intubation, especially as the damage caused by the stroke could not be significantly reversed. As hospitals manage patients with COVID-19 in many parts of the world, there may be tension between the need to provide higher levels of care for an individual patient and the need to preserve finite resources to maximise the benefits for most patients. This patient presented during a steep rise in intensive care admissions, which may have influenced the early decision not to treat the patient in an intensive care setting. Retrospective studies from Wuhan investigating mortality in patients with

multiple organ failure, in the setting of COVID-19, requiring intubation have demonstrated mortality can be up to 61.5%.¹⁷ The mortality risk is even higher in those over 65 years of age with respiratory comorbidities, indicating why this patient was unlikely to survive an admission to the intensive care unit.¹⁸

Regularly updating the patients' family ensured cooperation, empathy and sympathy. The patients' stroke was not seen as a priority given the severity of his COVID-19 pneumonia, therefore the least invasive, but most appropriate treatment was provided for his stroke. The British Association of Stroke Physicians advocate this approach and also request the notification to their organisation of COVID-19-related stroke cases, in the UK.¹⁹

Learning points

- ▶ SARS-CoV-2 (Severe Acute Respiratory Syndrome Coronavirus 2) is one of seven known coronaviruses that commonly cause upper and lower respiratory tract infections. It is the cause of the 2019–2020 global coronavirus pandemic.
- ▶ The significance of COVID-19 is illustrated by the rapid speed of its spread globally and the potential to cause severe clinical presentations, such as ischaemic stroke.
- ▶ Early retrospective data has indicated that up to 36% of COVID-19 patients had neurological manifestations, including stroke.
- ▶ Potential mechanisms behind stroke in COVID-19 patients include a plethora of hypercoagulability secondary to critical illness and systemic inflammation, the development of arrhythmia, alteration to the vascular endothelium resulting in atherosclerotic plaque displacement and dehydration.
- ▶ It is vital that effective, open communication between the multidisciplinary team, patient and patients relatives is conducted early in order to firmly establish the most appropriate ceiling of care for the patient.

Contributors SB was involved in the collecting of information for the case, the initial written draft of the case and researching existing data on acute stroke and COVID-19. He also edited drafts of the report. MH was involved in reviewing and editing drafts of the report and contributing new data. SP oversaw the conduction of the project and contributed additional research papers.

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REFERENCES

- 1 Who.int. Who | cumulative number of reported probable cases of SARS, 2020. Available: <https://www.who.int/csr/sars/country/2003_07_04/en/> [Accessed 13 Apr 2020].
- 2 Who.int. Coronavirus disease 2019 (COVID-19) situation report – 163, 2020. Available: <https://www.who.int/docs/default-source/coronavirus/situation-reports/20200701-covid-19-sitrep-163.pdf?sfvrsn=c202f05b_2> [Accessed 3 Jul 2020].
- 3 Alvord T, Kulick E, Cannine M, et al. Abstract 189: influenza-like illness and risk of stroke in New York state. *Stroke* 2019;50.
- 4 Council M. Key Facts About Mansfield District – Mansfield District Council. [online] Mansfield District Council, 2020. Available: <<https://www.mansfield.gov.uk/council-councillors-democracy/key-facts-mansfield/1>> [Accessed 4 May 2020].
- 5 Who.int. Coronavirus Disease (COVID-19) - Events As They Happen, 2020. Available: <<https://www.who.int/emergencies/diseases/novel-coronavirus-2019/events-as-they-happen>> [Accessed 13 Apr 2020].
- 6 Kahn JS, McIntosh K. History and recent advances in coronavirus discovery. *Pediatr Infect Dis J* 2005;24:S223–7.
- 7 Yin Y, Wunderink RG. Mers, SARS and other coronaviruses as causes of pneumonia. *Respirology* 2018;23:130–7.
- 8 van Doremalen N, Bushmaker T, Morris DH, et al. Aerosol and surface stability of SARS-CoV-2 as compared with SARS-CoV-1. *N Engl J Med* 2020;382:1564–7.
- 9 Zhou P, Yang X-L, Wang X-G, et al. A pneumonia outbreak associated with a new coronavirus of probable bat origin. *Nature* 2020;579:270–3.
- 10 Grasselli G, Pesenti A, Cecconi M. Critical care utilization for the COVID-19 outbreak in Lombardy, Italy. *JAMA* 2020;323:1545.
- 11 Li Y, Li M, Wang M, et al. Acute cerebrovascular disease following COVID-19: a single center, retrospective, observational study. *Stroke Vasc Neurol* 2020. doi:10.1136/svn-2020-000431. [Epub ahead of print: 02 Jul 2020].
- 12 American Stroke Association, 2020. Temporary emergency guidance to US stroke centers during the COVID-19 pandemic. Available: <https://www.ahajournals.org/doi/10.1161/STROKEAHA.120.030023> [Accessed 13 Apr 2020].
- 13 Zhang Y-H, Zhang Y-huan, Dong X-F, et al. ACE2 and Ang-(1-7) protect endothelial cell function and prevent early atherosclerosis by inhibiting inflammatory response. *Inflamm Res* 2015;64:253–60.
- 14 Huang C, Wang Y, Li X, et al. Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. *The Lancet* 2020;395:497–506.
- 15 Wang D, Hu B, Hu C, et al. Clinical characteristics of 138 hospitalized patients with 2019 novel coronavirus-infected pneumonia in Wuhan, China. *JAMA* 2020;323:1061.
- 16 Zhang Y, Xiao M, Zhang S, et al. Coagulopathy and antiphospholipid antibodies in patients with Covid-19. *N Engl J Med* 2020;382:e38.
- 17 Yao W, Wang T, Jiang B, et al. Emergency tracheal intubation in 202 patients with COVID-19 in Wuhan, China: lessons learnt and international expert recommendations. *Br J Anaesth* 2020;125:e28–37.
- 18 Yang X, Yu Y, Xu J, et al. Clinical course and outcomes of critically ill patients with SARS-CoV-2 pneumonia in Wuhan, China: a single-centered, retrospective, observational study. *Lancet Respir Med* 2020;8:475–81.
- 19 British Association of Stroke Physicians. British Association of Stroke Physician's statement with regards to COVID-19, 2020. Available: <https://basp.ac.uk/news/basp-covid-19-membership-statementdocx/> [Accessed 13 Apr 2020].

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