accelerated atrophy over time in HIV+ participants with higher cardiovascular risk factors (p = 0.002).
Conclusion The study demonstrates a three-hit model of subcortical injury in HIV+ individuals: HIV-driven atrophy in most subcortical structures; abnormal brain aging and HIV infection synergy in the caudate and pallidum; and cardiovascular-related injury linked to diffuse premature atrophy and emerging accelerated atrophy in the putamen.

031 COVID-19 DISEASE OUTCOMES IN A UK MYASTHENIA CENTRE DURING THE FIRST YEAR OF THE PANDEMIC

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Objectives Describe the outcomes of patients treated at the Myasthenia Centre at Oxford University Trust who developed COVID-19 during the first year of the pandemic.
Methods Retrospective audit of patients diagnosed with COVID-19 between 31st January 2020 – 31st January 2021. Outcomes of COVID-19 complications, including relapse of myasthenia gravis (MG), were analysed.
Results The Myasthenia Centre treated 487 patients, including 370 with acetylcholine receptor (AChR) MG, 74 sero-negative MG, 20 MuSK MG and 23 Lambert-Eaton Myasthenic Syndrome (LEMS). COVID-19 was diagnosed in a total of twelve patients (2.5%) including ten AChR, one MuSK and one LEMS patient, with a mean age of 63.8 years (range 20 – 92 years). Five patients were asymptomatic of MG prior to the diagnosis of COVID-19. Treatments prior to diagnosis included pyridostigmine (8/12), prednisolone (7/12), azathioprine (3/12), mycophenolate (1/12) and rituximab (1/12). The majority (8/12) had at least one other co-morbid risk factor for severe COVID-19.

COVID-19 resulted in hospital admission in six patients, with three requiring intensive care treatment. One patient with AChR MG (with NHL and NMO treated with rituximab) died from COVID-19 without MG relapse. Two elderly patients developed moderate COVID-19 after a single dose BioNTech vaccination without MG relapse.

MG relapse occurred in four patients post COVID-19, with two requiring inpatient management including IVIG.
Conclusion COVID-19 disease was associated with relapse of MG, with all patients in this group surviving. Further research is required to establish if COVID-19 precipitates MG relapse at a different rate compared to other infectious diseases.

032 COGNITIVE DEFICITS ARE ASSOCIATED WITH ANOSMIA BUT NOT ANXIO-DEPRESSIVE SYMPTOMS IN COVID-19

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Objectives To characterise cognitive performance and olfaction in recovered COVID-19 patients.
Methods Patients underwent cognitive, olfaction and mental health assessments 2 months after initial SARS-CoV-2 infection as part of the Sydney St. Vincent's Hospital ADAPT study, a prospective cohort study. Cognition was assessed with the Cogstate computerised battery and expressed as a demographically-corrected composite z-score and clinically classified as impaired/borderline/unimpaired. Anxio-depressive symptoms were assessed with the Depression in the Medical ill scale-10 (DMI-10), the Somatic and Psychological HEalth Report-34 (SPHERE) Psych sub-scale, and the Impact of Events Scale-Revised (IESR) and reduced into single Principal Component explaining 80% of the variance. Olfaction was assessed with the NIH Toolbox Odor Identification test and expressed as demographically-corrected T-scores, and impaired/unimpaired. Disease severity was classified as mild (40%), moderate (50%) or hospitalised (10%).
Results 132 patients (mean age=46±15; 40% women, median education=16 years, 10% Non-English-Speaking Background-NESB) were included. 17% had impaired cognition, 10% had borderline deficits, 25% has impaired olfaction. 25% had clinically elevated symptoms on the DMI-10, 13% on the IESR, and 35% on the SPHERE. Regression analyses showed that anxio-depression was not associated with cognitive performance (unadjusted p=.43; adjusted for sex & NESB p=.98) nor impaired/unimpaired status (unadjusted p=.50; adjusted for sex & NESB p=.78). Cognitively impaired patients were more likely to have impaired olfaction (p<.009). Results were independent of disease severity.
Conclusions Cognitive impairment is common and not related to psychological factors, may occur independent of disease severity and is associated with anosmia. These point to direct brain effects of COVID-19.

033 COVID:19 THE EPICENTRE OF NEUROLOGICAL EVENTS IN THE HUMAN BRAIN

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Objective Neuropathology caused by COVID-19 has been widely reported, and the characterisation of the spatial distribution of these pathology remains critical to assess long and short-term neurological *sequelae*.
Methods We performed a systematic review of the literature to quantify the locations of small neurological events identified with magnetic resonance imaging (MRI) among COVID-19 patients. Neurological events were localised into the Desikan-Killiany grey and white matter atlases. A mathematical network diffusion model was then used to test whether the spatial distribution of neurological events could be explained via a linear spread through the structural connectome of the brain.

Results The highest proportions (26%) of white matter events were observed within the bilateral corticospinal tracts. The highest proportions (~10%) of grey matter events were observed in areas including the bilateral superior temporal, precentral, and lateral occipital cortices. Subcortical events were most frequently identified in the Pallidum. The application of a mathematical network diffusion model suggested that the spatial pattern of the small neurological events in COVID-19 can be modelled with a linear diffusion of spread from epicentres in the bilateral cerebellum and basal ganglia (Pearson's $r=0.41$, $p<0.001$, corrected).

Conclusions To our knowledge, this is the first study to systematically characterise the spatial distribution of small neurological events in COVID-19 patients and test whether the spatial distribution of these events can be explained by a linear diffusion spread model. As such, initial sub-cortical events which manifest as altered consciousness could be expected to be followed by later cortical events manifesting as altered sensorimotor functioning.

034 MR SPECTROSCOPY AND DYNAMIC CONTRAST-ENHANCED PERFUSION STUDIES IN TWO SARS-COV-2 INFECTION PATIENTS WITH NEUROLOGICAL COMPLICATIONS AND NO OTHER MR ABNORMALITIES

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Objectives To understand NeuroCovid pathogenesis by using MR spectroscopy and dynamic contrast perfusion in 2 SARS-COV-2 patients with neurological complications.

Methods MR spectroscopy (MRS) and dynamic contrast-enhanced perfusion (k-trans), which is a sensitive marker of blood brain barrier (BBB) damage, were performed in 2 patients with respiratory viral PCR confirmed SARS-CoV-2 infection and 2 local controls. The MRS and k-trans results were also compared with published controls.

Results Case 1 was a 73-year-old man with critical SARS-CoV-2 infection requiring 4 days of mechanical ventilation. He had persistent anosmia, anorexia and apathy; saccadic pursuit eye movements, action tremor in the left hand and a positive right-sided palmomental reflex. These changes normalised by day 30 of presentation. MRI brain was undertaken on day 37.

Case 2 was a 61-year-old man with critical SARS-CoV-2 infection requiring 43 days of mechanical ventilation. He was slow to wake post weaning of sedation and had residual mild cognitive impairment. MRI brain was undertaken on day 62 of presentation.

No abnormalities were detected on T1, T2, FLAIR, DWI, SWI MR sequences. However, for both patients there was diffuse increase in k-trans especially in the frontal cortex and increased glutamate-glutamine MRS signal intensities in the pons compared to controls.

Conclusions The MRS and k-trans changes show excitotoxicity and BBB damage, in the absence of stroke or MR-defined white matter injury. They suggest a direct effect of SARS-CoV-2 on the brain. These MR techniques can offer insight into NeuroCovid pathogenesis when patients are no longer infectious.

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035 CASE-CONTROL STUDY OF RISK FACTORS FOR STROKE AMONG CRITICALLY-ILL PATIENTS WITH SARS-COV-2: AN ANALYSIS OF THE COVID-19 CRITICAL CARE CONSORTIUM (CCCC) GLOBAL REGISTRY

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Objective COVID-19 has been identified as a risk factor for severe cerebrovascular complications, albeit mostly in small patient populations, limited to specific regions, and including all severities of disease. Utilising the largest database of critically-ill COVID-19 patients, we investigated risk factors for stroke in intensive care unit (ICU) COVID-19 patients.

Methods Data for this matched case-control study were extracted from a large international registry of adult COVID-19 patients requiring ICU admission. Patients with imaging-confirmed cerebrovascular events identified following ICU admission were compared against five controls per case, matched for demographics, morphometrics, illness severity, and ICU days. Expert consensus determined key clinical and laboratory variables for risk assessment.

Results From January 1-December 21 2020, 2,715 ICU patients were registered across >370 sites spanning 52 countries; acute stroke was identified during the ICU stay in 59 (2.2%); 27(46%) haemorrhagic, 19(32%) ischaemic, 13(22%) unspecified. Stroke patients had higher SOFA and APACHE scores, more frequent hypertension and cardiovascular disease, and more often required mechanical ventilation, vasopressors, and ECMO. Diabetes, hypertension, smoking, and Caucasian ethnicity were identified as risk factors for ischaemic versus haemorrhagic stroke and being stroke-free. Ethnicity (Hispanic or black), higher PaO_2, and extracorporeal membrane oxygenation (ECMO) were significant risk factors for haemorrhagic stroke.Anticoagulation had no association with either stroke subtype.