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.