day functional outcome, mortality or stroke-onset to treatment compared with the directly presenting patients (all $p > 0.05$). Successful reperfusion rates and sICH were similar between cohorts (all $p > 0.05$).

Conclusion Inter-hospital transfer in the ETW but not LTW is associated with longer stroke-onset to treatment, worse 90-day functional outcome and higher mortality.

028

ADJUNCTIVE INTRAARTERIAL THROMBOLYSIS IN ENDOVASCULAR CLOT RETRIEVAL: A SYSTEMATIC REVIEW AND META-ANALYSIS

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Objective To evaluate the safety and efficacy of intra-arterial thrombolysis (IAT) as an adjunct to endovascular clot retrieval (ECR) in ischaemic stroke, we performed a systematic review and meta-analysis of the literature.

Methods Searches were performed using Medline, Embase, and Cochrane databases for studies that compared ECR to ECR with adjunctive IAT (ECR+IAT). Safety outcomes included symptomatic intracerebral haemorrhage (sICH) and mortality at three months. Efficacy outcomes included successful reperfusion (Thrombolysis in Cerebral Infarction score of 2b to 3), and functional independence, defined as a modified Rankin Scale score of 0 to 2 at three months.

Results Five studies were identified that compared combined ECR+IAT (IA alteplase or urokinase) to ECR-only, and were included in the random effects meta-analysis. There were 1693 ECR patients, including 269 patients treated with combined ECR+IAT and 1424 patients receiving ECR-only. Pooled analysis did not demonstrate any differences between ECR+IAT and ECR-only in rates of sICH (OR: 0.61, 95% CI: 0.20-1.85; $P=0.78$), mortality (OR: 0.77, 95% CI: 0.54-1.10; $P=0.15$), or successful reperfusion (OR: 1.05, 95% CI: 0.52-2.15; $P=0.89$). There was a higher rate of functional independence in patients treated with ECR+IAT, although this was not statistically significant (OR: 1.34, 95% CI: 1.00-1.80; $P=0.053$).

Conclusions Adjunctive IAT appears to be safe. In specific situations, neurointerventionists may be justified in administering small doses of intraarterial alteplase or urokinase as rescue therapy during ECR.

029

HIGH SENSITIVITY TROPONIN IN ACUTE ISCHAEMIC STROKE STUDY (TACIS)

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Objective We designed a multi-centre prospective cohort study to explore the hypothesis that early acutely elevated high sensitivity troponin (hsT) is associated with cardioembolic stroke (CES)

Methods Ischaemic strokes across three hospitals underwent hsTroponin testing and 2 blinded clinicians classified patients as CES, NCE (Non Cardioembolic) or ESUS by ESUS criteria. Characteristics included baseline NIHSS, renal function, hypertension, diabetes, smoking, ischaemic heart disease, past stroke and congestive cardiac failure. The odds of positive hsT for CES Vs NCE and ESUS Vs NCE were modelled with step-wise addition of patient characteristics.

Results 194 ischaemic stroke cases were included, with a mean age of 71 years and a 57:43 male:female ratio. 65 had a positive hsTroponin, which was associated with older age, hypertension, cardiac failure, coronary disease, an eGFR < 60 and a higher NIHSS. Positive hsTroponin was associated with CES (OR, 2.06; 95% CI, 1.12-3.79; $P = 0.02$). This association persisted after adjusting for confounders, such as age, sex, atrial fibrillation, renal impairment, ischaemic heart disease and previous stroke (Adjusted OR, 4.07; 95% CI, 1.41-11.75; $P=0.01$). ESUS was negatively associated with an elevated hs troponin (OR, 0.45; 95% CI, 0.22-0.94; $P=0.03$). This was not significant when adjusting for other variables ($P=0.09$).

Conclusions An elevated hs troponin after acute ischaemic stroke is independently associated with a cardioembolic mechanism. High sensitivity cardiac troponin was not significantly associated with ESUS after adjusting for confounders, suggesting that a cardio-embolic cause may not be the dominant mechanism in this group.

030

BRAIN AGING AND CARDIOVASCULAR RISK FACTORS IN CHRONIC HIV: A LONGITUDINAL MRI STUDY

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Objectives We aimed to examine the relative contributions of HIV infection, age, and cardiovascular risk factors to subcortical brain atrophy.

Methods Virally suppressed HIV+ participants with low neuropsychological confounds ($n = 75$) and demographically matched HIV- controls ($n = 31$) completed baseline and 18-month follow-up MRI scans, neuropsychological evaluation, cardiovascular assessments, and laboratory tests. HIV+ participants were evaluated for HIV associated neurocognitive disorder (HAND). Subcortical volumes were extracted with Freesurfer. Volumetric and shape analyses were conducted using linear mixed-effect models incorporating interactions between age, time, and each of HIV status, HAND status, HIV disease factors, and cardiovascular markers.

Results HIV+ participants had smaller volumes of most structures compared to HIV- participants. Premature aging was evident in the pallidum using volumetric ($p = 0.032$) and shape analyses. Accelerated aging was observed in the caudate volumes for the more severe HAND subgroup ($p = 0.008$) and was associated with longer HIV duration for putamen volumes ($p = 0.04$). Higher CD4 counts had a protective effect on hippocampal volumes in older participants ($p = 0.04$). Cardiovascular measures were associated with smaller volumes across time for most structures; only the putamen demonstrated

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% had 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.