Chair's Select Abstracts - Adult Neurology and Neurophysiology (CNS/CSCN)

A.1

Changes in ischemic stroke presentations and associated workflow during the first wave of the COVID-19 pandemic: A population study

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Background: Pandemics may promote hospital avoidance among patients with emergencies, and added precautions may exacerbate treatment delays. Methods: We used linked administrative data and data from the Quality Improvement and Clinical Research Alberta Stroke Program – a registry capturing stroke-related data on the entire Albertan population (4.3 million) – to identify all patients hospitalized with stroke in the pre-pandemic (01/01/2016-27/02/2020) and COVID-19 pandemic (28/02/2020-30/08/2020) periods. We examined changes in stroke presentation rates and use of thrombolysis and endovascular therapy (EVT), adjusted for age, sex, comorbidities, and pre-admission care needs; and in workflow, stroke severity (National Institutes of Health Stroke Scale/NIHSS), and in-hospital outcomes. Results: We analyzed 19,531 patients with ischemic stroke pre-pandemic versus 2,255 during the pandemic. Hospitalizations/presentations dropped (weekly adjusted-incidence-rate-ratio[aIRR]:0.48,95%CI:0.46-0.50), as did population-level incidence of thrombolysis (aIRR:0.49,0.44-0.56) or EVT (aIRR:0.59,0.49-0.69). However, proportions of presenting patients receiving thrombolysis/EVT did not decline (thrombolysis:11.7% pre-pandemic vs 13.1% during-pandemic, aOR:1.02, 0.75-1.38). For out-of-hospital strokes, onset-to-door times were prolonged(adjusted-coefficient:37.0-minutes, 95%CI:16.5-57.5), and EVT recipients experienced greater door-to-reperfusion delays (adjusted-coefficient:18.7-minutes,1.45-36.0). NIHSS scores and in-hospital mortality did not differ. Conclusions: The first COVID-19 wave was associated with a halving of presentations and acute therapy utilization for ischemic stroke at a population level, and greater pre-/in-hospital treatment delays. Our data can inform public health messaging and stroke care in future pandemic waves.

A.2

Identification of predictors of response to Erenumab in episodic and chronic migraine in a cohort of patients: a preliminary analysis

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Background: Erenumab is an antibody anti-calcitonin gene related peptide (CGRP) receptor approved for the treatment of episodic (EM) and chronic migraine (CM). In this study, we aimed to identify the predictors of response to the treatment. Methods: This is an ongoing retrospective cohort study of 120 patients (49 with cervicalgia) with EM or CM treated with Erenumab. The first endpoint was to identify the success rate of this treatment (at least 50% reduction in monthly migraine days during the third month of the treatment). The second endpoint was to identify the predictors of response to Erenumab treatment. Results: Seventy one percent of patients achieved a favorable response (P-value<0.001) to Erenumab. Patients with cervicalgia showed a lower treatment success rate (21.1% with vs 40.8% without cervicalgia) while patients without cervicalgia showed a higher treatment success rate (78. 9% without vs 59.2% with cervicalgia) with a P-value of 0.025 and an odd ratio of 0.388 (95% CI 0.174-0.869, P-value=0.021). A similar trend was observed in patients with occipital neuralgia and obesity (P-value<0.08). Conclusions: The preliminary analysis of this study demonstrates that cervicalgia (and to a lesser extend occipital neuralgia and obesity) is a negative predictor of response to Erenumab in patients with migraine.

A.3

A novel recessive TNNT1 congenital core-rod myopathy in French Canadians

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Background: Mutations in the slow skeletal muscle troponin T (*TNNT1*) gene cause a congenital nemaline myopathy resulting in death from respiratory insufficiency in early infancy. We report on four French Canadians with a novel congenital *TNNT1* myopathy. Methods: Patients underwent lower extremity and paraspinal MRI, quadriceps biopsy and genetic testing. *TNNT1* expression in muscle was assessed by quantitative PCR and immunoblotting. Wild type or mutated *TNNT1* mRNAs were co-injected with morpholinos in a zebrafish knockdown model to assess for rescue of the morphant phenotype. Results: Four patients shared a novel missense homozygous mutation in *TNNT1*. They developed from childhood slowly progressive limb-girdle weakness with spinal rigidity and contractures. They suffered from restrictive lung disease and recurrent episodes of rhabdomyolysis. Older patients remained ambulatory into their sixties. Lower extremity MRI showed symmetrical myopathic changes. Paraspinal MRI showed diffuse fibro-fatty involution. Biopsies showed multi-minicores. Nemaline rods were seen in half the patients. *TNNT1* mRNA expression was similar in controls and patients, while levels of *TNNT1* protein were reduced in patients. Wild type *TNNT1* mRNA rescued the zebrafish morphants but mutant transcripts failed to do so. Conclusions: This study expands the spectrum of *TNNT1*-related myopathy to include a milder clinical phenotype caused by a functionally-confirmed novel mutation.