GP.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(95%CI: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.

GP.2

Changes in Leptin, CCL16 and sTNF-RII as a distinctive plasma immune profile in patients with fast progressing ALS

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Background: Amyotrophic lateral sclerosis (ALS) is highly heterogeneous with survival rate ranging from months to decades. Approximately 10-20% of patients develop a rapidly progressive disease and may die within the first year. Therefore, there is an increasing need for an early detection of unique molecular signatures associated with more aggressive forms of disease as it may help identify therapeutic targets. Methods: To identify a unique molecular signature in fast progressing patients, we recruited 45 sporadic ALS (sALS) patients and 35 age-matched healthy controls and measured 62 immune markers in plasma using cytokines array.

Results: We found that leptin was significantly downregulated in plasma of sALS patients and more importantly in fast progressing disease. Immune markers CCL16 and sTNF-RII were significantly increased in rapidly progressing disease. We also found that leptin significantly downregulated in plasma of SOD1G93A mice across disease stage. This was caused by an increased in levels of phospho-AMPK in mice adipoocytes and in adipoocytes exposed to fast sALS patients’ plasma. Conclusions: We propose that the combination of decreased plasma leptin levels and up-regulation in CCL16/sTNF-RII may be used as a prognostic biomarker to identify fast progressing ALS patients. This unique immune/metabolic profile may cause dysfunction in metabolic homeostasis.

GP.3

Examining Aneurysmal Healing After Flow Diversion Treatment Using Endovascular Optical Coherence Tomography

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Background: The mechanism of aneurysmal healing after flow-diversion treatment of cerebral aneurysms remains unknown. The purpose of this research to is to utilize a novel technology called endovascular optical coherence tomography (OCT) to characterise and improve our understanding of aneurysmal healing after flow-diversion using a rabbit aneurysm model. Methods: Saccular aneurysms were created in 10 New Zealand white rabbits. The aneurysms were treated with a flow-diverting stent 28 days after creation. OCT and histopathologic examinations included: luminal thrombosis, endothelial loss, inflammation, fibrin, smooth muscle cell loss, disruption of the internal and external elastic lamina, and tunica adventitia changes.

Results: OCT revealed endothelialization across the stent, appearing to originate from the parent vessel, along with small amounts of thrombus on the stent-struts. Minimal thrombus was visualized within the aneurysm sac. Histologic examination revealed that OCT can accurately define endothelialization across the sent, and define patent segments across the neck. Conclusions: Aneurysmal healing appears to originate at the parent vessel/stent interface, and use the stent as a scaffold to grow across the neck of the aneurysm. Minimal thrombus was visualized within the aneurysm sac, with ongoing flow observed in the setting of incomplete neck endothelialization. This technology has great potential for assessing aneurysmal healing in real-time.