B.7

Rapid Glial Fibrillary Acidic Protein (GFAP) analysis in acute stroke: feasibility and diagnostic potential

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Background: Brain-specific glial fibrillary acidic protein (GFAP) can discriminate stroke type [ischemic stroke (AIS), intracerebral hemorrhage (ICH), stroke mimics (SM)]. Novel point-of-care technology (GFAP levels <15 minutes) is a promising diagnostic tool. We aim to evaluate the feasibility of rapid GFAP analysis in acute stroke. Methods: Exploratory analysis of an ongoing prospective study of suspected undifferentiated stroke <24h from onset. Rapid plasma GFAP levels (pg/mL) are measured at hospital arrival using the i-STAT Alinity® instrument and commercially-available cartridges. Study endpoints include quantitative GFAP levels according to final diagnosis and time from stroke onset. Results: Among 200 patients (mean(±SD) 70.7±15.5 years, 44.5% female, median (IQR) NIHSS 9(4-19), diagnosis was AIS (n=132 (59 large-vessel occlusion), ICH (n=17), and SM (n=51). Median time from hospital arrival to GFAP result was 56.0 (47.0-69.5) minutes. Median rapid GFAP levels were highest in ICH (878.0 (70.5-3,906.5) pg/mL) compared to AIS (49.5 (29.0-95)pg/mL) and SM (29(29-64)pg/mL), p=0.001. Median GFAP was higher in AIS-known onset >4.5h (n=9) (110.0 (44.0-216.0) pg/mL) compared to AIS<4.5h (40.5 (29.0-68.8) pg/mL) (n=72), (p=0.047), while AIS-unknown onset (n=51) (68.0 (29.0-108.5) pg/mL) fell between these two groups, likely reflecting the subgroup's heterogeneity. Conclusions: Preliminary findings suggest that rapid GFAP analysis is feasible in acute stroke and may inform treatment decisions.

Child Neurology (CACN)

C.1

Cognitive profile in pediatric seronegative autoimmune encephalitis

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Background: Seronegative autoimmune encephalitis (SAE) in children is associated with cognitive deficits, particularly executive dysfunction. However, the relationship between cognitive impairment, disease severity, and lesion burden remains unclear. Identifying these associations could improve patient management and outcomes. This study characterizes neuropsychological symptoms in pediatric SAE and compares patients with and without formal neuropsychological assessments to determine factors influencing cognitive impairment. Methods: A retrospective review was conducted on 155 pediatric autoimmune encephalitis cases, including 80 with SAE. Eleven had neuropsychological evaluations. Statistical analyses assessed differences in age, disease severity, lesion characteristics, hospitalization, and treatment needs. Results: Executive dysfunction was present in 75% of SAE cases. Patients with neuropsychological evaluations were older (median: 8 vs. 3 years, $p = 0.0115$) and had more severe encephalitis at admission ($p = 0.0391$) and one year later ($p = 0.0011$). Lesion burden did not differ ($p > 0.05$), but patients with assessments had longer hospitalizations and required more intensive treatments ($p < 0.005$). Conclusions: Executive dysfunction in pediatric SAE is linked to disease severity rather than lesion burden. Systematic neuropsychological assessments should be integrated into patient care. Deeper phenotyping of cognitive profiles and identifying risk factors for poor prognosis will help personalize care in order to improve outcomes.

C.2

Dexamethasone induced p57-mediated quiescence contributes to chemotherapy resistance in sonic hedgehog medulloblastoma

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Background: Medulloblastoma (MB), the most common malignant pediatric brain tumor, is often incurable upon recurrence, largely driven by treatment-resistant quiescent cells. While quiescent SHH MB populations have been identified, the mechanisms driving their chemoresistance remain unclear. Here, we investigate the role of the cell cycle inhibitor p57 in inducing quiescence and show that dexamethasone, widely used in MB management, promotes p57-mediated quiescence, potentially reducing treatment efficacy. Methods: To assess p57's role, we introduced a TMP-inducible p57 construct into *Ptch1*$^{+/-}$ SHH MB cells and treated them with vincristine. We also treated *Ptch1*$^{+/-}$ SHH MB cells with dexamethasone and quantified p57 levels and cell cycle states using high-throughput immunofluorescence imaging. Results: In culture, nuclear p57 was enriched in Sox2+ and Nestin+ stem-like SHH MB cells relative to rapidly-cycling Atoh1+ cells. Stabilizing p57 with TMP increased G_0-phase cells six-fold, exhibiting survival to vincristine doses that caused complete cell death in controls. Dexamethasone treatment increased nuclear p57 by 40% and G_0-phase cells by 15% in *Ptch1*$^{+/-}$ cells, while doubling G_0-phase cells in *Ptch1*$^{+/-}$;*Trp53*$^{-/-}$ cells. Conclusions: These findings suggest dexamethasone promotes p57-mediated quiescence, potentially contributing to chemoresistance in SHH MB. This raises critical concerns about the use of dexamethasone in MB treatment, as it may inadvertently enhance tumor recurrence.