

O-017

RELATIONSHIP BETWEEN IN-STENT RESTENOSIS FOLLOWING CAROTID ARTERY STENTING AND PLATELET REACTIVITY TO CLOPIDOGREL

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Background While platelets have been known to play a key role in the pathophysiology of ISR throughout the cascade of inflammatory responses leading to neointimal hyperplasia, the quantitative association between in-stent restenosis (ISR) and platelet function in patients treated with carotid artery stenting (CAS) remains poorly documented.

Objective To evaluate the degree of ISR following CAS using computed tomographic angiography (CTA), examine its relationship with platelet reactivity to clopidogrel, and determine the optimal thresholds of the P2Y12 reaction unit (PRU) and inhibition rate (IR) for identifying ISR.

Methods We retrospectively analyzed 171 patients who underwent CAS with extracranial carotid stenosis from January 2016 to December 2019. Dual antiplatelet therapy with 100 mg aspirin and 75 mg clopidogrel was started ≥ 5 days before CAS. Clopidogrel resistance was measured with the PRU and IR the day before CAS. The ISR degree was classified into R1 (mild luminal stenosis of $<50\%$ ranging $<50\%$ of the stented carotid artery total length), R2 (mild luminal stenosis of $<50\%$ ranging $\geq 50\%$ of the stented carotid artery total length), and R3 (moderate to severe luminal stenosis of $\geq 50\%$ or occlusion) through carotid CTA after 24–30 months. The quantitative association degree between platelet reactivity and ISR R3 was determined by the receiver operating characteristic curve method. The optimal cutoff values of PRU and IR were derived using the maximum Youden index.

Results There were 33 ISR of R3s (19.3%) and 9 ipsilateral ischemic strokes (5.3%). The PRU and IR were different between R1 + R2 (176.4 ± 50.1 , $27.5 \pm 18.7\%$) and R3 (247.5 ± 55.0 , $10.3 \pm 13.4\%$) ($p < 0.001$). The areas under the curves of PRU and IR were 0.841 and 0.781, and the optimal cutoff values were 220.0 and 14.5%, respectively. Multivariate logistic regression analysis showed that PRU ≥ 220 and IR $\leq 14.5\%$ were significant predictive factors for ISR R3 ($p < 0.001$, $p = 0.017$). ISR R3 was independently associated with ipsilateral ischemic stroke after CAS ($p = 0.012$).

Conclusions High PRU (≥ 220) and low IR ($\leq 14.5\%$) are related to ISR R3 following CAS, which may cause ipsilateral ischemic stroke. This is the first observational study to address the relationship between ISR following CAS and platelet reactivity to clopidogrel. Long-term ISR follow-up and modified antiplatelet preparation to improve ISR seems to be needed in patients with high clopidogrel resistance.

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O-018

BMI AND STROKE OUTCOMES BY SUBTYPES: A PROPENSITY-SCORE MATCHED ANALYSIS IN 1,454,298 PATIENTS

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Background Frailty predicts poor outcomes after stroke. Although obesity increases stroke risk, studies have shown better outcomes in patients with higher BMI, a phenomenon known as the obesity paradox. We investigated the impact of low BMI on various stroke subtypes.

Methods We created cohorts based on ICD-10s for varying stroke subtypes (I60-I63, I66) utilizing the US-based TriNetX database. We assessed outcomes and stroke recurrence in patients with low BMI compared to propensity-score-matched cohorts with high BMI (≥ 25 kg/m² or ICD-10:E66) within 90 days.

Results After matching, our study consisted of 1,454,298 patients. There were 727,149 patients with low BMI separated into 5 cohorts (subarachnoid hemorrhage[SAH]:52,920; intracerebral hemorrhage[ICH]:77,212; subdural/extradural hemorrhage[S/EDH]:91,458; Cerebral infarction[CI]:475,156; Transient ischemic attack[TIA]:30,403) and 727,149 patients in the respective propensity-score matched cohorts with high BMI. All patients with low BMI had increased mortality and respirator dependence but fewer seizures, PE/DVT, and MIs. Patients with low BMI and SAH, S/EDH, CI, and TIA were more frequently hospitalized. Critical Care/ICU was utilized significantly more often by patients with low BMI and ICH, S/EDH, CI, and TIA. Emergency endotracheal intubation was performed significantly more often in patients with low BMI and CI or TIA. SAH was significantly more common in patients with low BMI after S/EDH. ICH was more common in patients with low BMI after ICH, S/EDH, and CI. Recurrent CI was significantly more common in patients with low BMI. CI was significantly less common in patients with low BMI after SAH, ICH, S/EDH, and TIA.

Conclusions Patients with low BMI and various stroke subtypes have higher mortality and respirator dependence, but fewer seizures, PE/DVT, and MI. The different recurrence associations of certain stroke subtypes underscore the complex interplay between BMI and stroke outcomes, emphasizing the need for tailored interventions and monitoring strategies for individuals with low BMI and stroke.

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