vascular shunting that is associated with liver cirrhosis. The patient's multiple cortical bleeds of different ages, compounded by coagulopathy from cirrhosis, increased the suspicion for cerebral amyloid angiopathy (CAA) etiology as well. The patient was scheduled for outpatient follow-up for a brain biopsy to prognosticate CAA and liver transplant candidacy. This case highlights the importance of considering complex interactions between liver and cerebral vascular pathologies in patients with atypical presentations and comorbidities.

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E-261 VARIABILITY OF RESPONSE ON PROPHYLACTIC PRASUGREL FOR ENDOVASCULAR TREATMENT OF INTRACRANIAL ANEURYSMS: CLINICAL IMPLICATIONS

Y Son*, N Hong. *Neurosurgery, SMG-SNU Boramae Medical Center, Seoul, Korea, Republic of*

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Introduction/Purpose Prophylactic prasugrel for endovascular treatment of intracranial aneurysms has been introduced and increased, but HTPR (high on-treatment platelet reactivity) or LTPR (low on-treatment platelet reactivity) of prasugrel is not uncommon in clinical circumstances. To investigate the predisposing factors of HTPR and LTPR on prasugrel premedication in the neurointerventional field and to determine its clinical implications.

Materials and Methods Between February 2016 and December 2020, 191 patients treated with coil embolization using prophylactic prasugrel in 234 intracranial aneurysms were the final candidates for this study. Patient and aneurysm characteristics, clinical status, and laboratory study values were carefully reviewed retrospectively. We performed risk factor analyses for HTPR and LTPR on prasugrel.

Abstract E-261 Table 1 Risk factor analysis of high on-treatment platelet reactivity (HTPR)

Variables	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	P	OR (95% CI)	P
Age	1.02 (0.97-1.06)	0.53		
Female over male	0.66 (0.25-1.77)	0.41		
BMI	1.17 (1.01-1.34)	0.03	1.21 (1.04-1.41)	0.01
Hypertension	1.93 (0.67-5.57)	0.22		
Diabetes mellitus	1.70 (0.57-5.06)	0.34		
Chronic kidney disease	0.00 (0.00-∞)	1.00		
Arrhythmia	0.00 (0.00-∞)	0.99		
Coronary heart disease	2.60 (0.50-13.49)	0.25		
Previous ischemic stroke	1.22 (0.38-3.93)	0.73		
Current smoking	1.08 (0.29-3.97)	0.91		
Alcohol consumption	0.60 (0.21-1.73)	0.35		
Drug history of antithrombotics	2.96 (1.16-7.58)	0.02	3.79 (1.39-10.34)	0.01
Drug history of proton-pump inhibitors	3.47 (1.12-10.87)	0.03		
Level of LDL cholesterol	1.00 (0.99-1.02)	0.89		
Level of HDL cholesterol	0.98 (0.94-1.01)	0.21		
Platelet count	0.99 (0.99-1.01)	0.65		
Hematocrit	0.94 (0.88-1.02)	0.13	0.91 (0.84-0.99)	0.03

BMI, body mass index; CI, confidence interval; high-density lipoprotein; LDL, low-density lipoprotein; OR, odds ratio

Abstract E-261 Table 2 Risk factor analysis of low on-treatment platelet reactivity (LTPR)

Variables	Univariate Analysis		Multivariate Analysis	
	OR (95% CI)	P	OR (95% CI)	P
Age	0.98 (0.95-1.01)	0.20		
Female over male	1.39 (0.71-2.72)	0.34		
BMI	0.86 (0.78-0.95)	0.004	0.84 (0.76-0.94)	0.001
Hypertension	0.87 (0.48-1.59)	0.65		
Diabetes mellitus	0.46 (0.19-1.08)	0.07		
Chronic kidney disease	0.00 (0.00-∞)	1.00		
Arrhythmia	0.97 (0.22-4.18)	0.97		
Coronary heart disease	1.31 (0.34-5.05)	0.70		
Previous ischemic stroke	1.06 (0.49-2.29)	0.88		
Current smoking	1.35 (0.59-3.08)	0.47		
Alcohol consumption	0.66 (0.35-1.23)	0.19		
Drug history of antithrombotics	0.64 (0.33-1.21)	0.17		
Drug history of proton-pump inhibitors	1.09 (0.42-2.80)	0.86		
Level of LDL cholesterol	1.004 (0.99-1.01)	0.43		
Level of HDL cholesterol	1.01 (0.99-1.03)	0.40		
Platelet count	1.003 (0.99-1.01)	0.12	1.004 (1.00-1.01)	0.05
Hematocrit	1.01 (0.96-1.07)	0.66		

BMI, body mass index; CI, confidence interval; high-density lipoprotein; LDL, low-density lipoprotein; OR, odds ratio

Results Ultimately, 20 patients (10.5%) had HTPR, and 74 patients (38.7%) were categorized as having LTPR. In multivariable analyses, the factors related to HTPR were BMI (adjusted OR 1.21, 95% CI 1.04-1.41, p = 0.01), history of antithrombotics (adjusted OR 3.79, 95% CI 1.39-10.34, p = 0.01), and hematocrit (adjusted OR 0.91, 95% CI 0.84-0.99, p = 0.03). Low BMI was the only risk factor for LTPR (adjusted OR 0.84, 95% CI 0.76-0.94, p = 0.001).

Conclusion In the neurointerventional field, high BMI and prior use of antithrombotic agents were related to HTPR, and low BMI was associated with LTPR on prophylactic prasugrel. High hematocrit levels decreased the risk of HTPR. When preparing endovascular treatment for intracranial aneurysms, attention to patients with these clinical features is required to address the possibility of ischemic or bleeding complications.

Disclosures Y. Son: None. **N. Hong:** None.

E-262 PIPELINE EMBOLIZATION DEVICE FOR THE TREATMENT OF UNRUPTURED INTRACRANIAL SACCULAR ANEURYSMS: A SYSTEMATIC REVIEW AND META-ANALYSIS OF LONG-TERM OUTCOMES

[1]A Ghaith*, [2]J Rios-Zermeno, [2]E Greco, [1]M Bydon, [2]R Tawk. *[1]Mayo Clinic, Rochester, MN, USA; [2]Mayo Clinic, Jacksonville, FL, USA*

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Introduction The Pipeline Embolization Device (PED) is commonly used to treat intracranial aneurysms. From its initial use, the indications expanded significantly to treating fusiform, blister, and dissecting intracranial aneurysms in the anterior and posterior circulation. Still, the treatment of saccular

aneurysms continues to be controversial due to the ample variety of management modalities, from endovascular to microsurgical. This study analyzes the long-term occlusion rate and complications in saccular aneurysms treated with PED.

Methods We performed a systematic literature review and meta-analysis of studies of any design, including a minimum of 10 patients treated with PED for saccular aneurysms with at least 12 months follow-up. The primary effectiveness endpoint was the complete aneurysm occlusion rate. The primary safety endpoint was the rate of clinical complications measured by the cumulative symptomatic stroke (confirmed clinically and radiographically), intracranial hemorrhage, and aneurysmal rupture.

Results Our analysis comprised 11 studies, including 594 patients with 726 aneurysms. Most aneurysms were unruptured (72%, N=427) and small (77.4%). The mean age was 55.74 years, and most patients were women (78.3%; N= 465). One device was used in 580 aneurysms, and coils were added in 21. Previously treated aneurysms were 5.8%. Most aneurysms were small (89.75%; N= 1,340 aneurysms). The long-term complete occlusion rate was 81% (95%, CI 72% to 88%, p<0.01). The long-term symptomatic thromboembolic complication rate was 1% (95%, CI 0% to 4%, p=0.07). The rupture rate was 1% (95%, CI 0% to 3%, p=0.02), and the rate of intracranial hemorrhage was 3% (95%, CI 1% to 6%, p=0.81).

Conclusion The PED is a safe and effective method to treat intracranial saccular aneurysms: The long-term complete occlusion rate is high (81%), with almost a quarter of the patients persist with residual filling. Even longer follow-ups are expected to show higher occlusion rates.

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E-263 LIQUID EMBOLIC SURFACE AREA AS A PREDICTOR OF CHRONIC SUBDURAL HEMATOMA RESOLUTION IN MIDDLE MENINGEAL ARTERY EMBOLIZATION

[1]K John*, [2]S Syed, [3]T Kaestner, [4]R Dashti, [4]D Fiorella, [4]C Sadasivan. *[1]Radiology, Stony Brook University Hospital, Stony Brook, NY, USA; [2]New York Medical College, Valhalla, NY, USA; [3]Siemens Medical Solutions USA, Inc., Malvern, PA, USA; [4]Neurological Surgery, Stony Brook Medicine, Stony Brook, NY, USA*

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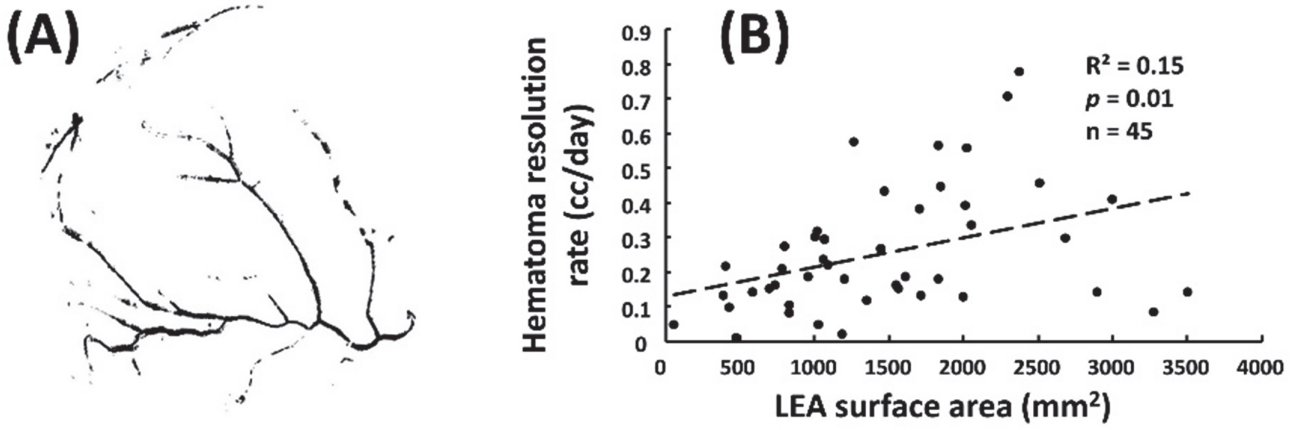
Introduction Liquid embolic agents (LEAs) such as cryanoacrylates or ethylene vinyl alcohol (EVOH) may be advantageous for middle meningeal artery embolization (MMAE) treatment of chronic subdural hematomas (cSDH) because they are radiopaque during fluoroscopy, can penetrate the microvasculature of the subdural membranes, allow for permanent embolization, and may be overall more effective radiographically for hematoma resolution compared to particulate agents. The segmentation of LEA surface area has not yet been described in the literature and is of interest in both refining MMAE technique and understanding cSDH pathophysiology.

Methods Under IRB approval, we retrospectively and anonymously collected CT scans from 74 patients (with 95 cSDH) who underwent first-line MMAE with an EVOH liquid embolic. Non-contrast head CTs acquired pre-embolization, and at 1-month (n=81), 3-month (n=69), and 6-month (n=52) follow-up, as well as immediate post-embolization flat-detector CTs were analyzed. Patients who received any neurosurgical intervention were excluded. 3D-Slicer (slicer.org) was used to segment and calculate hematoma volumes in the non-contrast CTs and the flat-detector CTs were segmented (Figure A) to calculate the surface area of the LEA. We hypothesized that greater LEA surface area would be correlated with greater improvements in cSDH volumetric resolution over follow-up imaging.

Results There was significant reduction in cSDH volumes over the follow-up period with respect to the preoperative volume (p<0.001). cSDH volumes did not differ significantly between 3-months and 6-months (p=0.9). The liquid embolic surface area was significantly correlated with the rate of cSDH resorption at 3-months (R^2=0.08, p=0.025, n=63) and at 6-months (Figure B). 33% of patients had complete hematoma resolution within 6 months, and this group showed a trend toward greater LEA surface area (1596±870mm^2) compared to the patients with residual hematomas (1298±695mm$^{\wedge 2}$) without statistical significance (p=0.15).

Conclusion Results indicate a plateau effect on cSDH resorption between the 3- and 6-month time frame after MMAE. LEA surface area (as opposed to volume) is a quantitative measure of distal penetration of the embolic cast. The positive correlation of LEA surface area with rate of hematoma resolution suggests greater LEA penetration can improve radiographic outcomes in cSDH patients. Surface area correlations to cSDH resolution were weak, which may be because the



Abstract E-263 Figure 1 (A) example of a MMAE liquid embolic cat segmented using a global threshold in 3-D slicer. This example has a surface area of 2683 mm^2. (B) liquid embolic agent (LEA) surface area correlated with rate of hematoma resolution at 6-month follow-up