performed at 24 hours post-procedure. Logistic regression and area under curve (AUC) characteristic were generated, with optimal PFT threshold determined by maximizing Youden's J statistic.

Results Forty-two of a total 94 PED consecutive procedures (44.7%) showed post-procedural ischemic lesions on DWI with two transiently symptomatic. The rate of hemorrhagic complications was zero. Baseline clinical, procedural, and radiographic parameters were similar between groups. P2Y12 reactivity was higher in ischemic patients (PRU 106.8 vs. 55.1, $p<0.0001$), and PRU was found to be a statistical predictor of ischemic lesion number and total surface area, with an increase in ten PRU increasing the odds of a DWI-positive lesion by seventeen percent (OR 1.017, CI 1.008-1.026) and AUC=0.74. The optimal PRU threshold was determined to be 100.5 (sensitivity 54.8%, specificity 82.7%), which is much lower than the generally accepted PRU threshold of 194.

Conclusions These results are the first to highlight the relationship between P2Y12 antagonism and DWI+ burden. Our findings confirm the important role of pre-procedural platelet function testing and post-procedural DW-MRI and lend support to the utility of the PRU assay for determining optimal reduction of PED-related peri-procedural ischemic events.

Disclosures C. Cebulla: None. **S. Burke:** None. **A. Malek:** 2; C; CereVasc Inc. 4; C; CereVasc Inc.

0-068 REGULATORY B CELL AND ADJUNCTIVE IMMUNE POPULATION DRIVES INTRACRANIAL ANEURYSM RUPTURE IN PATIENT POPULATION

J Antonios*, T Barak, B Gultekin, K Yalcin, A Chamberlain, R Hebert, C Matouk, M Gunel. *Neurosurgery, Yale, New Haven, CT, USA*

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Introduction Dynamic flow models have been useful in describing the development of intracranial aneurysms. However, our understanding of the mechanisms behind their remodeling and rupture is limited. Given that only a subset of patients who harbor this pathology go on to rupture, this question becomes especially relevant. There have been some studies, namely in reference to the natural history of thoracic and aortic aneurysms, that have described a role that regulatory B cells may play in rupture. However, they are limited in scope and application.

Hypothesis We hypothesized that there is an underlying immune driver that mediates the remodeling and rupture of intracranial aneurysms. Our goal was to describe the relevant immune populations and the phenotypic changes that occur between ruptured and unruptured pathologies. Ultimately, this study will inform the development of future immune-directed therapies for intracranial aneurysm management.

Methods We utilized endovascular techniques previously described to collect the endothelial lining of aneurysms in both ruptured and unruptured settings in our patient population. These cells were processed, sorted, and stained using the MaxPar Immune Profiling Panel surface markers (Standard Biotools, CA, USA) and data acquired using a time-of-Flight mass spectrometry (CyTOF Helios, Standard Biotools, CA, USA). We then developed Python clustering tools in conjunction with FlowJo Analysis Software (BD Biosciences, OR, USA) to perform unsupervised clustering visualization and analysis of high-parameter data.

Results We found a unique infiltrating population of regulatory B cells in ruptured aneurysms. This population is more prominent in high-grade ruptures and large, dysplastic aneurysms in the ruptured population. Further, we noted that while there are infiltrating immune populations in both ruptured and unruptured aneurysms, the myeloid/macrophage population in the ruptured aneurysms express a more inflammatory phenotype (associated with M2-type). Within the ruptured population, we compared patients with prior documented sentinel hemorrhages and re-ruptured aneurysms against new aneurysm ruptures. In the former population, we noted that there is an infiltrating memory T cell population that is not present in naïve ruptures.

Conclusion These findings all point to a complex, coordinated immune response that is driving both the remodeling of vessel wall and, ultimately, intracranial aneurysm rupture. This suggests that with further studies, we may be able to anticipate and prevent aneurysm rupture via adjunctive immune therapies.

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0-069 BRAIN ANEURYSMS: ENDOVASCULAR PHOTOBIOMODULATION TO ACCELERATE HEALING

[1]A Wakhloo*, [2]Y Uetake, [2]S Greenfield, [2]B Lieber. *[1]Radiology, TUFTS School of Medicine, Boston, MA, USA; [2]Radiology Beth Israel Lahey Health, TUFTS School of Medicine, Burlington, MA, USA*

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Background Endovascular treatment of brain aneurysms with the use of flow diverting stents, coils and intrasaccular flow disrupters, has been challenged by delayed and incomplete occlusion in more than 20% of treated subjects at 12-month follow-up imaging. Other shortcomings include delayed or incomplete aneurysm occlusion in senior population, early spontaneous rupture of large and giant aneurysms as well as continued aneurysm growth despite angiographic occlusion. Low Laser Light Therapy (LLLT, aka Photobiomodulation) is being used successfully for treatment of superficial birthmarks as well as ulcers of mouth mucosa.

Objective We developed a highly flexible optical microfiber technology to deliver LLLT in a preclinical aneurysm animal model and studied early healing response.

Methods Single highly flexible 0.014' optical microfiber system were developed to deliver low laser light through a standard microcatheter. Optimization of delivery technology was carried out in vitro studies using various human cerebrovascular/aneurysm replica. Subsequently the LLLT system was assessed in a rabbit elastase aneurysm model. Laser wavelength and energy dose calculations based on aneurysm surface calculation, was obtained from previous cell culture and animal studies. Aneurysms were treated with LLLT followed by coils or flow diverters (test group) or with FD or coil only (control group). Pre- and posttreatment angiograms as well as follow-up angiograms at 3- and 10 days were compared. Following angiograms, animals were sacrificed, and aneurysm and parent artery samples were processed for histology studies and immunofluorescence staining.

Results Control samples showed at 3 days still filling of the aneurysm and no significant endothelialization. Albeit partial filling, 3-day sample treated with LLLT, showed amorphous

clot filling the aneurysm with early endothelialization of the aneurysm neck and the implant within the parent vessel. At 10 days, FD treated with LLLT showed a complete aneurysm occlusion and neck endothelialization while control aneurysms were still patent with sparse endothelial cells attached to the FD at the aneurysm neck. At 10 days smooth muscle cell actin (SMA, non-specific for myoepithelial cells) and CD 31 (platelet endothelial cell adhesion molecule 1) were found in large numbers in samples treated with LLLT. Although coil compaction at 10 days was seen in both coiled groups, LLLT treated samples showed layering clot/content. Immunostaining revealed high concentration of Fibroblasts and Myofibroblast near the aneurysm wall small number of primarily Lymphocytes and Neutrophils inside the thrombus as compared to the coiled only aneurysms.

Conclusion Preliminary preclinical studies show that LLLT delivered in situ in an animal aneurysm model is feasible. Compared with the control group an early healing response with aneurysm scarring and neck occlusion is observed. Our technology represents a unique combination of implantable devices and LLLT, bringing regenerative medicine into endovascular therapeutic realm.

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O-070 IS IT SAFE TO COVER THE ANTERIOR CHOROIDAL ARTERY WITH FLOW DIVERSION DEVICES?

R Holland*, D Khatri, R Zampolin, N Haranhalli, D Altschul, A Brook, S Lee. *Neurosurgery, Montefiore Med Center/AECOM, Bronx, NY, USA*

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Introduction Unintended side branch occlusions after Flow Diversion device insertion (FD) for the treatment of intracranial arterial aneurysms have been reported for up to 15% of patients. Fortunately, the incidence of symptomatic side branch occlusion after FD appears to be more rare. However, it could be devastating if the side branch occlusion involves the anterior choroidal artery. We aim to review the incidence of angiographic occlusion rates of the anterior choroidal artery following coverage with FD and its clinical consequences.

Methods A retrospective review of distal ICA aneurysms (cavernous, ophthalmic, posterior communicating, and anterior choroidal artery) treated with FD at our institution since 2018 was performed. Coverage of anterior choroidal arteries and its patency was determined based on immediate post-endovascular treatment angiography and on the follow up diagnostic angiography, respectively. Clinical symptom was determined based on clinical examination. All patients received dual antiplatelet therapy before the endovascular treatment and for several months after the treatment. On the day of the procedure, Thromboelastogramy (TEG) was used to determine arachidonic acid (AA) and adenosine diphosphate (ADP) inhibition. Each patient's antiplatelet therapy was tailored or modified based on this testing.

Results The Anterior Choroidal Artery was covered with FD in 53 distal ICA aneurysm patients (M:F=8:45, mean age = 54.2 years). The location of aneurysms included cavernous (5), ophthalmic (37), posterior communicating artery (8), and anterior choroidal artery (3). The average time to follow-up was 409 days. The FD devices that were used include Pipeline Embolic Device (n=27), Surpass (n=25), and Fred (n=1). There were 2 patients (2/53, 3.77%) who had anterior choroidal artery occlusions. However, both patients were asymptomatic. 18 (34%) of the patients had subtherapeutic ADP inhibition based on TEG (defined as <70% inhibition at our institution). These patients had clopidogrel switched, mostly to prasugrel.

Conclusion Occlusion of the anterior choroidal artery after FD coverage rarely occurs and the incidence of clinically symptomatic occlusion appears to be rarer. Complications may have been alleviated with personalized dual anti-platelet therapy using TEG results.

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O-071 ESTIMATION OF OPTIMAL COIL PACKING DENSITY USING LAGRANGIAN PARTICLE TRACKING

[1]D Bass*, [2]L Marsh, [1]M Barbour, [3]V Chivukula, [1]P Fillingham, [1]L Kim, [4]A Aliseda, [1]M Levitt. [1]*Neurological Surgery, University of Washington, Seattle, WA, USA*; [2]*Department of Mechanical Engineering, University of Washington, Seattle, WA, USA*; [3]*Department of Biomedical and Chemical Engineering, Florida Institute of Technology, Melobourne, FL, USA*; [4]*Mechanical Engineering, University of Washington, Seattle, WA, USA*

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Background Aneurysm coil packing density has been studied as a surrogate for treatment outcome, but clinical results are conflicting. Previous work applying computational fluid dynamics simulations to coil embolization of cerebral aneurysms found that embolization increases residence time (RT) and lowers cumulative shear (shear history; SH) of blood components within the aneurysm dome, suggesting that embolization promotes clot formation via a low shear stress-mediated pathway associated with stagnation of flow.[1] The goal of this investigation is to determine whether RT and SH can be used to identify optimal coil packing density for coiled cerebral aneurysm outcome prediction, using particle tracking simulations.

Method Computational fluid dynamics simulations of patient-specific aneurysms were performed before and after coil embolization treatment. Massless particles were virtually injected and individually tracked in each simulation, and blood flow was simulated with patient-specific boundary conditions. The coil mass was treated as a porous medium with a porosity corresponding to the *in vivo* treatment packing density. Simulations were also run with porosities corresponding to 50%, 150%, and 200% of the *in vivo* treatment packing density for each subject.

Results Five subjects were included. The relative decrease in the rate of particles entering an aneurysm had a strong correlation with increasing packing density ($R^2 = 0.944$, $P < 0.001$). A packing density of approximately 33.3% resulted in approximately a 70% reduction in particles entering the aneurysm. Above this packing density, the absolute number of particles was considered to be too low to be statistically reliable, and therefore further particle tracking analyses focused on simulations with packing densities less than 33.3%. Within these simulations, increasing packing density was associated with a linear increase in RT ($R^2 = 0.462$, $P < 0.003$) and a decrease in SH ($R^2 = 0.547$, $P < 0.001$), with SH nearing 0 as packing density approached 33.3%.