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

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10.1136/jnis-2023-SNIS.68

**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.

**Disclosures J. Antonios:** None. **T. Barak:** None. **B. Gultekin:** None. **K. Yalcin:** None. **A. Chamberlain:** None. **R. Hebert:** None. **C. Matouk:** None. **M. Gunel:** None.

## 0-069 BRAIN ANEURYSMS: ENDOVASCULAR PHOTOBIOMODULATION TO ACCELERATE HEALING

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10.1136/jnis-2023-SNIS.69

**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