Background Aneurysmal subarachnoid hemorrhage (aSAH) is a devastating disease frequently leading to death or poor functional outcome. A major source of disability from aSAH is the development of cerebral vasospasm, which is defined as narrowing of the large and medium-sized intracranial arteries. Limited information exists regarding underlying anatomic mechanisms of vasospasm after aSAH. Based on the anatomical location of resident herpesvirus and their activation in response to adrenergic stress, we propose that herpesvirus reactivation in response to adrenergic activation of head and neck ganglia during aSAH will be temporally related to cerebral vasospasm.

Methods We developed an IRB-approved protocol for non-invasive bedside testing of viral shedding in tears and saliva in aSAH patients. The protocol was a joint effort with Infectious Disease and our virology laboratory. Viral specimens and catecholamines were obtained at admission and at days 4, 7, 10 and 14 post-aSAH. These values were compared to standard-of-care metrics including transcranial doppler, clinical examination and radiological studies. Herpesvirus serology was also obtained.

Results Our protocol successfully yielded samples for analysis in all cases. Initially, serum catecholamines were utilized but collection methodology and requirements resulted in unusable samples. Further, many patients require pressor support using parenteral catecholamines and serum results may not be valid during hospitalization. Instead, salivary alpha-amylase is being tested as a surrogate marker, with collection limited to those patients not requiring catecholamine pressor support within the previous 24 hours. In our preliminary dataset, integrity for evaluation. We present the tissue preservation protocol for dissemination and highlight the analytical challenges presented by low biomass, high host-contamination specimens. In addition, we discuss the benefits of room (ambient) temperature preservation media compared to others requiring refrigeration or freezing for storage and transport.

Conclusion We have developed a novel tissue banking protocol to preserve microbiota from arterial thrombi retrieved from ischemic stroke patients. While there is no consensus which class of pathogens is implicated in the susceptibility to stroke, multiple studies including our own, have shown bacterial fragments and communities in cerebral thrombi. The specimens captured during standard-of-care thrombectomy allow us to characterize and functionally define the microbiota associated with cerebral thrombi in ischemic stroke patients.

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