vascular malformations (4.5%), moya moya disease, and carotid cavernous fistulas. In our case, no identifiable source of rupture was discovered on initial and delayed repeat cerebral angiography. Several authors have hypothesized that hemodynamic stress and defects in the wall of the cavernous ICA or the PTA may account for cases of non-aneurysmal SAH in patients with PTA.

Methods We present a case of a rare anatomical variant of a persistent trigeminal anastomosing directly with the superior cerebellar artery in a patient presenting with non-aneurysmal subarachnoid hemorrhage.

Results No identifiable malformation or aneurysm was discovered to explain the presence of subarachnoid hemorrhage. The patient underwent repeat cerebral angiography on post bleed day 7 which remained negative for aneurysm or source of rupture. The patient recovered fully and was discharged home.

Conclusion To our knowledge, this is the first description of a non-aneurysmal subarachnoid hemorrhage in a patient with an extremely rare Saltzman Type IIIa PTA variant. Recognizing PTA and its variants has important clinical implications especially in patients undergoing endovascular procedures or in patients with atherosclerotic carotid disease.

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E-194 ONE ROAD TO TWO PLACES: A UNIQUE CASE REPORT OF BILATERAL THALAMIC INFARCTIONS WITH NEUROANATOMICAL ANOMALY

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Case Presentation A 61-year-old male with a PMHx of MI status post PCI, HTN, and HLD presented after being found unresponsive. On arrival to the ED, the patient had altered mental status with GCS of 8 and was intubated. The patient was not a thrombolytic candidate due to being outside the therapeutic window. On subsequent physical examination, he was found to have vertical gaze palsy on both upwards and downwards gaze. CT Head and CTA Head and Neck were negative for acute hemorrhage or LVO. However, MRI Brain WO showed bilateral thalamic infarcts with left thalamic hemorrhage making an occlusion of an Artery of Percheron neuroanatomical variant the most likely etiology. An extensive hypercoagulable workup showed the possibility of mild antithrombin 3 deficiency. Dual-antiplatelet therapy was initiated and the patient made substantial recovery.

Discussion Stroke is a frequent acute neurological presentation to an emergency department; however, strokes presenting as encephalopathy without clear lateralizing features require a high index of suspicion for appropriate recognition and management. The present case included altered mental status, memory impairment, and vertical gaze palsy, representing a triad consistent with artery of Percheron infarct, however given intact horizontal gaze, symptoms would have been non-lateralizing on NIHSS testing. Subsequent CT Head and MRI Brain demonstrated isolated bilateral thalamic infarcts with evidence of left thalamic hemorrhagic. These findings are a classic presentation for occlusion of the artery of Percheron, a neuroanatomical vascular variant of one artery arising from the Posterior Cerebral Artery supplying bilateral paramedian thalami. The Artery of Percheron is a rare entity, estimated to occur in 4% of the population with occlusion resulting in CVA even more rare (Lazzaro et al. 2010). It is important to consider bilateral ischemic pathology, even as might arise from a single artery, as a differential in cases of acute encephalopathy.

Conclusion The scientific relevance of the present case is to address various etiologies of bilateral thalamic stroke with special attention to single artery causes of bilateral strokes versus other causes of bilateral thalamic strokes. It is important to consider bilateral ischemic pathology, even as might arise from a single artery, as a differential in cases of acute encephalopathy. Reviewing the various etiologies of cerebral infarction can improve patient outcomes and provide a comprehensive understanding of the patients' presenting symptomatology.

A practice gap exists in the identification and treatment of bilateral thalamic stroke etiologies. Given that these rare strokes typically present in the classic triad of AMS, memory impairment and vertical gaze palsy, it can be reasonable to miss the diagnosis in a high acuity setting such as a stroke alert that places high emphasis on criteria such as the NIHSS. As the NIHSS considers lateral gaze palsies, vertical extraocular movements may not be evaluated in the ED.

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E-195 ANGIOGRAPHIC ANATOMY OF SACRAL DURAL ARTERIOVENOUS FISTULAS

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Background and Purpose The identification of sacral dural arteriovenous fistulas (DAVFs) can be challenging. We sought to review our experience, and the published literature, for the arterial supply and venous drainage of sacral DAVFs.

Methods We retrospectively reviewed our electronic medical record for patients with sacral DAVFs diagnosed at our institution. Catheter angiograms were reviewed for arterial supply and venous drainage. We reviewed the published literature for this information as well.

Results We identified three patients with sacral DAVFs diagnosed on catheter angiography between January 2016 and November 2022. One arose at S1 and the other two at S2. One (figure 1 - at S1) was supplied by the median sacral artery (MSA). The remaining two were supplied by branches of the lateral sacral artery (LSA). All three drained into the Filum Terminale Vein (FTV) before anastamosis with the coronal venous plexus of the cord. Published reports limit the supply of sacral DAVFs to the MSA, the LSA or the Iliolumbalis artery (ILA). All drained via the FTV.

Conclusions 1. A dilated FTV on cross-sectional imaging in a patient with a suspected spinal DAVF should raise suspicion for a sacral origin. 2. Selective injections of the MSA, LSA and ILA should be performed in the hunt for a spinal DAVF.