Thalamus Arteriovenous Malformation

A 70-year-old gentleman with history of hypothyroidism, hyperlipidemia, hypertension, and right superior cerebellar aneurysm presented to the neurosurgery service in 2008 with vertigo. Diagnostic cerebral angiography performed that year demonstrated a vermian arteriovenous malformations (AVM). The patient underwent stereotactic proton beam radiosurgery, which resulted in a decrease in flow and size of the lesion, and the patient was lost to follow-up. Now at the age of 80, the patient presented with acute gait instability. Cerebral angiogram demonstrated his stable vermian AVM and a new 1.1 cm AVM nidus in the region of the left posterior thalamus. Although AVMs are often described as congenital lesions, there is a growing body of literature suggesting that AVMs can grow, spontaneously regress, and even arise de novo in response to some insult. Understanding what leads to the growth, remodeling, regression, and hemorrhage of AVMs is crucial in order to better direct therapeutic endeavors. We would argue that this patient's AVM is secondary to endothelial cell damage from radiation therapy. Radiation can cause endothelial cell injury and upregulation of factors, such as vascular endothelial growth factor and transforming growth factor beta expression, which are implicated in AVM development pathways. We believe that this patient's new AVM is secondary to entrance radiation dosing affecting the thalamus during radiation therapy for the original vermian AVM [1)].

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Torres-Quinones C, Koch MJ, Raymond SB, Patel A. Left Thalamus Arteriovenous Malformation Secondary to Radiation Therapy of Original Vermian Arteriovenous Malformation: Case Report. J Stroke Cerebrovasc Dis. 2019 Jun;28(6):e53-e59. doi: 10.1016/j.jstrokecerebrovasdis.2019.03.033. Epub 2019 Apr 8. PubMed PMID: 30975463.