



The time frame of the brain AVM formation

Masaki Komiyama

The authors of *Choroid plexus AVM with anomalous origin of the capsulothalamic artery: a case report* believe the time frame of arteriovenous malformation (AVM) formation is in early gestation,¹ a belief which is shared by the author of this letter. In other words, a primordial, abnormal vascular structure caused by developmental failure of the embryos in the 40–80 mm length interval (approximately 10–14 weeks of gestation) persists during the entire gestational and the postnatal periods, and then becomes symptomatic.² In the letter, the time frame of developmental failure is described at the 30 mm interval. To my knowledge, however, there has been no proof of developmental failure of the cerebral vasculature for the pathogenesis of brain AVM at this stage, and a later event may be more likely.

Although there are rare exceptional forms of brain AVMs, such as vein of Galen aneurysmal malformations and dural sinus malformations with arteriovenous shunts in the neonatal period, the above idea (congenital formation of brain AVMs) is not necessarily true because recent recognition of late presentation of brain AVMs suggests a postnatal formation of most of brain AVMs.^{3,4} This is supported by the evidence of extremely low incidence of brain AVMs in patients younger than 6 years, rarest of all in neonatal and infantile periods.⁵ De novo formation of brain AVMs reported in the literature also supports the idea of postnatal formation of the AVMs.^{6,7} Furthermore, the rare presentation of brain AVMs in the neonatal and/or infantile periods does not support the idea that brain AVMs are formed in early gestation, suggesting rather that they could be formed in late gestational period sufficiently after the initial normal development of the brain vasculature (approximately 8 weeks of gestation).

Declaration of conflicting interests

The authors declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Funding

The authors received no financial support for the research, authorship, and/or publication of this article.

References

1. Yamauchi S, Kawakami T, Murata K, et al. Choroid plexus AVM with anomalous origin of the capsulothalamic artery: a case report. *Interv Neuroradiol* 2018; 24: 76–81.
2. Mullan S, Mojtahedi S, Johnson DL, et al. Embryological basis of some aspects of cerebral vascular fistulas and malformations. *J Neurosurg* 1996; 85: 1–8.
3. Lasjaunias P. A revised concept of the congenital nature of cerebral arteriovenous malformations. *Interv Neuroradiol* 1997; 3: 275–281.
4. Komiyama M. Pathogenesis of brain arteriovenous malformations. *Neurol Med Chir (Tokyo)* 2016; 56: 317–325.
5. Terada A, Komiyama M, Ishiguro T, et al. Nationwide survey of pediatric intracranial arteriovenous shunts in Japan-Japanese Pediatric Arterio-venous shunts Study (JPAS). *J Neurosurg Pediatr* 2018; 22: 550–558.
6. Friedman JA, Pollock BE and Nichols DA. Development of a cerebral arteriovenous malformation documented in an adult by serial angiography. *Case report. J Neurosurg* 2000; 93: 1058–1061.
7. Shimoda Y, Osanai T, Nakayama N, et al. De novo arteriovenous malformation in a patient with hereditary hemorrhagic telangiectasia. *J Neurosurg Pediatr* 2016; 17: 330–335.

Editor-in-Chief, Interventional Neuroradiology, Department of Neuro-Intervention, Osaka City General Hospital, Osaka, Japan

Corresponding author:

Masaki Komiyama, Editor-in-Chief, Interventional Neuroradiology, Department of Neuro-Intervention, Osaka City General Hospital, 2-13-22, Miyakojima-Hondori, Miyakojima, Osaka, Japan 534-0021.
 Email: komiya@japan-mail.com