Neurointervention 2020;15:2-3
https://doi.org/10.5469/neuroint.2020.00045

Where Did the Dura Mater Come from?

Dae Chul Suh, MD, PhD

Neurointervention Clinic, Department of Radiology, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

The meninges provide a protective cover to the brain and the spinal cord and attach to the bony skull or the vertebral column, and also contain cerebrospinal fluid space. The dura mater is a two-layered membrane attached to the inner surface of the skull.[1,2] The outer (endosteal or periosteal) layer serves as the periosteum of the internal surface of the skull bone. The inner layer (meningeal or dura mater proper) is fused to the endosteal layer in most regions, and they are separated at the dural venous sinuses. The dural reflections of the 2 meningeal layers fold and invaginate into the cranial cavity by forming the falx cerebri and the tentorium cerebelli.

Embryonic development of the meninges has been studied for more than a hundred years.[3] The primary meninx (also known as primitive meninx or meninx primitiva) give rise to the meninges, the calvaria, and the dermis of the scalp. Differentiation of the meninges progresses from a basal to apical direction. The meninges even provide a stem cell niche. During development and growth of the rostral central nervous system, the dura mater regulates events in the underlying brain and overlying skull through the release of soluble factors and cellular activity.[4] Interactions of the dura mater with the brain and skull are so dynamic that they exhibit mechanical and biochemical reciprocity. The dura mater has a profound influence on cell migration and differentiation in multiple regions of the embryonic and infant brain and skull.[4]

Early experiments on quail and chick chimeras showed that neural crest-derived cells, generated from caudal forebrain and midbrain levels, contribute to the meninges associated with the forebrain.[5] In contrast, mesoderm-derived cells give rise to the meninges of the midbrain and the hindbrain. Histological observations in human fetuses also suggest that the cranial meninges originated from both the neural crest and the mesoderm.[3,6] The most striking diversity of the neural crest derivatives is found in its cephalic domain where the neural crest replaces the role of mesoderm.[7] The neural crest is a vertebrate-specific migratory stem cell that generates diverse cell types and structures. Comparative analysis suggests that neural crest cells in the vertebrates evolved at least 3 specific features—multipotency, long-range migration, and cellular communication systems for guidance.[8]

Although the pathogenesis of dural arteriovenous fistula (DAVF) still remains unclear, sinus thrombosis, head trauma, surgery, and hormonal influence are the predisposing factors that initiate this disease. Current classifications of DAVF mainly focus on the presence of leptomeningeal reflux related to cerebral

Correspondence to:
Dae Chul Suh, MD, PhD
Neurointervention Clinic, Departments of Radiology and Research Institute of Radiology, Asan Medical Center, University of Ulsan College of Medicine, 88 Olympic-ro 43-gil, Songpa-gu, Seoul 05505, Korea
Tel: +82-2-3010-4366
Fax: +82-2-3010-0090
E-mail: dcsuh@amc.seoul.kr

Received: February 17, 2020
Revised: February 24, 2020
Accepted: February 24, 2020

pISSN 2093-9043
eISSN 2233-6273

venous hypertension leading to cerebral infarction or hemorrhage. Presentation patterns can be different according to lesion location. Progression of DAVF may reveal 3 phases (proliferative, restrictive and late restrictive), especially in the cavernous sinus DAVF.[9] Regardless of the locations or phases of DAVF, presence of the cortical venous reflux is an important feature to access bleeding risk of DAVF. However, direct communication from the fistula to the pial vein leading to brain edema or hemorrhage due to the pial venous reflux may occur in some areas of the calvarial convexity.[10]

The pial venous reflux from the trans-dural or trans-osseous feeders to the pial veins requires that the fistula crosses the subarachnoid space via the transdural emissary-bridging vein.[11,12] Such crosses may be possible either through the isolated cortical vein (caused by reasons like thrombosis or adhesion in the cortical vein) or other channels (like the emissary-bridging vein, which does not drain into the cortical vein in the subarachnoid space and does drain directly into the venous sinus or the extradural vein via certain trans-dural segments, as in the spinal DAVF).[13]

Such dural shunts with the direct pial venous reflux seem to be observed at the overlapped border of the primary meninx forming meninges and calvaria, which are separately originated from the neural crest in meninges, and mesoderm in calvaria, as in the areas like the parietal convexity.[3,14]

Fund

This work was supported by the National Research Foundation of Korea (NRF) grant funded by the Korea government (MSIT) (No. 2018R1A2B6003143).

Ethics Statement

This study waived approval of the institutional ethics committee.

Conflicts of Interest

The author has no conflicts to disclose.

REFERENCES

1. Tanaka M. Embryological consideration of dural avfs in relation to the neural crest and the mesoderm. *Neurointervention* 2019;14:9-16
2. Adeeb N, Mortazavi MM, Tubbs RS, Cohen-Gadol AA. The cranial dura mater: a review of its history, embryology, and anatomy. *Childs Nerv Syst* 2012;28:827-837
3. Dasgupta K, Jeong J. Developmental biology of the meninges. *Genesis* 2019;57:e23288
4. Gagan JR, Tholpady SS, Ogle RC. Cellular dynamics and tissue interactions of the dura mater during head development. *Birth Defects Res C Embryo Today* 2007;81:297-304
5. Le Douarin NM, Couly G, Creuzet SE. The neural crest is a powerful regulator of pre-otic brain development. *Dev Biol* 2012;366:74-82
6. Etchevers HC, Dupin E, Le Douarin NM. The diverse neural crest: from embryology to human pathology. *Development* 2019;146:dev169821
7. Dupin E, Calloni GW, Coelho-Aguiar JM, Le Douarin NM. The issue of the multipotency of the neural crest cells. *Dev Biol* 2018;444 Suppl 1:S47-S59
8. York JR, McCauley DW. The origin and evolution of vertebrate neural crest cells. *Open Biol* 2020;10:190285
9. Suh DC, Lee JH, Kim SJ, Chung SJ, Choi CG, Kim HJ, et al. New concept in cavernous sinus dural arteriovenous fistula: correlation with presenting symptom and venous drainage patterns. *Stroke* 2005;36:1134-1139
10. Zhao LB, Suh DC, Lee DG, Kim SJ, Kim JK, Han S, et al. Association of pial venous reflux with hemorrhage or edema in dural arteriovenous fistula. *Neurology* 2014;82:1897-1904
11. Baltsavias G, Parthasarathi V, Aydin E, Al Schameri RA, Roth P, Valavanis A. Cranial dural arteriovenous shunts. Part 1. Anatomy and embryology of the bridging and emissary veins. *Neurosurg Rev* 2015;38:253-263; discussion 263-264
12. Geibprasert S, Pereira V, Krings T, Jiarakongmun P, Toulgoat F, Pongpech S, et al. Dural arteriovenous shunts: a new classification of craniospinal epidural venous anatomical bases and clinical correlations. *Stroke* 2008;39:2783-2794
13. Cho SH, Suh DC. Transdural segment of the radicular vein in spinal dural arteriovenous fistula. *Neurointervention* 2017;12:57-58
14. Dasgupta K, Chung JU, Asam K, Jeong J. Molecular patterning of the embryonic cranial mesenchyme revealed by genome-wide transcriptional profiling. *Dev Biol* 2019;455:434-448