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Letter to the Editor: Perianeurysmal Edema

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15. Jho HD, Ha HG: Endoscopic endonasal skull base surgery: Part 1—The midline anterior fossa skull base. **Minim Invasive Neurosurg 47:**16–23, 2004
16. Jones TM, Almahdi JM, Bhalla RK, Lewis-Jones H, Swift AC: The radiological anatomy of the anterior skull base. **Clin Otolaryngol Allied Sci 27:**101–105, 2002
17. Kaptain GJ, Vincent DA, Sheehan JP, Laws ER Jr: Transsphenoidal approaches for the extracapsular resection of midline suprasellar and anterior cranial base lesions. **Neurosurgery 49:**94–101, 2001
18. Kassam A, Snyderman CH, Mintz A, Gardner P, Carrau RL: Expanded endonasal approach: the rostrocaudal axis. Part I. Crista galli to the sella turcica. **Neurosurg Focus 19(1):**E3, 2005
19. Kawahara G, Matsuda M, Sugiyama K, Nakazawa R, Shima K: [Studies on the Japanese lamina cribrosa—statistical observation on its shape, number of pores and area.] **Zasshi Tokyo Ika Daigaku 26:**185–194, 1968 (Jpn)
20. Keros P: [On the practical value of differences in the level of the lamina cribrosa of the ethmoid.] **Z Laryngol Rhinol Otol 41:**809–13, 1962 (Ger)
21. Kuhn FA, Javer AR: Low-grade fibrosarcoma of the anterior skull base: endoscopic resection and repair. **Am J Rhinol 17:**347–350, 2003
22. Lee JM, Ransom E, Lee JY, Palmer JN, Chiu AG: Endoscopic anterior skull base surgery: intraoperative considerations of the crista galli. **Skull Base 21:**83–86, 2011
23. Lee JT, Kingdom TT, Smith TL, Setzen M, Brown S, Batra PS: Practice patterns in endoscopic skull base surgery: survey of the American Rhinologic Society. **Int Forum Allergy Rhinol 4:**124–131, 2014
24. Liu JK, Christiano LD, Patel SK, Tubbs RS, Eloy JA: Surgical nuances for removal of olfactory groove meningiomas using the endoscopic endonasal transcribriform approach. **Neurosurg Focus 30(5):**E3, 2011
25. Liu JK, Eloy JA: Expanded endoscopic endonasal transcribriform approach for resection of anterior skull base olfactory schwannoma. **Neurosurg Focus (Suppl) 32(1):**Video 3, 2012
26. Vasvari G, Reisch R, Patonay L: Surgical anatomy of the cribriform plate and adjacent areas. **Minim Invasive Neurosurg 48:**25–33, 2005
27. Verillaud B, Bresson D, Sauvaget E, Mandonnet E, Georges B, Kania R, et al: Transcribriform and transplanum endoscopic approach for skull-base tumors. **Eur Ann Otorhinolaryngol Head Neck Dis 130:**233–236, 2013
28. Weiss DD, Robson CD, Mulliken JB: Transnasal endoscopic excision of midline nasal dermoid from the anterior cranial base. **Plast Reconstr Surg 102:**2119–2123, 1998
29. Wigand ME, Hosemann WG: Results of endoscopic surgery of the paranasal sinuses and anterior skull base. **J Otolaryngol 20:**385–390, 1991
30. Zacharek MA, Han JK, Allen R, Weissman JL, Hwang PH: Sagittal and coronal dimensions of the ethmoid roof: a radioanatomic study. **Am J Rhinol 19:**348–352, 2005

Response

We thank the authors for providing this thoughtful discussion of the nomenclature and anatomy of the endonasal approach to the anterior fossa. The authors are correct to point out that the term "transcribriform approach" is often used incorrectly to refer to either the actual transcribriform approach or the transethmoidal-transfovea-ethmoidalis approach, or a combination of the two.[1] As the authors mention, our group at Cornell discussed this distinction in our paper on the subject. We agree with the authors that it is critically important to differentiate the two approaches since the transcribriform approach by itself does not, in principle, require one to open the ethmoid air cells. However, in practice it is almost impossible to appropriately address pathology of the cribriform plate without also opening the ethmoid air cells since the corridor is so narrow. In the paper in question, we used the terminology "transcribriform-transethmoidal" to refer to the combination of approaches that included the "transcribriform," the "transethmoidal-transfovea-ethmoidalis," and the combination of the two approaches. The approaches were grouped together so that we could have high enough numbers of cases to try and achieve statistical significance with regard to patterns of pneumocephalus. Nevertheless, we should have been more clear about our definitions and very much appreciate the opportunity to clarify our meaning.

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Reference

1. Greenfield JP, Anand VK, Kacker A, Seibert MJ, Singh A, Brown SM, et al: Endoscopic endonasal transethmoidal transcribriform transfovea ethmoidalis approach to the anterior skull base and anterior cranial fossa. **Neurosurgery 66:**883–892, 2010

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Perianeurysmal edema

TO THE EDITOR: We read with great interest the article by Pahl et al.[4] (Pahl FH, de Oliveira MF, Ferreira NPFD, et al: Perianeurysmal edema as a predictive sign of aneurysmal rupture. *J Neurosurg 121:*1112–1114, November 2014). The authors reported on 2 cases of middle-aged women with previous histories of headaches in which MRI examinations revealed the presence of surrounding parenchymal edema related to aneurysms. These findings were attributed to a progressive inflammatory process possibly triggered by enlargement of the bleb formation.[4] In a recent report by Nussbaum et al., the authors (one of whom is an author of this letter) described 13 patients with hemosiderin staining of the pial surface immediately adjacent to the aneurysm dome, suggesting a remote and unrecognized history of microbleeding from an aneurysm.[3] We identified that those patients presented with a recurrent history of episodic, unusual type of headaches, mimicking flu-like symptoms lasting for several days. In all cases, a diagnosis of a sentinel bleed or subarachnoid hemorrhage had never been suggested, and no patient had been admitted to the hospital for formal evaluation of prior headache episodes. In addition, the intervals between these headaches shortened between clinical presentations.[3]

Similar to those reported by Pahl et al., most cases in our series were female patients; 53.8% of these patients had a history of smoking, 30.8% had hypertension, and 23.1% had a history of alcohol abuse. Dyslipidemia and a family history of aneurysms were present in 15.4% of the patients, and hypercholesterolemia was noted in 1 patient (8%). The aneurysms were considered particularly "thin-walled" in 8 cases.[3]

Some authors have highlighted the role of surrounding brain edema as an early manifestation in the course to aneurysm rupture.[1] They have also suggested that the edema might be a result of a progressive inflammatory process culminating with rupture.[4] We are very aware of the fact that inflammation and apoptosis occur intraluminally, from the intima-internal elastic lamina layer (triggered by hemodynamic stress) and spread toward the media and adventitia.[5] A description by Pahl et al. of perianeurysmal edema portrays it as basically intraparenchymal in location. The possibility exists that perianeurysmal edema is nothing more than a progressive inflammatory process caused by a microbleed at the aneurysm site.

Finally, based on prior reports, it may also be possible to identify radiological evidence of minor hemorrhages by performing thin-cut studies utilizing T2 gradient recall echo or MRI–susceptibility weighted imaging (SWI) techniques. MRI-SWI is 3–6 times more sensitive than conventional T2-weighted gradient echo sequences for hemosiderin and perianeurysmal edema detection.[2] We are currently investigating the use of this technology when evaluating patients with "unruptured" aneurysms who present with a suspicious history of remote atypical headache.[2]

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DISCLOSURE
The authors report no conflict of interest.

References

1. Hiu T, Tsutsumi K, Kitagawa N, Hayashi K, Ujifuku K, Yasunaga A, et al: Progressive perianeurysmal edema preceding the rupture of a small basilar artery aneurysm. **Clin Neurol Neurosurg 111:**216–219, 2009
2. Mittal S, Wu Z, Neelavalli J, Haacke EM: Susceptibility-weighted imaging: technical aspect and clinical applications, part 2. **AJNR Am J Neuroradiol 30:**232–52, 2009
3. Nussbaum ES, Defillo A, Zelensky A, Pulivarthi S, Nussbaum L: "Microbleeding" from intracranial aneurysms: Local hemosiderin deposition identified during microsurgical treatment of unruptured intracranial aneurysms. **Sur Neurol Int 5:**28, 2014
4. Pahl FH, de Oliveira MF, Ferreira NPFD, de Macedo LL, Brock RS, de Souza VC: Perianeurysmal edema as a predictive sign of aneurysmal rupture. **J Neurosurg 121:**1112–1114, 2014
5. Turjman AS, Turjman F, Edelman ER: Role of fluid dynamics and inflammation in intracranial aneurysm formation. **Circulation 129:**373–382, 2014

Response

We completely agree with points stated by Defillo and Kennedy in their letter. Subarachnoid hemorrhage (SAH) following intracranial aneurysmal rupture is a major cause of morbidity and mortality. Much effort has been made toward avoiding aneurysmal rupture and establishing prediction patterns. Although several factors may interfere with the probability of rupture, such as smoking; use of alcohol; size, shape, and location of the aneurysm; presence of intraluminal thrombus; and even the sex of the patient, there are still scarce data to correlate such findings with the timing of aneurysmal rupture.

In this light, after a pooled analysis of 6 prospective cohort studies, Greving et al.[1] proposed a score to estimate the 5-year aneurysm rupture risk (PHASES score). They evaluated ruptures that occurred in 230 patients during 29,166 person-years of follow-up. The mean observed 1-year risk of aneurysm rupture was 1.4% (95% CI 1.1%–1.6%) and the 5-year risk was 3.4% (95% CI 2.9%–4.0%).

Predictors were age, hypertension, history of SAH, aneurysm size, aneurysm location, and geographic region. According to the given score, which may vary from 0 to 22 points, 5-year rupture risk may be between 0.4% and 17.8%.[1]

Additionally, Korja et al.[2] published their observations after following 118 Finnish patients with unruptured aneurysms until their death or SAH. Twenty-nine percent of patients presented with SAH during lifelong follow-up. The annual rupture rate per patient was 1.6%. They found that female sex, current smoking, and aneurysm size of 7 mm or greater in diameter were risk factors for a lifetime SAH, and, depending on the risk factor burden, the lifetime risk of an aneurysmal SAH varied from 0% to 100%. The still intriguing finding was that even among the 96 patients with small (< 7 mm) unruptured aneurysms, 24 (25%) had an aneurysmal SAH during the follow-up.[2]

Those reports, as well as other studies, have identified factors clearly related to aneurysmal bleeding. However, such factors were still linked to demographic and epidemiological data and anatomical characteristics of the aneurysm. Nevertheless, the finding that even small aneurysms bleed together with the failure to prevent aneurysmal bleeding in low-risk patients has brought attention to microstructural and chemical environment involved in the development of aneurysms and their rupture.

The intraoperative finding of microbleeding adjacent to incidental aneurysms submitted to microsurgery was reported by Nussbaum et al.[4] It reinforces the underlying microstructural pathophysiology of aneurysmal rupture, which probably involves regional blood flow disturbances and the presence of an inflammatory process, allowing for bleb formation, enlargement of a cerebral aneurysm, and microbleeding before definitive bleeding.[4,5]

The marker of such a microscopic phenomenon is the surrounding tissue edema, revealing inflammatory status near the aneurysm. Such edema may probably be the origin of oligosymptomatic headaches in patients harboring such findings, mimicking flu-like symptoms and happening especially in middle-aged women.[4] Such edema probably has a temporal link with risk of rupture, including risk of immediate rupture.