Predictors of outcome for gunshot wounds

TO THE EDITOR: I have read with great interest the article by Gressot et al.1 (Gressot LV, Chamoun RB, Patel AJ, et al: Predictors of outcome in civilians with gunshot wounds to the head upon presentation. J Neurosurg 121:645–652, September 2014). The authors concluded that several factors, including patient age, Glasgow Coma Scale score, nonreactive pupils, and the path of the bullet and its fragments on CT scans, have predictive value for patient survival, and they created a scoring system based on these parameters. In their series of 119 patients 19% had good functional survival. We published an article in 1979 dealing with the same issues.2 In our study of 82 patients with gunshot wounds sustained in civilian life, we evaluated the same presentation parameters as the authors and we assessed functional outcome. We also evaluated surgical findings, specifically the incidence of hematomas either at surgery or at autopsy in all patients who did not survive. Although we evaluated the importance of the same criteria, we concluded that the only truly meaningful parameter predicting patient survival was the state of consciousness on admission. While all the other parameters had some value, they were of secondary importance in predicting survival and its quality. There were 4 groups of patients based on the state of consciousness at the time of presentation: I) alert, awake; II) obtundation with or without neurological deficit; III) unresponsive to all but noxious stimulation in appropriate or semi-appropriate fashion; and IV) comatose inappropriate or no response at all.

We have found that this system provides as useful information as the point system proposed by the authors, although it is substantially simpler. Adding additional information did not increase the accuracy or reliability of our basic scale. For instance, while a bihemispheric bullet path generally indicates a poor prognosis, if the examination results fall into Group I or II, it is the state of consciousness that determines the prognosis rather than the path of the bullet. Similarly, if a patient has fixed, dilated pupils, the prognosis is poor, but patients in this case will undoubtedly be comatose with, at best, a decerebrate posture, which would place them in Group IV in which there are no survivors. Again, it is the state of consciousness that determines outcome. This simplified neurological examination makes it easier to rapidly assess patients with gunshot wounds, particularly if non-neurosurgeons in the emergency department triage the patients. It provides rapid and accurate early information for patients and their families.

I was intrigued by the fact that in our study, 39% of patients achieved a functional survival status compared to 19% in the authors’ study. The extent of injury caused by a bullet is determined to the greatest degree at the time of impact and is dependent on bullet mass and exit muzzle velocity squared. Passage of the bullet through brain tissue creates waves of massive increases in intracranial pressure in the wake of the bullet. Based on the above formula, the damage is greater with a greater bullet mass and greater muzzle exit velocity such as that seen in military grade weapons. Thus, the degree of neurological deficit is determined at time of impact in most instances. The development of mass lesions, such as hematomas, is rare and was seen in only 10% of our cases. Admitting that neither the authors nor we have studied the ballistic profile of the guns used, it is tempting to think that the difference in functional survival between our studies most likely reflects the currently greater availability of better quality weapons with larger bullet sizes and greater muzzle exit velocities. These cause much more damage than the “Saturday night specials” commonly used at the time of our study and could explain this discrepancy.

The authors are to be congratulated for continuing to study the problem of gunshot injuries, which are becoming more and more serious, particularly with easier access to higher-grade weapons in the civilian population.

Otakar R. Hubschmann, MD
Saint Barnabas Medical Center,
Institute of Neurology and Neurosurgery, West Orange, NJ

DISCLOSURE
The author reports no conflict of interest.

References
Response

We recognize Dr. Hubschmann and colleagues for their pioneering work in this area. Their study was performed between 1973 and 1975, and they did not have the benefit of routine CT scanning in evaluating patients with gunshot wounds to the head. While they found that the state of consciousness upon arrival was the only meaningful predictive measure of outcome, we found that the Glasgow Coma Scale score upon arrival was less important than pupillary reactivity and bullet trajectory as seen on CT scan upon arrival. As imaging technology continues to improve, we expect our understanding and evaluation of these patients to evolve as well.

Loyola Gressot, MD
Shankar Gopinath, MD
Baylor College of Medicine, Houston, TX

The blood-hammer effect and aneurysmal basilar artery bifurcation angles

TO THE EDITOR: The interesting study by Tütüncü et al.3 (Tütüncü F, Schimansky S, Baharoglu MI, et al: Widening of the basilar bifurcation angle: association with presence of intracranial aneurysm, age, and female sex. J Neurosurg 121:1401–1410. December 2014) seems to support the idea that unfavorable vessel design in arterial bifurcations may be a risk factor in intracranial aneurysm formation and progression. The hypothesis that aneurysmal basilar artery (BA) bifurcations present increased angles compared with nonaneurysmal basilar bifurcations was scrutinized. The BA bifurcation (α) angle, estimated in age-matched cohorts, was found to be significantly wider in patients with a BA aneurysm (146.7° ± 20.5°) than in patients with an aneurysm in another cerebral artery (non-BA) (111.7° ± 18°) and in a nonaneurysmal control group (103° ± 20.6°). Also in the non-BA aneurysm group, the α was significantly wider than in the control group.

Aneurysm size, height, and neck presented a significant positive correlation with the α angle, which was also independently predicted by the size of the aneurysm neck in multivariate analysis. In nonaneurysmal controls, α grew wider with increasing age, with a steeper dependence in female than in male patients. In computational fluid dynamics (CFD) simulations, done on parametric BA models (with radii of mother and symmetrical daughter vessels following the vascular optimality principle) as well as on patient-derived models of the BA bifurcation, low wall shear stress (WSS) at the area of flow impingement at the bifurcation apex was spatially contiguous with an acceleration area along the daughter branches (where WSS increased abruptly to maximal values) that was followed by a deceleration area (where WSS gradually decreased). Both the flow impingement area and the acceleration area increased with increasing α angles on CFD simulations. The vertebrobasilar junction angles were also evaluated, but no significant geometrical changes were observed in any patient group.

It is appealing to speculate that the observed strong correlation between the BA α angle and aneurysm presence, and the finding that in patients with aneurysms at sites other than the BA the α angle was significantly wider than in the control group (the authors suggested that this denotes a global arterial weakening at bifurcation sites throughout the cerebral circulation in patients with aneurysms), indicates that an increased α angle can precede aneurysm formation. The authors cautiously and correctly dismissed such a causal relation, but this hypothesis surely is worth keeping in mind in future observations (prospective observations would be necessary to ascertain temporal priority). Moreover, the difference in α angles in the BA aneurysm group and in the non-BA aneurysm group (35° on average) was much larger than the difference between the non-BA cases and the controls (8.7°), and can reasonably result from aneurysm formation and progression. Apart from the intuitively understandable fact that a saccular aneurysm is a focus of vessel wall rupture and that a lumen herniation in a pouch of vessel wall remnants usually located just at the apex of the α angle would increase this angle even more, in terminal aneurysms every systolic blood pressure wave exposes the aneurysm sac to the “blood-hammer” effect.1,2 This phenomenon, known in hydraulic engineering as the “water hammer,” occurs when flow of fluid in a pipe is stopped by abrupt closure of a valve, reducing the kinetic energy of the upstream fluid to zero, briefly creating an overpressure at the valve that adds to the steady pressure, and causing a pressure wave to move upstream at the speed of sound that is followed by secondary (“bouncing”) waves until the fluid comes to rest. Ahlqvist1 provided a simple equation to estimate the overpressure (ΔP); ΔP = vρ[(ρ + d/Eh)]/2.

Assuming physiological or nearly analogous values for blood flow velocity (v), blood density (ρ), blood compressibility (K), vessel wall thickness (h), and elastic modulus (E) as provided by Ahlqvist1 in his hypothetical considerations about embolic occlusion of the middle cerebral artery, but adjusting the internal arterial diameter (d) to 3.5 mm for the BA and assuming the presence of a BA tip aneurysm whose neck is as wide as the BA, the overpressure provoked by the impact of the blood flow at the domus of the aneurysm, that is reflected upstream in the BA bifurcation, is approximately 55 mm Hg.

In other words, at the apex of the BA aneurysm sac a sudden arterial pressure increase of 55 mm Hg is added to the blood pressure at the parent artery at every heartbeat, and this pressure wave is transmitted upstream along the aneurysm wall to the aneurysm ostium and parent artery. The overpressure is added to and does not depend on the baseline arterial blood pressure. Compared to abrupt embolic occlusion of an artery, the primary wave probably spreads along a shorter distance (the posterior cerebral
arteries may function as a shunting system that damps the primary overpressure wave) and thus with less intense secondary waves, if any, but a new primary wave is produced after the next heartbeat.

This estimate, even if admittedly rough, indicates an amplification of the hemodynamic stresses in terminal BA aneurysms that could drive the BA tip upward, and increase the \( \alpha \) angle over time. The blood-hammer effect is intuitively similar to the impingement force, because both express the transformation of kinetic energy in pressure load at the aneurysm domus. The blood-hammer effect can also be related to the propensity of wide-necked terminal BA and middle cerebral artery aneurysms to recur after simple coiling procedures, and the necessity to complement the endovascular treatment of many of these aneurysms with stents.

Sandro Rossitti, MD, PhD
University Hospital, Linköping, Sweden

DISCLOSURE
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References

Response
No response was received from the authors of the original article.

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Minimally invasive technique for epilepsy surgery

TO THE EDITOR: We read with great interest the article by Quigg and Harden7 (Quigg M, Harden C: Minimally invasive techniques for epilepsy surgery: stereotactic radiosurgery and other technologies. J Neurosurg 121 (Suppl 2):232–240, December 2014). Surprisingly, the authors completely missed the recent progress of stereotactic radiofrequency amygdalectomy for mesial temporal lobe epilepsy (MTLE), a recently modified method that is the only one having seizure control results comparable with anterior temporal resection. Only less effective stereotactic treatments for MTLE published by Parrent and Blume6 in 1999 and by Guénot et al.1 are mentioned.

We would like to focus readers’ attention on our stereotactic radiofrequency method, which has been used since 2004. For radiofrequency amygdalectomy the occipital access is used and thermolesions are administered by an electrode with a 10-mm bold active tip; usually 24 lesions are made in 8 segments. Thermocoagulative lesions are made in the long axis of the amygdalectomy complex (AHC).2 After surgery, thermocoagulative necrosis affects nearly the whole AHC. One year after surgery an irregular pseudocyst developed in the AHC, which caused partial destruction of AHC, the entorhinal and perirhinal cortices included.3 As of this writing we have treated 63 patients with MTLE, and we recently published long-term clinical seizure outcomes in the group of 61 patients. The mean clinical seizure follow-up was 5.3 years, and 70.5% of our treated patients have been seizure free since surgery (Engel I).4 Neuropsychological outcomes were very good; patients did not decline in any memory parameters 1 and 2 years after therapy, despite the fact that two-thirds of patients were treated on the left side.4 We are not aware of any other stereotactic method that achieves similar clinical outcomes that are fully comparable with open surgery procedures.

Hana Malikova, MD, PhD
Roman Liscak, MD, PhD
Zdeněk Vojtěch, MD, PhD
Epilepsy Center Na Homolce Hospital, Prague, Czech Republic

DISCLOSURE
The authors report no conflict of interest.

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Malignant meningiomas

TO THE EDITOR: I read with great interest the paper by Sughrue et al.14 (Sughrue ME, Sanai N, Shangari G, et al: Outcome and survival following primary and repeat surgery for World Health Organization Grade III meningiomas. J Neurosurg 113:202–209, August 2010), and I would like to ask the authors some questions.

1. In the Abstract the authors wrote they studied all malignant meningioma cases operated on over a 16-year period while subsequently, in the Patient Demographics paragraph, it is stated that the 63 cases of malignant meningioma included in the study were taken from a total of 1228 meningiomas evaluated over a 22-year period. Does this mean that the authors didn’t observe malignant meningioma cases over a 6-year period?

2. The sex ratio of malignant meningioma patients studied by Sughrue et al. shows a female predominance. This comes as a bit of a surprise to me, since previous studies on malignant meningiomas point to the opposite (i.e., a definite male predominance). At the same time, it should be noted that an equal male-to-female ratio or even a slight female predominance has occasionally been found for patients with atypical meningiomas.

3. One third of the malignant meningioma patients who were surgically treated by the authors were asymptomatic. Presumably, these lesions were incidentally discovered. How many incidental/asymptomatic meningiomas did the authors observe during the study period, and what was the global incidence of malignant forms among them? How long was the mean observation period before the discovery of malignant progression? In the literature on incidental/ asymptomomatic meningiomas, the incidence of malignant meningiomas among the small number of patients who needed surgery was of 1.5%, and that of atypical meningiomas was of 4.5%—similar to what is commonly observed in primary symptomatic meningiomas.15 Since 29 of 63 patients had only the second surgery at the University of California, San Francisco (UCSF), it can be assumed that the incidence of asymptomatic/incidental cases among patients with malignant meningiomas having initial surgery at UCSF was surprisingly high, at 62% (21/34). Is this so? In my opinion, a possible partial explanation could be the overgrading of cases of atypical meningioma submitted to preoperative embolization. How many patients had undergone preoperative embolization in Sughrue and colleagues’ case series of malignant meningiomas? In the literature there has been some concern about the possibility of overgrading of preoperatively embolized benign meningiomas.3,8,10,11 Can the authors exclude the possibility of having classified as malignant cases of atypical meningioma that were embolized before resection? Given such doubt, did the authors confirm the malignancy of their cases by means of a cytogenetic analysis?

4. Perhaps the most surprising result of this study is that patients who had subtotal resection (STR) fared better than those who underwent gross-total resection (GTR), a finding not explained by the authors. Here again, I think preoperative embolization might matter. Such a procedure not only could have led to histological overgrading of an otherwise atypical meningioma as proposed above but, as hypothesized by the authors themselves in their paper on benign meningiomas, also could have induced better control of residual tumor after STR through its involution following devascularization and partial infarction. Sughrue et al. wrote: “Preoperative embolization was performed for larger tumors...” What actually was the respective incidence of such procedures in the GTR and STR groups? Another potential confounding variable leading to the suggested benefit of STR over GTR in the series studied by Sughrue et al. lies in the concept of grouping together Simpson Grade I, II, and III resections. It cannot be denied that a true total resection of a meningioma, classically defined as Simpson Grade I, can be obtained with confidence in convexity tumors only. Accordingly, a paper from the same San Francisco group showed recurrence rates of 0% and 17% after Simpson Grade 0–I and Grade II resection, respectively, in their series of benign convexity meningiomas. In our series of 29 malignant meningiomas, we found that Simpson Grade I resection was successful only in convexity tumors. Simpson Grade II or III actually means incomplete resection, even if less incomplete than Simpson Grade IV. In a case of malignant meningioma it is hard for me to imagine what a difference it could make to have many cells inside the dura mater (Simpson Grade II–III) or a small cluster (Simpson IV) left behind. In addition it should be considered that the biology of meningiomas, whether benign or malignant, is so far largely unknown and highly variable. So it cannot reasonably be excluded that patients with malignant meningioma undergoing Simpson Grade II–III resection and included in the GTR group had biologically more malignant lesions and then had a more downhill clinical course than patients included in STR group.

In conclusion, before reversing the intuitive benefit of radical resection on survival in their series of malignant meningiomas, I suggest that Sughrue et al. should separate the cases of convexity tumors with Simpson Grade I resection to be compared to all the others. Notwithstanding all the above observations, I think this is an excellent paper and I have enjoyed reading it very much.

Lucio Palma, MD
Clinica Neurochirurgica dell’Università di Siena, Siena, Italy


Response

No response was received from the authors of the original article.

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Endoscopic transnasal transcribriform approach

TO THE EDITOR: The article by Banu et al. is an important contribution to the analysis of predictive factors of postoperative CSF leaks after endoscopic skull base surgery (Banu MA, Szentirmai O, Mascarenhas L, et al: Pneumocephalus patterns following endonasal endoscopic skull base surgery as predictors of postoperative CSF leaks. J Neurosurg 121:961–975, October 2014). However, we think that the term “transcribriform” has been misused in this article.

Although the endoscopic approach to the anterior skull base had been already described in several case series,4,5,11,13,17,21,28,29 it was definitely systematized in 2004 by Jho and Ha,15 in 2005 by Har-El and Casiano,14 and, in the same year, by Kassam et al.18 These authors introduced in their article the term “transcribriform” to refer to the endoscopic approach to the anterior skull base. In the following years, the indication of the endoscopic route for the anterior skull base lesions had been progressively expanded, and it became common for authors to start using the term “transcribriform” to refer to the removal of the cribiform plate and foramen ethmoidalis, thus describing an approach that implied the demolition of the anterior part of the ethmoidal roof. Using the term “transcribriform” in these cases, in our opinion, is not correct because it describes only the removal of the cribiform plate and therefore, it is not sufficient, in several cases, for creating a surgical corridor that is useful to approach the anterior skull base lesions, with the exception of CSF leaks.

An accurate description of the anatomy of the cribiform plate and anterior skull base has been given in the literature,19–26 in particular, the analysis of dimensions and asymmetries, proposing various classifications on the basis of form and height of the lateral lamina.20 According to the article by Vasvari et al.,26 the dimensions of the cribiform plate are on average 20.7 mm in length (range 14.1–28.4 mm), 3.1 mm in width at the tip of the crista galli and the olfactory sulcus until exposure of the dura mater. According to Kassam et al.,18 and consists of the following surgical steps: resection in modern neurosurgical treatment of World Health Organization Grade I meningiomas.

Since then, in our opinion, the term “transcribriform” has been commonly used incorrectly, indicating indeed the approach to the anterior part of the skull base and usually being associated with “transplanum” to indicate the approach to the entire anterior skull base.24,28,29...
We think that using the term “transcribriform” to indicate the endoscopic approach to the anterior skull base is quite synecdochic and misleading. In our opinion, a descriptive name has to correctly represent the approach, reflecting the real surgical procedure and not only a part of it. When a skull base team deals with tumors of the anterior skull base, the demolition steps involve of course not only the cribiform plate but possibly reach laterally to the superomedial margins of the orbit.

Is the “transcribriform-transplanum approach” really representative of the anterior skull base approaches? Anatomically speaking, does the so-called transcribriform approach only involve the cribiform plate, or is the resection extended to the fovea ethmoidalis of the frontal bone?

The anatomical measures of the cribiform plate are sufficient to clarify that the approach can be purely transcribriform, and sometimes unilateral, only in select cases (CSF leak repair) and will have to be extended bilaterally to the ethmoidal fovea in cases of intracranial lesions.

The uniformity of nomenclature is an important feature to improve the communication among different groups, and we support the idea that representative and descriptive definitions could be a strong starting point to share and improve our knowledge.

We think that the term “transcribriform transfovea ethmoidalis” (used only once in the literature) is to be preferred to indicate the endoscopic approach to the anterior skull base in cases of dural exposure from orbit to orbit.

On the basis of our analysis, we think that the endoscopic endonasal approaches to the anterior skull base could be classified as follows:

1. **Transethmoidal**: when a partial or complete ethmoidectomy and only extracranial procedure has been performed;
2. **Transcribriform**: when only the cribiform plate is resected and some intracranial procedure has been performed;
3. **Transfovea**: when only the fovea is resected and some intracranial procedure has been performed;
4. **Transcribriform-transfovea**: when the anterior part of the ethmoidal roof is resected from orbit to orbit;
5. **Transcribriform-transfovea-transplanum**: when the entire ethmoidal roof is resected to access the anterior cranial base.

Sometimes, the most common scientific terms are semantically incorrect and it is mandatory to correct any evident error in order to create a common and shared lexicon that is accepted by the entire scientific community. The term “transcribriform” has been commonly used incorrectly when it indicates the standard endoscopic approach to the anterior skull base with the complete demolition of the ethmoidal roof from orbit to orbit. Actually, the anatomically appropriate term to indicate this approach is “transcribriform transfovea,” whereas the term “transcribriform” should be used when the bone demolition involves the cribiform plate.

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Cesare Zoia, MD
Paolo Gaetani, MD
IRCCS Fondazione PoliClinico San Matteo, Pavia, Italy

Iacopo Dallan, MD
Azienda Ospedaliero-Universitaria Pisana, Pisa, Italy
Davide Lepera, MD
Paolo Battaglia, MD
Paolo Castelnuovo, MD, FRCSed
University of Insubria, University of Pavia, Pavia, Italy
Antonio Fratto, MD
University of Pavia, Pavia, Italy

DISCLOSURE
The authors report no conflict of interest.
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 cribiform 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 “trans-ethmoidal-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.

Theodore H. Schwartz, MD
Weill Cornell Medical College, New York, NY

Reference


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.5 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.5

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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.3 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

Archie Defillo, MD
Jerone Kennedy, MD
St. Cloud Hospital, St. Cloud, MN

DISCLOSURE
The authors report no conflict of interest.

References

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

These 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 ologosymptomatic 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.
Magnetic resonance imaging utilizing T2 gradient recalled echo or MRI-SWI techniques to evaluate perianeurysmal edema and perianeurysmal hemosiderin deposits become essential tools in the routine assessment of unruptured aneurysms. Magnetic resonance spectroscopy with evaluation of chemical environment surrounding aneurysms may also be a potential target for future discussions.

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