CLINICAL PRACTICE

Unruptured Intracranial Aneurysms

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This Journal *feature begins with a case vignette highlighting a common clinical problem. Evidence supporting various strategies is then presented, followed by a review of formal guidelines, when they exist. The article ends with the author's clinical recommendations.*

A 55-year-old woman with well-controlled hypertension and a 20-pack-year smoking history presents to her primary care physician with occasional throbbing headaches on the left side and a recent episode of blurred vision. Her mother died of a subarachnoid hemorrhage at 62 years of age, and her uncle was treated for an unruptured brain aneurysm. On examination, her blood pressure is 135/85 mm Hg, with no focal neurologic deficits. Magnetic resonance angiography (MRA), performed as part of her headache evaluation, reveals a 6-mm saccular aneurysm in the posterior communicating artery, arising from the proximal aspect of the artery; the finding was confirmed by digital subtraction angiography (Fig. 1). The patient is currently asymptomatic and concerned about the risk of aneurysm rupture and the risks associated with potential interventions. How should her care be managed?

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CME

THE CLINICAL PROBLEM

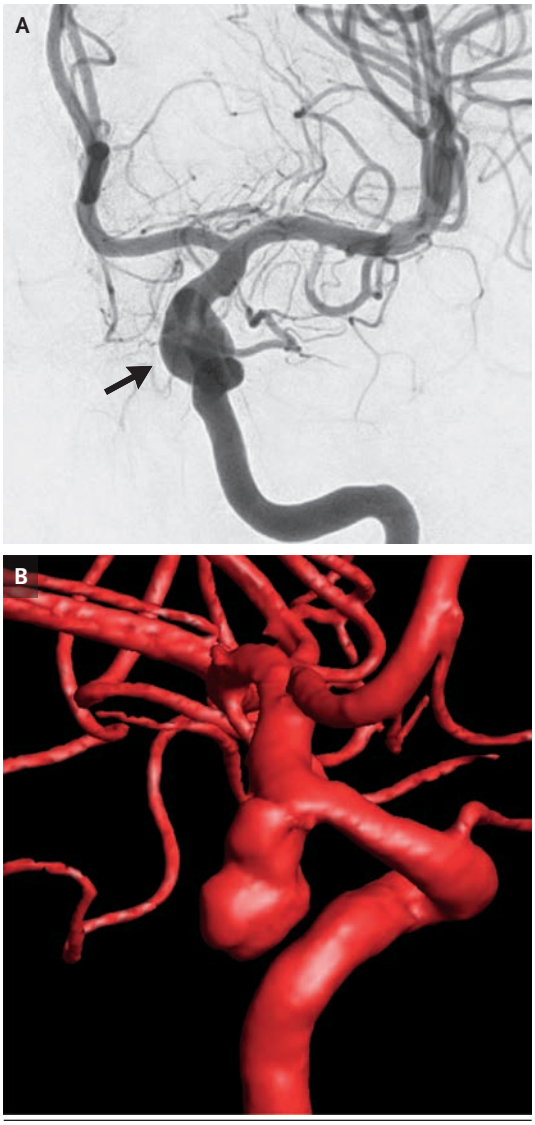
Intracranial aneurysms (abnormal dilatations of the arterial wall of intracranial blood vessels[1]) are the most common cause of nontraumatic subarachnoid hemorrhage.[2] Subarachnoid hemorrhage is associated with high mortality, which makes early detection and measures to reduce the risk of unruptured intracranial aneurysm (UIA) formation a rational strategy.[3] In a systematic review of 68 prevalence studies, covering 83 study populations and 94,912 patients from 21 countries, the overall prevalence of UIA was estimated at 3.2% among persons without coexisting medical conditions with an average age of 50 years. A higher prevalence was observed among women, persons 30 years of age or older (peaking in the sixth decade of life), and those with a family history of UIA, subarachnoid hemorrhage, hypertension, or autosomal dominant polycystic kidney disease.[4]

Saccular aneurysms account for approximately 90% of lesions and typically form at arterial bifurcations in the internal carotid artery, the anterior and posterior communicating arteries, and the middle cerebral artery. In the posterior circulation, they commonly occur at the basilar artery bifurcation and cerebellar artery branch points (Fig. 2).[1] Less common types of UIAs include fusiform aneurysms involving elongated segments of the artery, mycotic aneurysms associated with infections, and dissecting aneurysms resulting from arterial injury.[5] Up to 20% of patients with UIAs have multiple intracranial aneurysms.[6] The formation of aneurysms is thought to result from degeneration of the internal elastic lamina, endothelial dysfunction, and hemodynamic stress; inflammation, which leads to instability of the vascular wall, plays a crucial role in both their formation and rupture.[7]

KEY POINTS

UNRUPTURED INTRACRANIAL ANEURYSMS

- The risk of intracranial aneurysm rupture is influenced by aneurysm size, location, morphologic features, and patient-specific factors such as hypertension, smoking, and family history.
- Magnetic resonance angiography and computed tomographic angiography are preferred for the detection of intracranial aneurysms, with digital subtraction angiography reserved for complex cases or treatment planning.
- Conservative management focuses on smoking cessation, blood-pressure control, and routine imaging follow-up, whereas intervention (endovascular or surgical) is reserved for higher-risk aneurysms (i.e., ≥7 mm in diameter and located in the anterior circulation).
- Endovascular techniques, including coiling and flow diversion, are associated with lower perioperative risks but higher risks of recurrence, whereas surgical clipping provides durable results but with greater procedural risks.
- Living with an intracranial aneurysm can cause considerable anxiety. Patient education, psychological support, and shared decision making are essential in addressing patient concerns and ensuring adherence to the management plan.



RISK FACTORS

Risk factors for UIA formation are classified as modifiable and nonmodifiable. Modifiable risk factors include cigarette smoking and hypertension. A case–control study involving 206 patients with UIAs without previous subarachnoid hemorrhage and 574 controls identified smoking and hypertension as independent and additive risk factors for UIA formation.[8] The study also showed that hypercholesterolemia, possibly due to concomitant statin use, and regular physical exercise are associated with a decreased risk of UIA formation. A multicenter case–control study involving 113 matched pairs confirmed the synergistic effect of risk factors, showing that the risk of UIA formation was four times as high among female smokers and seven times as high among hypertensive female smokers as among nonsmoking normotensive women.[9] Heavy alcohol use, a risk factor for subarachnoid hemorrhage, may also be a risk factor for UIA formation.[10]

Nonmodifiable risk factors for UIA formation include female sex, increasing age, and genetic predisposition (family history of aneurysm). The risk of UIA formation is higher among women, especially those older than 50 years of age, than

Figure 1. Imaging in a Patient with a Posterior Communicating Aneurysm.

In Panel A, digital subtraction angiography shows a 6-mm posterior communicating aneurysm (arrow) in a woman 55 years of age. A three-dimensional reconstruction of the aneurysm is shown in Panel B.

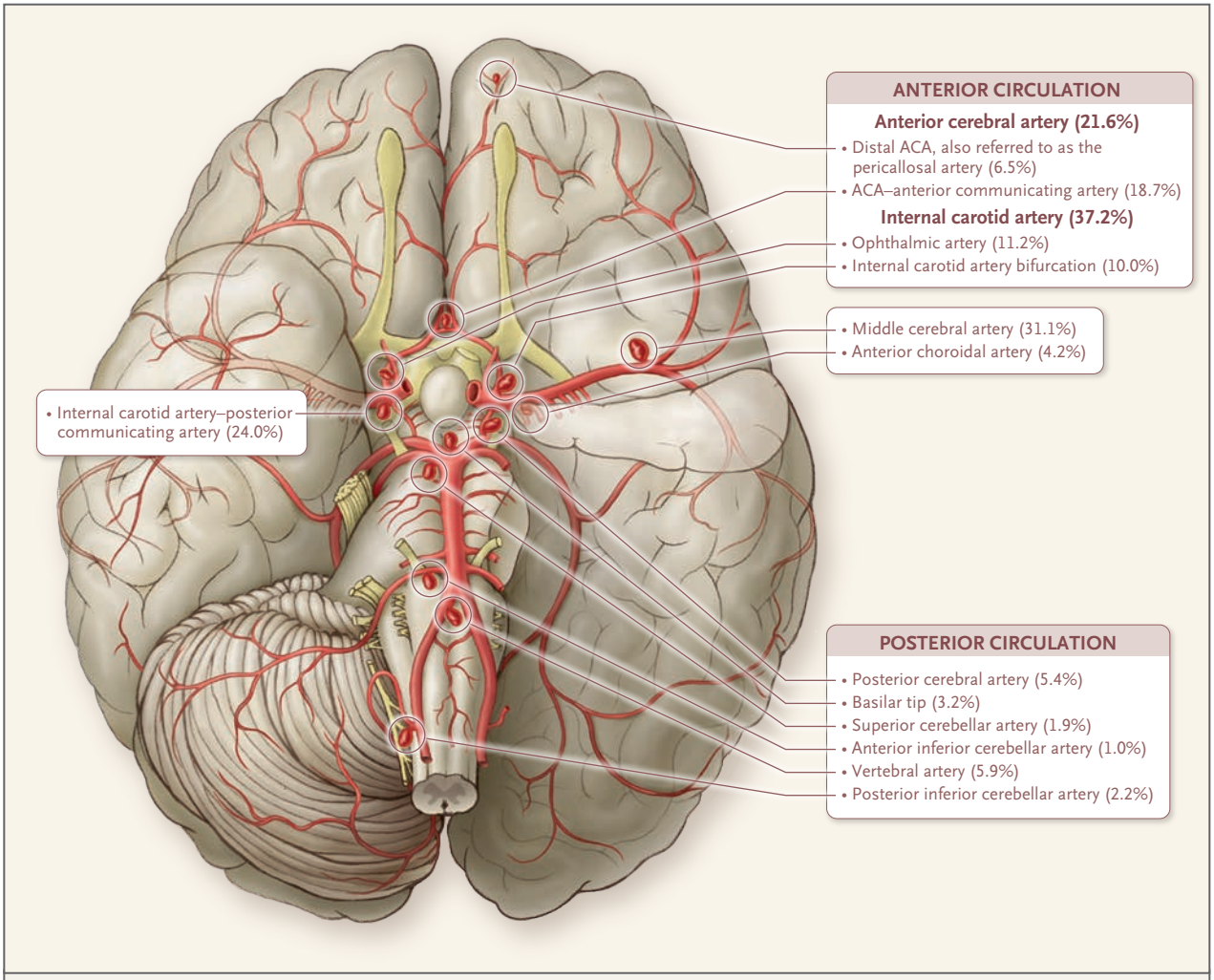


Figure 2. Distribution of Unruptured Intracranial Aneurysms According to Arterial Location.

Data were synthesized from the most recent global prevalence systematic review by Vlak et al. (see the Supplementary Appendix).[4] ACA denotes anterior cerebral artery.

among men, with a female-to-male risk ratio of 2:1 — a finding that may indicate the role of hormones as a mediating factor.[4] Genetic factors have been associated with UIA formation. A large systematic review and meta-analysis involving 116,570 patients identified single-nucleotide polymorphisms in the *CDK2NB*, *EDNRA*, and *SOX17* genes as major contributors to UIA formation.[11] These genes are linked to cell-cycle regulation and vascular smooth-muscle proliferation (CDK2NB), vascular endothelial maintenance (SOX17), and vascular smooth-muscle response to hemodynamic stress through endothelin-1 production (EDNRA). Heritable conditions such as autosomal dominant polycystic kidney disease, coarctation of the aorta, the Ehlers–Danlos syndrome, glucocorticoid-remediable aldosteronism (familial aldosteronism type I), moyamoya disease, and thoracic aortic aneurysms are also associated with a higher risk of UIA formation.[12] A family history of UIA or subarachnoid hemorrhage increases the risk of UIA formation by a factor of 3.6[4] and raises the risk of UIA rupture by a factor of 2.5 (up to a factor of 17 among smokers with hypertension).[13,14] Persons with a family history of UIA or subarachnoid hemorrhage are more likely to have a subarachnoid hemorrhage at smaller aneurysm sizes and younger ages and with worse outcomes than persons with sporadic UIAs.[15,16]

NATURAL HISTORY OF UIA

The risk of UIA rupture is important for informing treatment decisions and risk management; however, the precise natural history is not well understood, and our ability to predict prognosis is uncertain, with limited data available from longitudinal studies. Risk factors for aneurysm growth and rupture include female sex, hypertension, smoking, large aneurysm size (≥7 mm), irregular aneurysm shape or a "daughter sac" (a small, irregular outpouching less than half the size of the parent aneurysm), the presence of multiple aneurysms, location in the internal carotid or basilar artery, and concurrent arteriovenous malformations.[17,18]

STRATEGIES AND EVIDENCE

CLINICAL PRESENTATION

UIAs are usually asymptomatic and often diagnosed incidentally through imaging.[19] Symptomatic UIAs can cause cranial nerve palsies, seizures, facial pain, hemiparesis, ischemia, and visual disturbances; symptoms may arise from compression or thromboembolism and vary according to location.[20] Headaches are common in patients with UIAs but are often not directly related to the aneurysm. Although the headaches associated with UIAs are typically nonspecific, they may manifest as sudden, severe, or persistent pain and occasionally mimic migrainelike throbbing or unilateral pain. It is crucial to distinguish a UIA-associated headache from a "thunderclap" headache, which is characterized by abrupt, intense pain (often described as "the worst headache of my life") that warrants immediate imaging to rule out aneurysmal subarachnoid hemorrhage. New-onset headaches may indicate a UIA becoming symptomatic or a UIA linked to conditions such as reversible cerebral vasoconstriction syndrome, cervical artery dissection, cerebral venous thrombosis, intracranial hypotension, and intracranial infection.[21] The history taking should also explore risk factors for UIA formation, including smoking, hypertension, family history of UIA or subarachnoid hemorrhage, and heritable syndromes. Unruptured posterior communicating artery–internal carotid artery aneurysms that point downward can manifest with a third nerve palsy, and fusiform basilar aneurysms can be very symptomatic and rarely cause optic chiasm compression.

DIAGNOSTIC IMAGING

UIAs are typically diagnosed by means of computed tomographic angiography (CTA) or MRA, either incidentally during routine screening of high-risk patients or when screening patients with symptoms (e.g., chronic headache, dizziness, cranial nerve palsies, visual-field defects, hemiparesis, ptosis, mydriasis, diplopia, facial pain, or ischemic symptoms due to thromboembolism from the aneurysm). CTA and MRA are preferred for routine screening of high-risk patients because of their high sensitivity and noninvasive nature.[22] Digital subtraction angiography remains the reference standard for UIA imaging; it provides superior detail of aneurysmal features, detects very small aneurysms, and facilitates pretreatment planning. Digital subtraction angiography is also used when there is high clinical suspicion (e.g., suggestive symptoms and strong family history) of UIA despite normal CTA and MRA findings.[23] Patients undergoing CTA, MRA, and digital subtraction angiography should be evaluated for contraindications (e.g., risk of contrast nephropathy) and counseled about potential adverse effects (radiation exposure, nephrotoxic effects, and, in rare cases with digital subtraction angiography, thromboembolic events, infection, and injury to vessels).

RISK ASSESSMENT AND TREATMENT DECISION MAKING

Management of UIAs involves a multidisciplinary approach and shared decision making in which both patient- and aneurysm-related risk factors for aneurysm rupture are considered. These assessments can be informed by treatment scoring systems. The PHASES score (population, hypertension, age, size of aneurysm, earlier subarachnoid hemorrhage, and site of aneurysm) was developed with data from a meta-analysis of six prospective cohort studies and may be used in practice to assess the risk of aneurysm rupture, once identified.[24] This analysis incorporated data from 10,272 cases of UIAs in 8382 patients, who were followed for a median of 1 to 21 years, and the results led to the development of this clinical prediction score for the estimation of the 5-year risk of aneurysm rupture (Table 1). Although key factors such as ethnic group were adjusted for in order to enhance the generalizability of the model, as compared with the models used in earlier studies, the PHASES score is limited by

the omission of known risk factors (e.g., smoking, familial history, and aneurysm shape) and by the fact that it is only a post hoc derivation score that has not been further validated in a prospective study.[27,28] The 1-year risk of UIA rupture that was estimated with the use of the PHASES score was 1.4% (95% confidence interval [CI], 1.1 to 1.6), and the 5-year risk was estimated to be 3.4% (95% CI, 2.9 to 4.0), findings that contrast with those from earlier studies (Fig. 3).

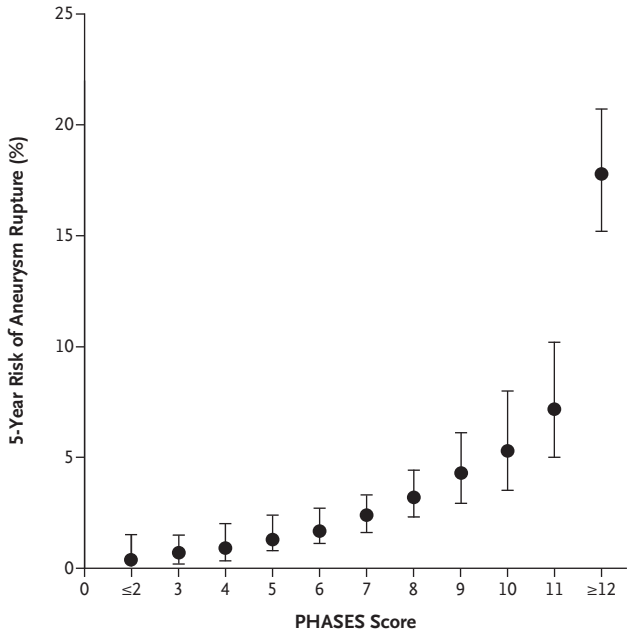
In the International Study of Unruptured Intracranial Aneurysms, the annual risk of rupture was reported to be as low as 0.05% for small anterior circulation aneurysms (<10 mm) in patients without previous subarachnoid hemorrhage (Table 1).[26] In contrast, the annual risk of rupture was up to 1% for aneurysms 10 mm or larger and as high as 6% for aneurysms 25 mm or larger. Patients with previous subarachnoid hemorrhage had higher annual risks of rupture (approximately 0.5%), even for small aneurysms. The size-dependent increase in the risk of rupture was further supported by the Japanese Unruptured Cerebral Aneurysm Study (UCAS Japan) and the Small Unruptured Intracranial Aneurysm Verification study, which showed annual risks of rupture of 0.54% for aneurysms smaller than 7 mm, which increased to 1 to 2% for aneurysms 7 mm or larger, particularly for those located in the posterior circulation (Table 1).[25,29]

The Unruptured Intracranial Aneurysm Treatment Score (UIATS) was developed by a multidisciplinary team of 69 experts through a Delphi consensus and incorporates treatment-related risk factors to personalize aneurysm management.[30,31] Two other scoring systems for the prediction of the risk of UIA rupture were developed with data from single-population cohorts (Table 2). In one of these systems, only four variables (age, cigarette smoking, aneurysm diameter, and aneurysm location) were used to create a treatment scoring system for UIAs, which showed better prediction performance than the UIATS (area under the curve, 0.76 vs. 0.62; P=0.02) in a Finnish population of 142 patients with UIA, among whom aneurysm rupture occurred in 34 over a follow-up period of 21 years.[33] A similar simple prediction model, developed with data from a Japanese cohort in the UCAS Japan, included 6608 cases of UIAs in 5651 patients.[25,34] Over a follow-up period totaling

Table 1. Incidence of Aneurysm Rupture According to the UCAS (1-Year Incidence) and the ISUIA (Overall Incidence).*

Location	1-Yr Incidence of Rupture According to Size of Aneurysm					Overall Incidence of Rupture According to Size of Aneurysm: No Previous SAH		Overall Incidence of Rupture According to Size of Aneurysm: Previous SAH	
	3–4 mm	5–6 mm	7–9 mm	10–24 mm	≥25 mm†	<10 mm	≥10 mm	<10 mm	≥10 mm
	percent								
UCAS Japan[25]									
ACA	0.9	0.8	2.0	5.2	39.8	—	—	—	—
Basilar tip or SCA	0.2	0.5	1.0	6.9	117.8	—	—	—	—
ICA	0.1	0	1.2	1.1	10.6	—	—	—	—
ICA–PComA	0.4	1.0	3.2	6.1	127.0	—	—	—	—
MCA	0.2	0.3	1.6	4.1	16.9	—	—	—	—
VA–proximal BA	0	0	0	3.5	0	—	—	—	—
ISUIA[26]	—	—	—	—	—	0.05	1.0	0.5	1.0

* ACA denotes anterior cerebral artery, BA basilar artery, ICA internal carotid artery (not cavernous carotid artery), ISUIA International Study of Unruptured Intracranial Aneurysms, MCA middle cerebral artery, PComA posterior communicating artery, SAH subarachnoid hemorrhage, SCA superior cerebellar artery, and VA vertebral artery.
† Incidences of rupture exceeding 100% in the Unruptured Cerebral Aneurysm Study (UCAS) most likely reflect statistical anomalies due to extremely small sample sizes and model-based estimates in these subgroups.



PHASES Score	Hazard Ratio for Rupture over 5 Yr (95% CI)
≤2	0.4 (0.1–1.5)
3	0.7 (0.2–1.5)
4	0.9 (0.3–2.0)
5	1.3 (0.8–2.4)
6	1.7 (1.1–2.7)
7	2.4 (1.6–3.3)
8	3.2 (2.3–4.4)
9	4.3 (2.9–6.1)
10	5.3 (3.5–8.0)
11	7.2 (5.0–10.2)
≥12	17.8 (15.2–20.7)

Figure 3. Five-Year Risk of Intracranial Aneurysm Rupture According to PHASES Score.
The PHASES score is based on population, hypertension, age, size of aneurysm, earlier subarachnoid hemorrhage, and site of aneurysm.[18] Scores range from 0 to 22, with higher scores indicating a higher risk of rupture.

11,482 aneurysm-years (the total number of years in which patients lived with an aneurysm), 107 ruptures were observed. The model, with the use of predictors such as patient age, sex, hypertension, aneurysm size, location, and presence of a daughter sac (Table 2), showed good discrimination and calibration after external validation. Age has a considerable influence on UIA treatment decisions, because interventions in older patients pose higher risks and must be weighed against life expectancy and coexisting medical conditions.[35]

In addition to aneurysm rupture, aneurysm growth can be monitored with the use of the externally validated ELAPSS score (earlier subarachnoid hemorrhage, location of aneurysm, age, population, and size and shape of aneurysm), which was developed with pooled data from 10 cohorts to assess a patient's risk of UIA growth within the next 3 or 5 years.[32] By incorporating these six factors as predictors of aneurysm growth, the ELAPSS score estimates the likelihood of aneurysm growth over time and may help guide follow-up imaging intervals, particularly by identifying patients with higher risk of growth who may benefit with more frequent monitoring.

Conservative Treatment

Generally, asymptomatic patients with small (<7 mm), incidental UIAs should be treated conservatively because of their low risk of rupture, with a focus on modifiable factors known to be associated with aneurysm growth. Key conservative measures include smoking cessation and blood-pressure control, supported by patient education and shared decision making.[36] Patients should also be informed about the risks of subarachnoid hemorrhage, warning signs (e.g., thunderclap headache and cranial nerve palsies), when to seek immediate medical attention, and the importance of long-term follow-up. Uncontrolled hypertension was associated with a higher risk of growth, which was 6.1 times as high as that among patients without hypertension and 3.9 times as high as that among those with controlled hypertension.

Routine noninvasive CTA and MRA monitoring (every 6 months until stable and then annually

Table 2. Scoring Systems That Form the Basis of the Clinical Rationale for Monitoring Aneurysm Growth and Rupture.*

Aneurysm Growth	Aneurysm Rupture		
ELAPSS Scoring System[32]	PHASES Scoring System[30]	Finnish Scoring System[33]	UCAS Scoring System[34]
Earlier SAH	Population	Age at diagnosis	Age
Yes — 0	North American, European (not Finnish) — 0	<40 yr — 2	<70 yr — 0
No — 1	Japanese — 3	Smoking status at baseline	≥70 — 1
Location of aneurysm	Finnish — 5	Current smoker — 2	Sex
ICA, ACA, or AComA — 0	Hypertension	Maximum diameter of UIA	Male — 0
MCA — 3	No — 0	≥7 mm — 3	Female — 1
PComA or posterior circulation — 5	Yes — 1	Aneurysm location	Hypertension
Age	Age	ACA — 5	No — 0
≤60 yr — 0	<60 yr — 0	ICA — 4	Yes — 1
>60 yr — 1 (per 5-yr interval after 60 yr)	≥70 yr (per 5 yr) — 1	PComA — 2	Location
Population	Size		ICA — 0
North America, China, Europe (other than Finland) — 0	<7.0 mm — 0		ACA or VA — 1
Japan — 1	7.0 to 9.9 mm — 3		MCA or BA — 2
Finland — 7	10.0 to 19.9 mm — 6		AComA or ICA–PComA — 3
Size of aneurysm, mm	≥20.0 mm — 10		Daughter sac
1.0 to 2.9 — 0	Earlier SAH from another aneurysm		No — 0
3.0 to 4.9 — 4	No — 0		Yes — 1
5.0 to 6.9 — 10	Yes — 1		
7.0 to 9.9 — 13	Site of aneurysm		
≥10.0 — 22	ICA — 0		
Shape of the aneurysm	MCA — 2		
Regular — 0	ACA, PComA, or posterior circulation — 4		
Irregular — 4			
Score and Growth Risk	**Score and Rupture Risk**		
Score <5	Score ≤2	Score of 0	Score of 0
3-yr risk: 5.0%	5-yr risk: 0.4%	Annual risk: 0%	3-yr risk: 0.2%
5-yr risk: 8.4%	Score of 3	10-yr risk: 0%	Score of 1
Score of 5–9	5-yr risk: 0.7%	30-yr risk: 0%	3-yr risk: 0.4%
3-yr risk: 7.8%	Score of 4	Score of 1–4	Score of 2
5-yr risk: 13.0%	5-yr risk: 0.9%	Annual risk: 0.6%	3-yr risk: 0.6%
Score of 10–14	Score of 5	10-yr risk: 3%	Score of 3
3-yr risk: 11.7%	5-yr risk: 1.3%	30-yr risk: 18%	3-yr risk: 0.9%
5-yr risk: 19.3%	Score of 6	Score of 5–8	Score of 4
Score of 15–19	5-yr risk: 1.7%	Annual risk: 2.2%	3-yr risk: 1.4%
3-yr risk: 17.5%	Score of 7	10-yr risk: 16%	Score of 5
5-yr risk: 28.1%	5-yr risk: 2.4%	30-yr risk: 49%	3-yr risk: 2.3%
Score of 20–24	Score of 8	Score of 9–12	Score of 6
3-yr risk: 25.8%	5-yr risk: 3.2%	Annual risk: 6.8%	3-yr risk: 3.7%
5-yr risk: 39.9%	Score of 9	10-yr risk: 60%	Score of 7
Score ≥25	5-yr risk: 4.3%	30-yr risk: 80%	3-yr risk: 5.8%
3-yr risk: 42.7%	Score of 10		Score of 8
5-yr risk: 60.8%	5-yr risk: 5.3%		3-yr risk: 7.6%
	Score of 11		Score of 9
	5-yr risk: 7.2%		3-yr risk: 17%
	Score ≥12		
	5-yr risk: 17.8%		

* Scores in the ELAPSS (earlier subarachnoid hemorrhage, location of aneurysm, age, population, and size and shape of aneurysm) system range from 0 to 40, with higher scores indicating a greater risk of growth. Scores in the PHASES (population, hypertension, age, size of aneurysm, earlier subarachnoid hemorrhage, and site of aneurysm) system range from 0 to 22, with higher scores indicating a greater risk of rupture. Scores in the Finnish system range from 0 to 12, with higher scores indicating a greater risk of rupture. Scores in the UCAS system range from 0 to 15, with higher scores indicating a greater risk of rupture. AComA denotes anterior communicating artery, and UIA unruptured intracranial aneurysm.

thereafter) in conservative UIA treatment is used primarily to track interval growth, which carries a risk of aneurysm rupture. Although growth rates and intervention thresholds vary, aneurysms showing growth of at least 0.5 mm over 36 months on follow-up imaging may prompt closer monitoring. Also, size thresholds for intervention have decreased over time, probably driven partly by the advent of safer and less invasive treatments; treatment is being increasingly considered for aneurysms smaller than 7 mm in diameter on the basis of additional risk factors such as age, location, and coexisting medical conditions.[37]

Endovascular Treatment

Endovascular techniques for UIA treatment include coil embolization, which involves introducing platinum coils into the aneurysm sac; scaffolding techniques to prevent coil prolapse by inflating a balloon in the parent artery (balloon-assisted coiling) or deploying a stent across the aneurysm neck (stent-assisted coiling); flow diversion, which involves the placement of flow-diverter devices into the parent artery to divert blood flow away from the aneurysm sac; and flow disruption, which involves placement of an intrasaccular device within the aneurysm sac to disrupt blood flow from the aneurysm. The choice of endovascular technique is made on the basis of the morphologic features and location of the aneurysm and operator expertise.

In the Collaborative Unruptured Endovascular versus Surgery trial, 291 patients with UIAs (97% of which were in the anterior circulation; mean aneurysm size, 7.8 mm) were randomly assigned to receive treatment with surgical clipping or through endovascular approaches.[38] At the 1-year follow-up, surgical clipping resulted in a lower incidence of treatment failure (9% vs. 19%; relative risk, 2.07; P=0.02), defined as failure of aneurysm occlusion, intracranial hemorrhage during follow-up, or residual aneurysms, as adjudicated by a core laboratory. However, perioperative safety outcomes favored endovascular treatment, which led to fewer neurologic deficits (12% vs. 22%, P=0.04) and shorter hospital stays than surgical clipping.

Surgical Treatment

Surgical clipping is the traditional treatment for UIAs and involves placing microsurgical clips across the neck of the aneurysm through a craniotomy and microsurgery. Clipping is more effective than endovascular treatment for aneurysm occlusion but is associated with longer hospitalization and a higher incidence of neurologic deficits.[38,39] Surgical clipping is typically preferred for younger patients with smaller aneurysms (<10 mm) in the anterior circulation.[40] For complex intracranial aneurysms, particularly those arising from small, distal vessels or in cases in which endovascular techniques are not feasible or have failed, cerebrovascular bypass surgery remains an effective treatment option.[41]

AREAS OF UNCERTAINTY

The management of UIAs is controversial owing to the lack of high-quality clinical data that directly compare the outcomes of conservative and interventional approaches. Regular imaging follow-up is advised, although the appropriate frequency and duration are unclear. Patients with UIAs often have considerable psychological effects; in a recent meta-analysis, the prevalences of anxiety and depression among patients with UIAs, regardless of management strategy (surveillance or treatment), were estimated to be 28% and 21%, respectively. However, data on psychological outcomes are limited and warrant further study to establish standardized methods and identify predictors of these conditions and their response to UIA treatment.[42] In addition, the discovery of UIAs in patients with other cerebrovascular disease presents a unique challenge because there is no consensus on whether to repair these aneurysms on account of the risk of rupture and future ischemic events.[43,44] Large-scale, prospective studies that include a diverse patient population and detailed aneurysm characteristics are needed to refine guidelines, standardize care, and ensure that management strategies are evidence-based and patient-centered.

Emerging high-resolution vessel-wall imaging detects vessel-wall enhancement, which has been associated with inflammation and instability. With unstable aneurysms having an odds of showing wall enhancement on imaging that is 20 times as high as that with stable aneurysms,[45] high-resolution vessel-wall imaging has shown 95.0% sensitivity and 62.7% specificity for detecting aneurysm growth and rupture, which make it a promising tool for stratifying rupture risk and guiding management.[46] Further studies evaluating its clinical usefulness and effect on outcomes are needed.

GUIDELINES

The American Heart Association and American Stroke Association recommend screening for women, smokers, and persons who have hypertension, at least two relatives with a UIA or subarachnoid hemorrhage, or autosomal dominant polycystic kidney disease (class I, level of evidence B). Screening should also be offered to those with coarctation of the aorta and microcephalic osteodysplastic primordial dwarfism (class IIa, level of evidence B).[3] Cost-effectiveness analyses support screening high-risk persons 20 years of age or older with the use of CTA and MRA every 5 to 7 years, noting net harm with screening the general population.[47,48] Patients eligible for screening should be counseled on its benefits, and relatives should be considered for screening if a UIA is identified in the patient.

For small, asymptomatic aneurysms, the guidelines recommend initial follow-up at 6 to 12 months, with imaging interval extended to every 1 or 2 years thereafter for stable aneurysms on imaging (class IIb, level of evidence C).[3] On the other hand, symptomatic or larger aneurysms, especially those exceeding 10 mm in diameter, may warrant surgical interventions, such as clipping or endovascular treatment. Guidelines recommend clipping on the basis of aneurysm size, location, and patient age (class I, level of evidence B) and endovascular treatment for carefully selected cases, particularly in high-volume centers (class IIb, level of evidence B or C). Long-term annual follow-up imaging is recommended to monitor for recurrence or new aneurysms after treatment (class IIb, level of evidence B).

In our practice, we closely follow screening recommendations for high-risk patients, especially smokers, persons with hypertension, and those with a family history of UIA or subarachnoid hemorrhage. For treatment, we perform follow-up imaging for small, asymptomatic aneurysms every 6 to 12 months initially and follow the guidelines for surgical and endovascular treatment. We consider newer endovascular treatment strategies, such as flow diversion, which offers a promising alternative to specific patients, particularly those with anterior circulation internal carotid aneurysms.

CONCLUSION

The patient in the vignette has a small (6 mm), asymptomatic saccular aneurysm emanating from the posterior communicating artery, her medical history includes hypertension and smoking, and she has a family history of subarachnoid hemorrhage. According to size alone, the risk of rupture is low; however, given the patient's moderate risk of rupture (a PHASES score of 5 [on a range of 0 to 22, with higher scores indicating a higher risk of rupture]) based on her relatively young age, the irregular aneurysm shape, a positive family history, the presence of hypertension, and her smoking history, I would favor an invasive treatment strategy. Discussions with the patient will involve weighing the two treatment approaches, with surgical clipping offering a more definitive yet more invasive option, whereas endovascular techniques are less invasive, safer, and have similar long-term efficacy. The treatment for this patient will follow a shared decision-making process that involves weighing the risks, benefits, and the patient's lifestyle preferences. While waiting for treatment, or if the patient prefers surveillance monitoring, I would counsel the patient on signs of aneurysm rupture and recommend seeking immediate medical attention if symptoms arise, including a sudden severe headache that feels like the worst headache she has ever had, neck stiffness, and changes in vision.

Disclosure forms provided by the author are available with the full text of this article at NEJM.org.

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