Spontaneous Symptomatic Intracranial Vertebrobasilar Dissection: Initial and Follow-up Imaging Findings[1]

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Purpose: To evaluate initial radiologic findings of symptomatic intracranial vertebrobasilar dissections (VBDs) as well as the results at follow-up imaging of dissections that are conservatively managed.

Materials and Methods: The respective institutional review boards approved this retrospective study and waived the need for informed consent. The initial radiologic findings of 210 patients with 230 symptomatic intracranial VBDs were retrospectively evaluated (48 ruptured, 182 unruptured). Those patients had undergone conventional angiography as well as magnetic resonance imaging and/or computed tomographic angiography, so that angiographic shapes and pathognomonic findings (eg, intramural hematoma, intimal flap) could be reviewed. The primary angiographic shapes of the symptomatic intracranial VBDs were subdivided into three groups: *(a)* dilatation without stenosis, *(b)* pearl-and-string, and *(c)* stenosis without dilatation. Furthermore, the radiologic evolution of conservatively managed symptomatic intracranial VBDs was evaluated. The respective frequencies of the radiologic findings at initial and follow-up imaging studies were compared by using χ^2 tests.

Results: Primary shape differed significantly between ruptured and unruptured symptomatic intracranial VBDs. Most ruptured dissections presented with one of two main structures: dilatation without stenosis or pearl-and-string appearance. The primary shape of unruptured dissections was evenly distributed among the three types of findings. Intramural hematomas were most frequently found in the stenosis-without-dilatation group (42 of 60 [70%]), followed by the pearl-and-string group (27 of 90 [30%]). Intimal flap was most frequently found in the pearl-and-string group (21 of 90 [23%]), followed by the stenosis-without-dilatation group (eight of 60 [13%]). Follow-up results significantly differed by initial VBD shapes: Seventy-four percent (25 of 34) of the dilatation-without-stenosis group showed no change, whereas improvement was observed in 91% (39 of 43) of the stenosis-without-dilatation group ($P < .05$). Intracranial VBDs with intramural hematoma showed improvement in 63% (34 of 54) of cases, progression occurred in 20% (11 of 54), and only 17% (nine of 54) exhibited no change ($P < .05$).

Conclusion: Primary angiographic shapes of symptomatic intracranial VBDs differed between ruptured and unruptured lesions. The stenosis-without-dilatation lesions most frequently exhibited radiologic improvement at follow-up imaging, followed by pearl-and-string and dilatation-without-stenosis lesions.

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Spontaneous intracranial vertebrobasilar dissection (VBD) can manifest with various clinical symptoms, including subarachnoid hemorrhage, ischemic symptoms from impaired posterior circulation, or even local symptoms (most commonly headache), in young adults (1,2). Dissection is believed to occur secondary to a sudden disruption of both the internal elastic lamina and media, with subsequent penetration of circulating blood into the arterial wall, creating an intramural hematoma (3,4). A second hypothesis dictates that formation of an intramural hematoma derives from the vasa vasorum within the media (5). Depending on the tear depth, symptomatic intracranial VBD may exhibit various shapes at angiographic imaging, including stenosis, dilatation, or both (6,7). Specifically, subintimal dissection tends to result in luminal stenosis or occlusion, whereas subadventitial dissection often causes dilatation. Although the radiologic features for dissections of the cervical internal carotid artery and vertebral artery have been well established at both initial and follow-up time points, those of intracranial VBD are less clear. Several small studies have suggested that the angiographic abnormalities of vertebral artery dissections often improve. Data from one such report suggest that in 76% of patients, follow-up imaging shows the angiographic features of the previously identified spontaneous VBD to have subsided or improved (6). Another similar study detailing the serial radiographic findings of unruptured intracranial VBDs showed that spontaneous angiographic cure was obtained in 62% of cases (7).

We hypothesized that primary lesion shape of symptomatic intracranial VBDs may differ between unruptured and ruptured cases and that morphologic changes of symptomatic intracranial VBDs are probably different at follow-up imaging compared with the respective initial angiographic findings. Better characterization of other radiologic features of VBDs—regardless of rupture—at the time of symptomatic and the subsequent morphologic changes at follow-up imaging will greatly help in accurate diagnosis and appropriate treatment. Accordingly, the purpose of the current study was to evaluate the initial radiologic findings of symptomatic intracranial VBD as well as those at follow-up imaging of conservatively managed VBDs.

Advances in Knowledge

- The angiographic structures of symptomatic intracranial vertebrobasilar dissection (VBD) differ between ruptured and unruptured lesions; the former typically manifest with dilatation without stenosis or pearl-and-string appearance, whereas the latter manifests with an evenly distributed spectrum of angiographic findings.
- Follow-up results significantly differ depending on initial angiographic structure of the dissection; dilatation without stenosis frequently shows no change at follow-up imaging, and stenosis without dilatation usually shows improvement.

Materials and Methods

The respective institutional review boards for all participating hospitals approved this retrospective study and waived the need for informed consent.

Patients

All enrolled patients underwent digital subtraction angiography for intracranial VBDs within 2 weeks of symptom onset between January 2001 and December 2010. Patients were recruited by review of a neurointerventional database encompassing four tertiary referral hospitals. All patients met the following inclusion criteria: *(a)* a history of acute clinical symptoms and/or signs relevant to intracranial VBD, *(b)* angiographic evidence of VBD (ie, aneurysmal dilatation of the intracranial vertebrobasilar arterial trunk, the pearl-and-string sign, or tapered steno-occlusion at digital subtraction angiography), and *(c)* results from digital subtraction angiography as well as magnetic resonance (MR) imaging and/or computed tomographic (CT) angiography performed at the time of symptom onset.

Clinical symptoms and signs relevant to VBD included subarachnoid hemorrhage, ischemic symptoms secondary to compromise of the posterior circulation, sudden-onset severe posterior neck pain and/or headache, or any combination of these. Exclusion criteria were as follows: *(a)* definitively traumatic VBD secondary to major trauma, *(b)* iatrogenic VBD, *(c)* incidentally found asymptomatic fusiform dilatations of the vertebrobasilar arteries, *(d)* laboratory or angiographic findings suggestive of vasculitis or fibromuscular dysplasia, or *(e)* the lack of documented MR or CT angiography during the initial evaluation.

In total, 63 VBDs (45 ruptured and 18 unruptured) were excluded because of insufficient initial imaging studies (n = 60), major trauma (n = 1), and probable vasculopathy (n = 2). Accordingly, 210 patients with 230 symptomatic intracranial VBDs (48 ruptured and 182 unruptured) were identified and enrolled; there were 127 men and 83

Implication for Patient Care

- Knowledge of initial and follow-up imaging findings of spontaneous intracranial VBD will greatly help clinicians to predict subsequent morphologic changes on the basis of the initial angiographic findings.

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Abbreviation:
VBD = vertebrobasilar dissection

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Potential conflicts of interest are listed at the end of this article.

women, with ages ranging from 21 to 80 years (median, 47 years).

Initial and Follow-up Radiologic Evaluation

The primary angiographic shapes of the symptomatic intracranial VBDs were subdivided into three groups based on lesion morphologic type: aneurysmal dilatation without stenosis, pearl-and-string (aneurysmal dilatation alternating with stenosis), and steno-occlusive without aneurysmal dilatation. The pathognomonic findings, intramural hematomas, and intimal flap or double-lumen sign were identified by using digital subtraction angiography, unenhanced thin-section CT, T1-weighted fat-suppressed thin-section MR imaging with flow compensation, and/or source images from CT or MR angiography (1,8–10). When there was disagreement in defining the dissection, the final diagnosis was made by consensus among neuroradiologists, stroke neurologists, and/or vascular neurosurgeons on the basis of both clinical and radiologic findings. The relationships between angiographic shape and presentation (ruptured vs unruptured) and pathognomonic findings were evaluated retrospectively.

Natural radiologic evolution of the symptomatic intracranial VBDs was also subdivided into three types according to findings at follow-up imaging: normalization or improvement, no interval change, or progression. For the dilatation-without-stenosis group, improvement was defined as a decrease in aneurysm size and progression was defined as aneurysmal enlargement. For the pearl-and-string group, improvement was defined as a decrease in aneurysm size and an increase in lumen size of the stenotic portion; progression was defined as any aneurysmal enlargement or worsening of the stenotic occlusion. For the steno-occlusion-without-dilatation group, improvement was defined as any increase in lumen size and progression was defined as any stenosis or occlusion progression. Natural radiologic evolution was evaluated among the conservatively managed intracranial VBDs by using follow-up vascular imaging (MR or CT angiography or conventional angiography); this evaluation allowed us to assess the association between initial angiographic shape and VBD morphologic evolution.

Management of Symptomatic Intracranial VBD

All ruptured dissections were endovascularly treated by using internal coil trapping (n = 22), proximal coil occlusion (n = 4), stent-assisted coil placement (n = 4), two or three overlapping stents only (n = 4), or two or three overlapping stents with coil placement (n = 14). The type of endovascular treatment was at the discretion of each interventional neuroradiologist according to the patients' clinical and anatomic status. The balloon-expandable coronary stents were used for reconstructive treatment before the introduction of self-expanding neurovascular stents. Since their introduction, self-expanding neurovascular stents (Neuroform, Boston Scientific, Natick, Mass; Enterprise, Codman Neurovascular, Warsaw, Ind) were preferentially used.

The patients with ruptured VBD who were treated with a stent or stents received a loading dose of dual-antiplatelet medication (100–500 mg of aspirin and 300 mg of clopidogrel) immediately after completion of the procedure, as reported elsewhere (11–13). For unruptured dissection, the treatment strategy was as described in a previous report (10). Briefly, in the early period, most patients with a dissecting aneurysm underwent endovascular treatment because of concern that the dissecting aneurysm would rupture. In the later period, however, as clinicians gained experience with symptomatic intracranial VBD, most patients were medically treated. Forty-nine VBDs were endovascularly treated at the initial presentation. Of the VBDs initially conservatively treated, 10 underwent endovascular treatment for a recurrence or progression of ischemic symptom (n = 4) or an enlarging dissecting aneurysm (n = 6). As a result, 19 VBDs were treated with internal coil trapping, four with proximal coil occlusion, six with stent-assisted coil placement, 15 with a single stent, and 14 with two or three overlapping stents only. The patients with unruptured VBDs who were treated with a stent or stents received dual-antiplatelet premedication (100 mg of aspirin and 75 mg of clopidogrel) for at least 3 days before the treatment. Dual-antiplatelet therapy was maintained for 12–24 weeks and then changed to aspirin monotherapy indefinitely. All patients with ischemic symptoms received anticoagulant or antiplatelet medication for 1–6 months. Patients without ischemic symptoms received analgesic agents with or without antiplatelet medication at their responsible physicians' discretion on the basis of the patients' medical condition and shape of the lesion.

Statistical Analysis

All statistical analyses were performed with software (SAS, version 9.2; SAS Institute, Cary, NC). The respective frequencies of the radiologic findings at initial and follow-up imaging studies were compared with χ^2 tests. Bonferroni correction was also applied to correct for multiple rounds of testing. In addition, imaging findings were tested by using generalized estimating equations (binomial logit model), which considered the correlations within each patient because some patients had multiple lesions. In all cases, statistical significance was defined as a P value less than .05, allowing a 95% confidence interval.

Results

No statistically significant difference in age was observed between patients with ruptured and those with unruptured intracranial VBD (median, 45 vs 48 years; P = .179). Twenty patients (9.5%) had bilateral intracranial VBDs.

Primary lesion shape differed significantly between ruptured and unruptured symptomatic intracranial VBDs: Ruptured VBDs were more likely to present with dilatation without stenosis (20 of 48 [42%]) or pearl-and-string appearance (24 of 48 [50%]) at initial angiography, whereas only 8% (four of 48) of ruptured cases showed stenosis without dilatation. Conversely, the primary angiographic shape for unruptured VBDs was evenly distributed among all three types (Table 1). Among all 48 ruptured

Table 1

Initial Radiologic Findings of Symptomatic Intracranial VBD

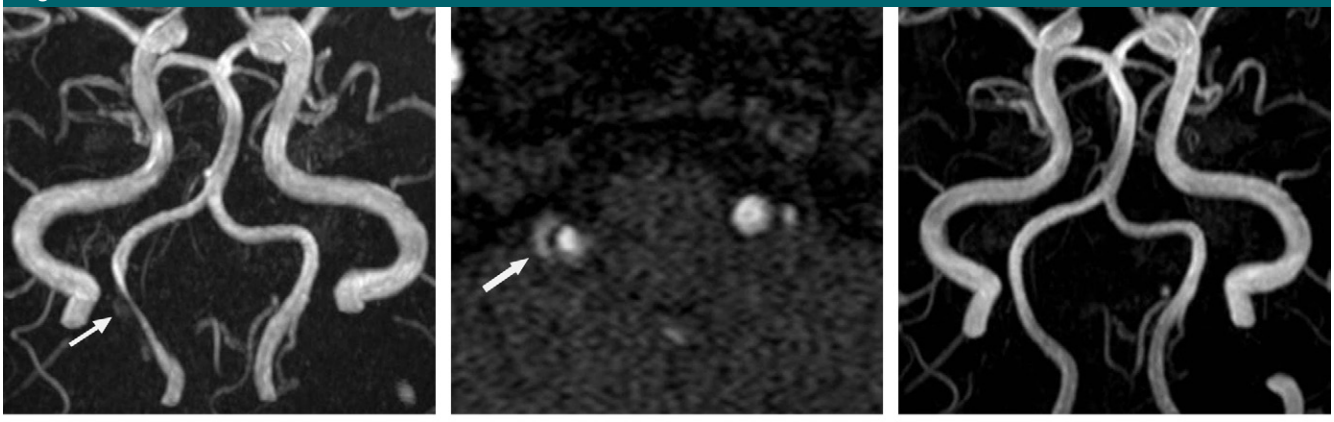
Variable	Dilatation-without-Stenosis Group ($n = 80$)	Pearl-and-String Group ($n = 90$)	Stenosis-without-Dilatation Group ($n = 60$)	*P* Value*	*P* Value†
Rupture				.007	.015
Yes ($n = 48$)	20 (25)	24 (27)	4 (7)		
No ($n = 182$)	60 (75)	66 (73)	56 (93)		
Intramural hematoma				<.001	<.001
Yes ($n = 78$)	9 (11)	27 (30)	42 (70)		
No ($n = 152$)	71 (89)	63 (70)	18 (30)		
Intimal flap				.007	.017
Yes ($n = 34$)	5 (6)	21 (23)	8 (13)		
No ($n = 196$)	75 (94)	69 (77)	52 (87)		

Note.—Data are numbers of cases, with percentages in parentheses.

* *P* value for χ^2 test (per lesion).

† *P* value for generalized estimating equations (per patient).

Figure 1



a. b. c.

Figure 1: VBD shows stenosis without dilatation initially and improvement at follow-up imaging in 36-year-old man with posterior ischemia and headache. **(a)** Time-of-flight MR angiogram and **(b)** its source image show tapered stenosis with subacute hyperintense intramural hematoma at the right distal vertebral artery (arrows). **(c)** Time-of-flight MR angiogram obtained 7 months later shows complete resolution of the previous stenosis and intramural hematoma.

VBDs, only four cases with intimal flap were identified, and no cases with intramural hematoma could be found.

Intramural hematomas were present in 33.9% (78 of 230) of cases, most frequently occurring in lesions that featured stenosis without dilatation (42 of 60 [70%]) (Fig 1), followed by lesions with a pearl-and-string appearance (27 of 90 [30%]) (Fig 2) and dilatation-without-stenosis appearance (nine of 80 [11%]) ($P < .05$). Intimal flaps were present in 14.8% of cases, most frequently occurring in pearl-and-string lesions (21 of 90 [23%]), followed by lesions featuring stenosis without dilatation (eight of 60 [13%]) and those featuring dilatation without stenosis (five of 80 [6%]) ($P < .05$).

During clinical follow-up (range, 8–105 months; median, 47 months), none of the initially unruptured intracranial VBDs subsequently ruptured. Follow-up vascular images were available for 114 of 133 unruptured intracranial VBDs that were initially managed conservatively without surgical or interventional treatment, at a mean of 15 months (range, 1–84 months). Follow-up imaging of these 114 VBDs revealed radiologic findings suggestive of improvement (Figs 1, 2) in 57.9% (66 of 114) of cases, no change in 29.8% (34 of 114), and progression (Fig 3) in 12.3% (14 of 114). Notably, results differed significantly by initial VBD shape (Table 2): The dilatation-without-stenosis group showed no change in 74% (25 of 34) of cases, whereas the stenosis-without-dilatation group showed improvement in 91% (39 of 43) of cases. Similarly, among the 10 VBDs with occlusion at initial imaging, only one lesion remained occluded at follow-up; the other nine lesions were recanalized. Although improvement was

Figure 2

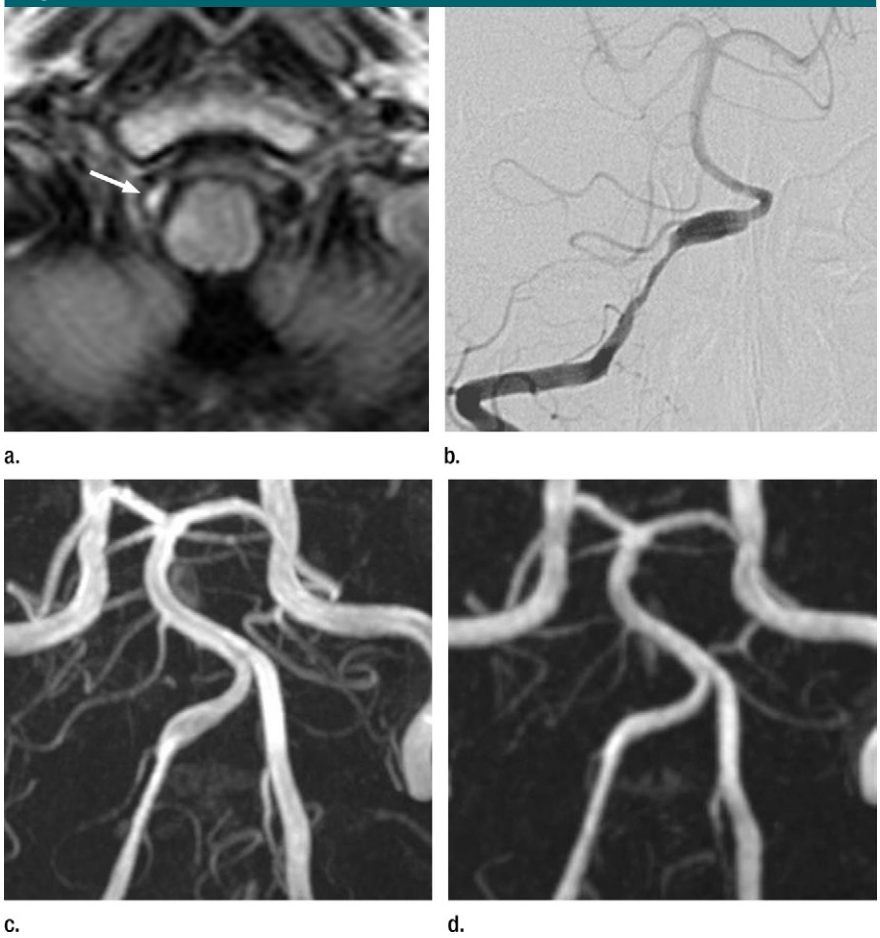


Figure 2: VBD with primary shape of pearl-and-string shows improvement at follow-up imaging in 33-year-old woman with posterolateral medullary infarct. **(a)** Axial precontrast T1-weighted MR image shows hyperintensity of intramural hematoma (arrow). **(b)** Conventional angiogram shows a corresponding pearl-and-string lesion. **(c)** MR angiogram obtained 4 months later shows an improvement of the stenosis, but the fusiform dilatation still remains. **(d)** MR angiogram at 49-month follow-up reveals a normalized right vertebral artery.

common in the pearl-and-string group (21 of 37 [57%]), seven of the 37 cases (19%) in this group were also worsened, a rate approximately two times greater than in the other morphologic groups. In fact, only 9% (three of 34) and 9% (four of 43) of cases exhibited lesion worsening in the dilatation-without-stenosis and stenosis-without-dilatation groups, respectively.

Intracranial VBDs without intramural hematoma at initial angiography showed improvement in 53% (32 of 60) and no change in 42% (25 of 60) of cases. Conversely, VBDs with intramural hematoma exhibited improvement in 63% (34 of 54), progression in 20% (11 of 54), and no change in 17% (nine of 54) of cases. Intracranial VBDs with intramural hematoma showed progression approximately four times more than did VBD without intramural hematoma (20% vs 5%). Follow-up results did not significantly differ by intimal flap presence at initial angiography (Table 2).

Analysis with generalized estimating equations confirmed that statistical significance was not compromised because of the relatively small number of patients with multiple lesions.

Discussion

This study is, to our knowledge, the largest case series to evaluate the evolution at follow-up vascular imaging as well as initial radiologic findings of symptomatic intracranial VBD. Primary lesion shape was found to significantly differ between ruptured and unruptured VBD: Dilated lesions were more likely to rupture than were undilated lesions. Investigators of a previous study postulated that the dilated structure may prove more prone to rupture because the wall of such lesions is composed solely of stretched adventitia (12). The intramural hematoma and intimal flap frequencies also differed by VBD primary shape.

Chronologic changes in symptomatic intracranial VBD are significantly dependent on initial lesion shape. The radiologic feature most predictive of improvement is stenosis without dilatation, whereas most cases that featured dilatation without stenosis did not show dynamic change; this finding is consistent with those noted in a previous report (7).

Several studies suggested several radiologic findings of the VBDs: narrowed taper or occluded, aneurysmal pouching, intimal flap, retention of contrast agent, and pearl-and-string sign (1,2,7). However, because disruption of the internal elastic lamina and the subsequent hyperplasia of the neointima are the two key processes in VBD, we simply categorized VBDs into three morphologic types: dilatation without stenosis, pearl-and-string, and stenosis without dilatation.

We included only patients with acute to subacute symptomatic intracranial VBDs because it was impossible to determine whether incidental asymptomatic findings resulted from spontaneously healed dissections or other underlying vascular abnormalities. In addition, we included both ruptured and unruptured VBDs to see whether any morphologic differences at initial imaging occurred between groups; we chose to include both sets of lesions because the natural course of ruptured VBDs is completely different from that of unruptured lesions.

Follow-up images of the conservatively managed VBDs were available only for unruptured VBDs—all ruptured VBDs require aggressive endovascular or surgical treatment because of the high risk for early rebleeding (13,14).

Although intramural hematoma is a well-known pathognomonic finding of dissection, it occurred in only 33.9% of intracranial VBDs, in accordance with a previous report (1). Such results probably result from the fact that intramural hematoma in the subacute to early chronic stage can be readily detected only with MR imaging because of its paramagnetic effects (15). Conversely, intramural hematoma in the acute stage is rarely detectable at T1-weighted imaging because isointense hematomas are often obscured by surrounding tissue. Intracranial VBDs with concurrent intramural hematomas are more likely to change as the intramural hematoma retracts, and they may eventually resolve in the chronic stage. As such, although any associated stenosis may improve, any associated dilatation may worsen.

As seen in several other studies, the intimal flap or double-lumen sign—another pathognomonic finding of dissection—was uncommon among our patients (14.8%) (6,7,16), perhaps because the repair process with intimal hyperplasia partially reinforces the disrupted internal elastic lamina (4). According to the pathologic classification proposed previously, intimal flaps may be visualized with MR imaging in type 3 dissecting aneurysms with intimal thickening, an infrequent occurrence (3). Other data suggest that intramural hematomas resulting from the rupture of the vasa vasorum may lack connections between false and true lumens (5).

Although dissecting aneurysms are believed to heal through neointimal hyperplasia, many cases exhibiting dilatation without stenosis remained stable in our study. A recent report suggested that several conditions may delay the healing mechanisms: large defects in the aneurysmal wall, abundant thrombus, and the complete separation of the media and internal elastic lamina from the adventitia (12). However, only ruptured intracranial dissections were included in that study, and unruptured dissection may prompt an entirely different healing response. Furthermore, a small number of unruptured dissecting aneurysms exhibited progressive enlargement in the current study. Because damaged internal elastic lamina cannot be regenerated, the healing process should occur by neointimal hyperplasia. Thus, several scenarios can be suggested in cases of unruptured dissecting aneurysm. First, when the repaired neointima can overcome the local hemodynamic stress, the dissecting aneurysm may fully resolve. Second, if the repaired neointima can balance the hemodynamic stress, the dissecting aneurysm may remain stable. However, if repaired neointima cannot withstand the hemodynamic stress because of extensive disruption of the internal elastic lamina, the dissecting aneurysm may progressively enlarge. Hemodynamic stress may also play an important role

Figure 3

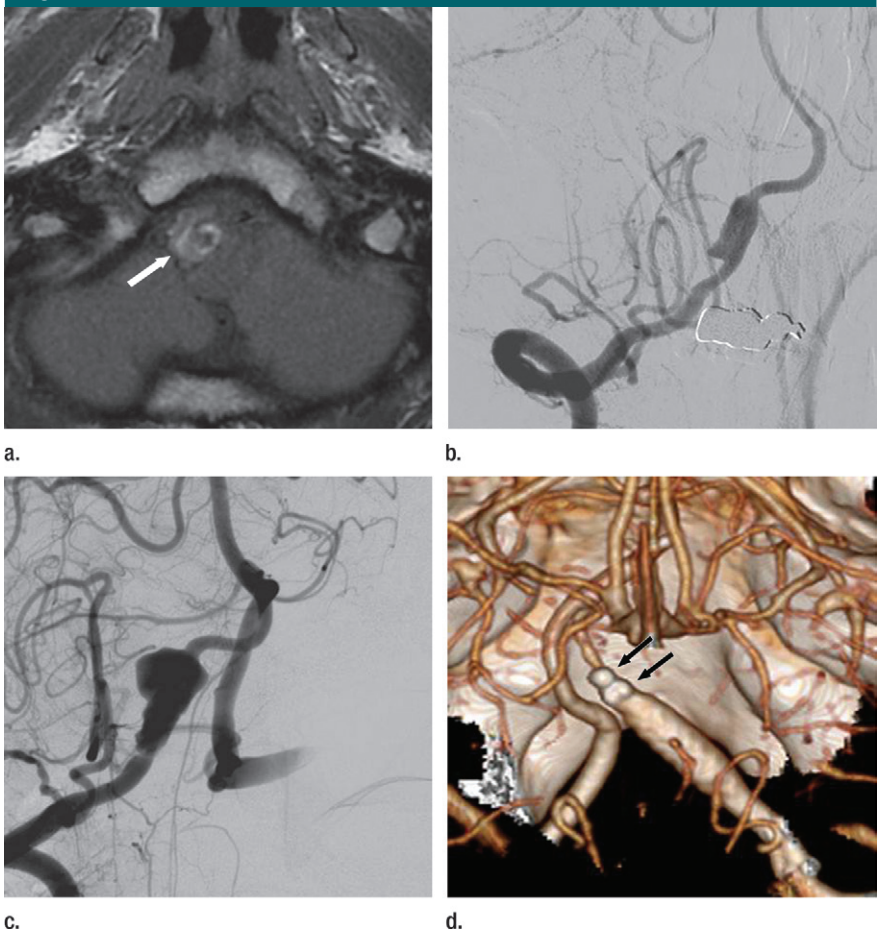


Figure 3: VBD featuring dilatation without stenosis shows progression at follow-up imaging in 34-year-old man with sudden-onset severe posterior headache and neck pain. **(a)** Axial precontrast T1-weighted MR image shows hyperintense intramural hematoma (arrow) in the right vertebral artery. **(b)** Conventional angiogram of right vertebral artery shows fusiform dilatation of the distal vertebral artery without any stenosis. **(c)** Conventional angiogram obtained 3 months after the initial presentation reveals enlargement of aneurysmal dilatation with newly developed proximal stenosis. Therefore, the patient underwent reconstructive treatment with two overlapping self-expanding stents. **(d)** Three-dimensional reconstruction of CT angiogram 6 months after treatment shows disappearance of the dissecting aneurysm with reconstruction of the parent artery. Arrows = distal markers of two stents.

Table 2

Morphologic Changes at Follow-up Imaging According to Initial Radiologic Findings

Variable	Improvement ($n = 66$)	No Change ($n = 34$)	Progression ($n = 14$)	*P* Value*
Shape				<.001
Dilatation without stenosis ($n = 34$)	6 (18)	25 (74)	3 (9)	
Pearl-and-string ($n = 37$)	21 (57)	9 (24)	7 (19)	
Stenosis without dilatation ($n = 43$)	39 (91)	0 (0)	4 (9)	
Intramural hematoma				.003
Yes ($n = 54$)	34 (63)	9 (17)	11 (20)	
No ($n = 60$)	32 (53)	25 (42)	3 (5)	
Intimal flap				.925
Yes ($n = 15$)	8 (53)	5 (33)	2 (13)	
No ($n = 99$)	58 (59)	29 (29)	12 (12)	

Note.—Data are numbers of cases, with percentages in parentheses.

* *P* value for χ^2 test (per lesion).

in repeat dissection, resulting in chronically enlarging dissecting aneurysms with multilayered intramural hematomas in some cases with extensive damage to the internal elastic lamina.

This study had several limitations. First, because our cases were collected from four institutions, the imaging techniques were not standardized. Moreover, the methods used for conservative management were entirely at the physicians' discretion, possibly affecting morphologic changes at follow-up imaging. Second, we did not analyze vascular risk factors. However, the goal of this study was to evaluate and compare the initial radiologic findings with later morphologic changes at follow-up imaging, and the results would be unlikely to be affected by vascular risk factors. Third, follow-up radiologic changes of VBD were evaluated at various time periods because of the retrospective nature of this study.

In summary, primary angiographic shapes of symptomatic intracranial VBDs differed significantly between ruptured and unruptured lesions, and their chronologic changes are significantly dependent on initial lesion shape.

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