Strategy for Treating Unruptured Vertebral Artery Dissecting Aneurysms

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BACKGROUND: The natural course of unruptured vertebral artery dissecting aneurysms (VADAs) remains unclear.

OBJECTIVE: The purpose of this retrospective study was to develop a strategy for treating unruptured VADAs based on long-term follow-up.

METHODS: Our study population consisted of 100 patients with unruptured VADAs; in 66, the initial symptom was headache only, 30 presented with ischemic symptoms and 4 with mass effect. All underwent magnetic resonance imaging and magnetic resonance angiography at the time of admission and 2 weeks and 1, 3, 6, 12, and 24 months after the onset. If the dissection site was demonstrated to be enlarged on magnetic resonance imaging and magnetic resonance angiography without the manifestation of new symptoms, the patients received additional treatment to prevent bleeding.

RESULTS: Of the 100 patients, 4 underwent early intervention because of symptom exacerbation. The other 96 were initially treated conservatively; during follow-up, 5 manifested lesion enlargement on magnetic resonance angiography. Nine patients received additional treatment; 1 underwent direct surgery with trapping of the dissection site, and 8 underwent coil embolization. The other 91 patients continued to be treated conservatively; the dissection site remained unchanged in 70, improved or healed in 18, and disappeared in 3 patients. We treated 38 patients with recurrent ischemic attacks with antiplatelet therapy. No patients experienced bleeding or permanent neurological deficits during follow-up.

CONCLUSION: The nature of an unruptured VADA is not highly aggressive. However, if the dissection site enlarges without the manifestation of new symptoms, it should be occluded. In patients with recurrent ischemic attacks antiplatelet therapy should be considered.

KEY WORDS: Antiplatelet drugs, Cerebrovascular disease, Dissection, Magnetic resonance imaging, Vertebrobasilar disease

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The primary presentation of an unruptured intracranial vertebral artery (VA) dissection is severe occipital headache only or focal neurological deficits caused by vertebrobasilar artery ischemia; in patients with rupture, it is subarachnoid hemorrhage (SAH). The natural course and prognosis of these lesions strongly depend on the initial pattern of presentation.[1-6] Patients with SAH are usually treated by early open surgery to repair or by endovascular procedures to obliterate the aneurysm because rebleeding in the acute stage is often fatal.[1,2,4,7] On the other hand, as unruptured VA dissection tends not to follow an aggressive clinical course, and because the prognosis tends to be satisfactory, conservative treatment of these lesions has been advocated.[1,3,4,8,9] Because their optimal treatment and appropriate follow-up period have not been established, we studied the natural course of unruptured VA dissecting aneurysms (VADAs) and discuss strategies for their treatment.

ABBREVIATIONS: MRA, magnetic resonance imaging; **PICA,** posterior inferior cerebellar artery; **SAH,** subarachnoid hemorrhage; **VA,** vertebral artery; **VADA,** vertebral artery dissecting aneurysm

MATERIALS AND METHODS

Between January 2003 and December 2009, 100 patients with unruptured VADAs presented to our hospital and associated institutions. They were 72 men and 28 women ranging in age from 33 to 83 years (mean, 61.2 years). The initial symptoms were headache only in 66, ischemic symptoms in 30, and mass effect in 4 patients. The interval between symptom onset and the first examination ranged from 0 to 45 days (mean, 5.8 days); it was 5.6 days if the initial symptom was headache, 3.7 days if it was ischemic, and 9.6 days in patients with mass effect. All patients underwent magnetic resonance imaging (MRI) and magnetic resonance angiography (MRA) studies; our MRI protocol revealed nonhemorrhagic findings. To detect VADAs, imaging studies included the conventional spin-echo technique with T1-weighted, proton density, T2-weighted, fluid-attenuated inversion recovery, and magnetization-prepared rapid acquisition with gradient echo study of axial images. The MRA technique used 3-dimensional time-of-flight imaging. Diagnostic criteria included long stenotic segments exhibiting the "string sign," tapered stenosis or occlusion, pseudoaneurysm, intimal flap formation, and luminal irregularity. In addition, the presence on standard MRI scans of areas exhibiting crescent-shaped high signal intensity within a vessel wall (mural hematoma or double lumen) was considered indicative of dissection. All imaging studies were independently evaluated by 2 neurologists (T.H., M.W.); disagreement was resolved by consensus. Dissections identified by only 1 of the 2 imaging techniques were further evaluated retrospectively by both neurologists. All patients and/or their families gave informed consent for this study. The study protocol was approved by the clinical investigation committee of Kumamoto University.

The treatment strategy used at our institution is shown in Figure 1. Patients with progressive ischemic symptoms in the week after the insult received antiplatelet therapy; those in whom symptoms from mass effect failed to improve were treated with steroids. If there was no symptom improvement despite these treatments within 2 weeks of onset, we performed angiography to further evaluate the VADA.

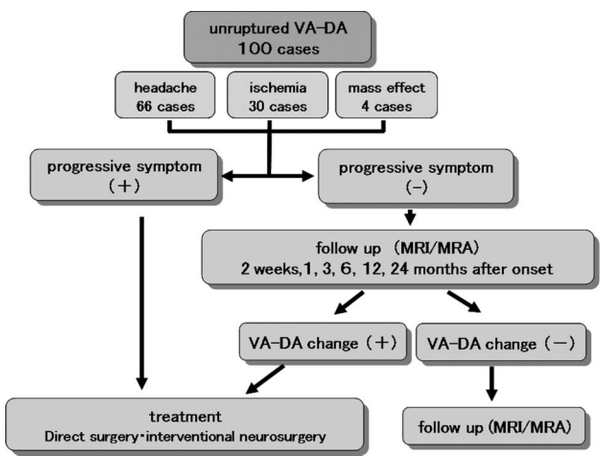

FIGURE 1. *Strategy for treating vertebral artery dissecting aneurysm (VADA). MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.*

Patients were treated conservatively if their symptoms were headache only or their ischemic and mass effect symptoms were not aggravated. Their systolic blood pressure was controlled at less than 140 mm Hg with or without antihypertensive agents. The target blood pressure was as set forth by the American Heart Association.[10] All conservatively treated patients underwent MRI and MRA studies at 2 weeks and 1, 3, 6, 12, and 24 months after the onset. Angiography was performed if the dissection site was shown to be enlarged MRI and MRA scans without the manifestation of new symptoms and in patients with progressive neurological symptoms despite the delivery of antiplatelet therapy for recurrent ischemic attacks or steroid treatment for mass effect.

A 4-French catheter (Medikit, Tokyo, Japan) was inserted in the affected VA. We assessed the relationship between the VA dissection site and the ipsilateral posterior inferior cerebellar artery (PICA). In patients with progressive enlargement of the dissection site, we undertook additional treatment. In such cases, we inserted a 6-French guiding catheter (Medikit) into the affected VA. To test occlusion of the VA dissection, we used a nondetachable balloon; the occlusion site was adjacent or just proximal to the dissection site. The balloon was inflated with contrast medium to create an arterial occlusion, the patient was monitored for 20 to 30 minutes, and neurological findings were recorded. During these procedures, the contralateral VA was studied to evaluate flow in this vessel and to detect retrograde filling of the aneurysm.[10] The relationship between the dissection site and the ipsilateral VA was confirmed. The type of additional treatment depended on the relationship between the VA dissection site and the ipsilateral PICA. When the dissecting aneurysm did not involve the PICA, it was trapped with Guglielmi detachable coils (Boston Scientifics, Natick, MA, USA) using the double microcatheter technique.[11] If there was PICA involvement, we performed direct surgery, trapping the aneurysm and reconstructing the PICA via occipital artery–PICA anastomosis.

The management outcome was evaluated using the modified Rankin scale at 2-year follow-up. The evaluation was performed by 2 neurologists (T.H., M.W.) at outpatient follow-up visits.

RESULTS

In 4 of our 100 patients, we observed progression of the initial symptoms within 2 weeks of onset; all ischemic symptoms worsened despite antiplatelet therapy. Consequently, 3 patients underwent proximal occlusion of the VA with Guglielmi detachable coils; the other patient was treated by direct surgery to trap the dissection site and to create an occipital artery–PICA anastomosis because the dissection site involved the origin of the PICA.

The other 96 patients were treated conservatively; their systolic blood pressure was controlled to less than 140 mm Hg, and they underwent MRI and MRA studies 2 weeks and 1, 3, 6, 12, and 24 months post-onset (Figure 2). At 2-week follow-up, 2 patients manifested symptom progression (ischemic neurological deficits, n = 1) and changes in MRA findings (progressive aneurysmal dilation, n = 1); they underwent coil embolization of the dissection site. At 1-month follow-up, 2 patients were found to have progressive symptoms (dysphagia caused by mass effect in 1 patient and enlargement of the pearl-and-string sign on MRA in the other); they also underwent coil embolization of the dissection site. At 3-month follow-up, 1 patient exhibited a change in

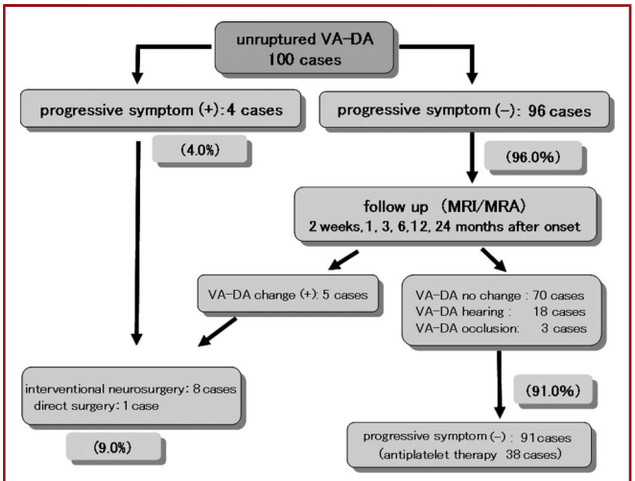

FIGURE 2. *Treatment results obtained in 100 patients with vertebral artery dissecting aneurysm (VADA). MRA, magnetic resonance angiography; MRI, magnetic resonance imaging.*

MRA findings; enlargement of the aneurysmal dilation was identified on pearl-and-string findings; this patient underwent coil embolization of the dissection site (Figure 3). At 6-, 12-, 24-month follow-up, no patient presented with findings that required additional treatment.

Consequently, 4 patients were treated in the acute stage within 2 weeks of onset (direct surgery in 1 patient and interventional treatment in 3 patients), and 5 were treated during follow-up (interventional treatment, n = 5). In this series, 9 of 100 patients with VADAs were treated early post-onset or during follow-up by direct surgery or interventional treatment using Guglielmi detachable coils. Direct surgery consisted of trapping the dissection site and occipital artery–PICA anastomosis (n = 1); interventional treatment was coil embolization in 8 (proximal occlusion, n = 5; coil trapping, n = 3). None of the patients experienced bleeding, and all but 1 had good treatment outcomes. We encountered neither additional neurological deficits nor aneurysmal ruptures. One patient required steroid pulse treatment because of transient worsening of brainstem compression after coil embolization of the dissection site; dysphagia attributable to brainstem edema resolved completely (Figure 4).

None of the 91 conservatively treated patients manifested bleeding or additional neurological deficits. In 70 patients, the dissection site resolved, and there was no change on subsequent MRI and MRA scans (Figure 5). The dissection site was visualized as aneurysmal dilation in 10, the pearl-and-string sign in 52, and the string sign only in 8 of these 70 patients. In 18 patients, the dissection site showed healing on follow-up MRA; we noted aneurysmal dilation in 5, the pearl-and-string sign in 8, and the string sign in 4. The dissection site disappeared completely in 3 patients, 2 had manifested the pearl-and-string sign, and the other the string sign only; disappearance of the dissection site was noted at 1- and 6-month follow-up in 2 patients and 1 patient, respectively. Follow-up MRA studies confirmed that no additional neurological symptoms or recanalization developed in these 3 patients.

The 38 patients with recurrent ischemic attacks were treated with antiplatelet therapy (initial symptoms: headache, n = 18, ischemic episodes, n = 20); it was started after the second ischemic attack that occurred between 2 days and 3 months after the first (mean, 7.2 days). Only 1 patient underwent interventional treatment at 2-week follow-up because the ischemic symptoms persisted despite antiplatelet therapy. In these patients, there was no aneurysmal dilation; 20 manifested the pearl-and-string sign and 18 the string sign only. All 38 patients receiving antiplatelet therapy remained free of bleeding or permanent neurological deficits during follow-up; antiplatelet therapy was continued for at least 2 years.

All 100 patients in this series remained free of bleeding during follow-up; their modified Rankin Scale score at 2-year follow-up was 0 to 1 (Figure 2).

DISCUSSION

Most unruptured VADAs do not have an aggressive clinical course[1,4,9,12]; however, their optimal treatment has not been established, and it is unclear how long they should be followed because few large series of patients with VADAs have been reported. Our study population consisted of 100 patients with unruptured VADAs, and we followed them for 2 years. Although sudden-onset occipital headache or neck pain is highly suspect of a VADA, the diagnosis of an unruptured VADA remains difficult. Conventional angiography, although useful for the diagnosis of dissecting aneurysms, places patients under stress and may elicit complications. Because MRI and MRA studies are noninvasive, they can be performed repeatedly to monitor the site of dissection for changes over time.[13] On T1-weighted and fluid-attenuated inversion recovery MRI scans, intraluminal VA hematomas can be detected and the pearl-and-string and the double lumen signs are characteristic MRA findings of VADAs.[12,14-16] On 3-dimensional contrast-enhanced images, the double lumen at the site of dissection is visualized.[15] Basi-parallel anatomic scanning MRI, a technique that reveals the surface contour of the intracranial vertebrobasilar artery, aids in the evaluation of the clinical course of unruptured VA dissections.[17-19] In our study of VADAs, we performed MRI and MRA during long-term follow-up. If these imaging findings showed a change in a VADA, we acquired conventional angiograms.

The appropriate length of follow-up in patients with unruptured VADAs remains to be established. In some cases, changes at the site of dissection were observed 2 to 3 months post-onset,[1,9] whereas in others, MRI performed within 6 months failed to show hyperintensity indicative of intraluminal hemorrhage at the dissection site.[20,21] Nakagawa et al[15] reported that tapered occlusion demonstrated on initial examination required a relatively long time for complete recanalization. In 5 of our patients, the need for additional treatment was recognized within

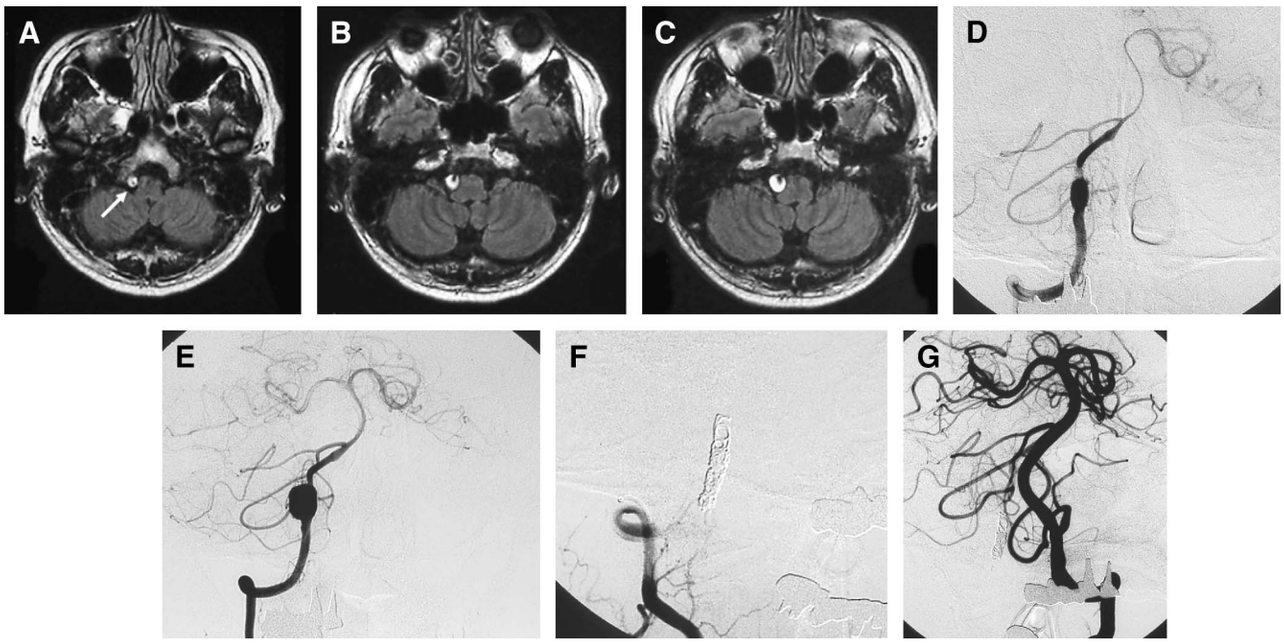

FIGURE 3. *A 44-year-old man who presented with right occipital headache.* **A-C,** *fluid-attenuated inversion recovery magnetic resonance imaging. Changes in the right vertebral artery (VA) are visualized as hyperintensity areas. A, admission scan. Hyperintensity signal (arrow) indicative of intramural hematoma in the right VA.* **B,** *1 month post-onset. The lesion shows gradual enlargement.* **C,** *3 months post-onset. The lesion shows further enlargement.* **D,** *right vertebral angiogram obtained at admission shows dilation of the right VA proximal to the right posterior inferior cerebellar artery (PICA).* **E,** *3 months post-onset. Right vertebral angiogram shows enlargement of the right VA.* **F,** *right vertebral angiogram obtained after embolization of the aneurysmal dilation shows disappearance of the vertebral artery dissecting aneurysm.* **G,** *left vertebral angiogram obtained after embolization of the aneurysmal dilation shows preservation of the basilar artery and bilateral PICA, posterior cerebral arteries, and superior cerebellar arteries.*

3 months post-onset; none of the 100 patients required additional treatments at 6, 12, or 24 months post-onset. Therefore, we suggest that the minimum follow-up period in patients with an unruptured VADA is 6 months and that active follow-up for more than 2 years is not necessary.

Serial angiography studies during follow-up revealed that occlusive changes were not necessarily predictive of a poor outcome unless the contralateral VA was hypoplastic.[21] Recanalization was observed in patients whose initial angiograms revealed nontapered occlusion or only the string sign,[9] and in some patients,

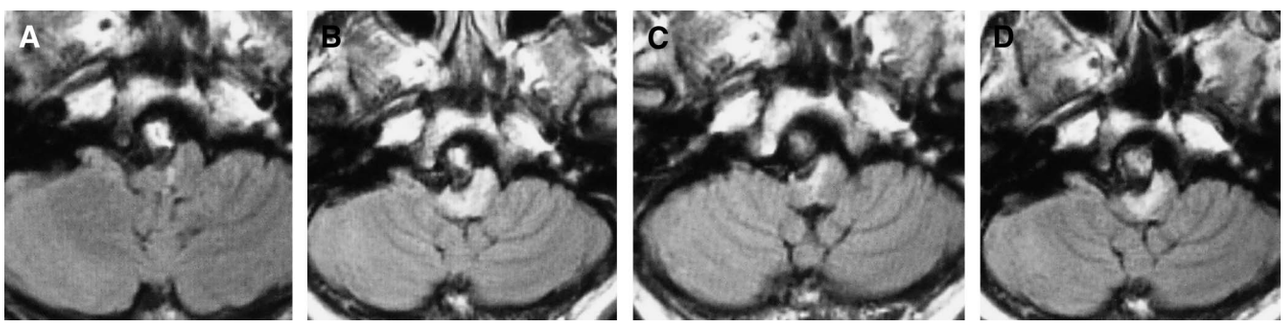

FIGURE 4. *A 66-year-old man who presented with right occipital headache and mild double vision.* **A,** *admission scan. Fluid-attenuated inversion recovery (FLAIR) magnetic resonance imaging (MRI) revealed a hyperintensity mass lesion in the right vertebral artery (VA). The right side of the medulla was compressed by the mass lesion and a small hyperintensity area was shown in the medulla.* **B,** *1 month post-onset. FLAIR MRI showed progression of the hyperintensity in the medulla. There was no change in the hyperintensity of the right VA whose size remained almost constant.* **C,** *after embolization of the dissecting aneurysm of the right VA, FLAIR MRI demonstrated the disappearance of the hyperintensity area in the medulla.* **D,** *1 month post-embolization. FLAIR MRI demonstrated the reappearance of the hyperintensity area in the medulla.*

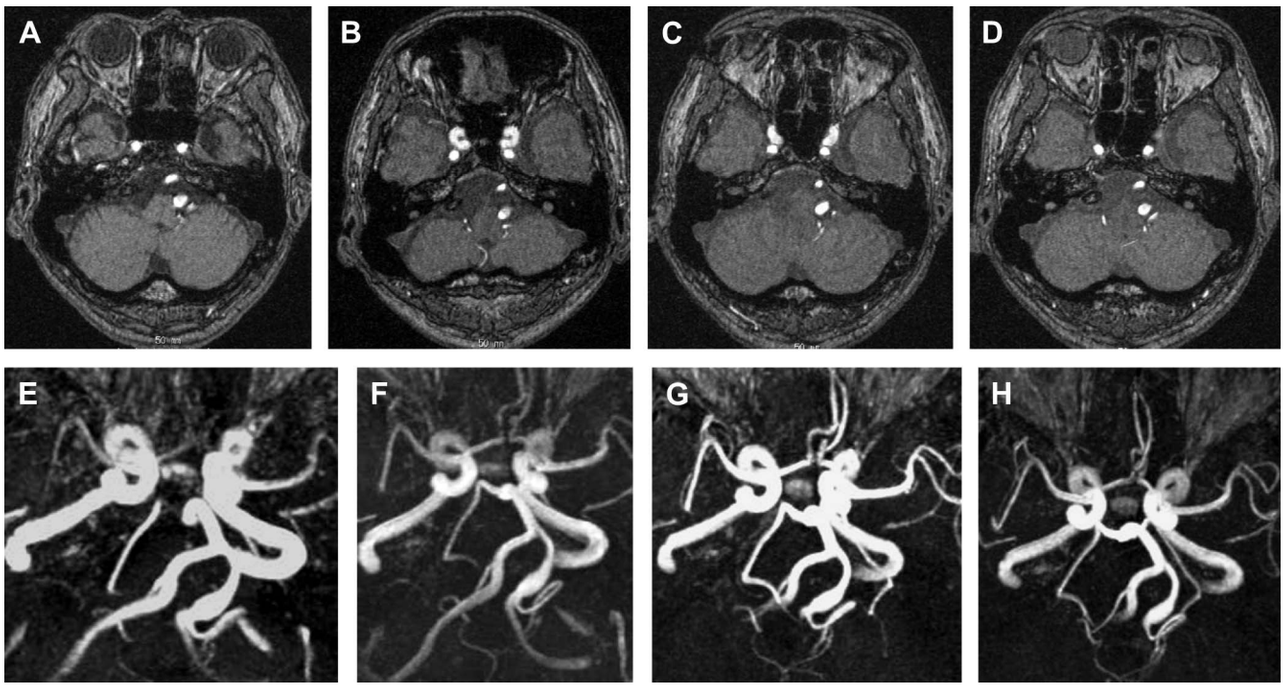

FIGURE 5. *A 66-year-old man who presented with left occipital headache.* **A-D,** *magnetization-prepared rapid acquisition with gradient echo magnetic resonance imaging (MRI) demonstrating hyperintensity and dilation of the left vertebral artery.* **E-H,** *Magnetic resonance angiography showing the left vertebral artery dissecting aneurysm (VADA).* **A,E,** *on admission;* **B, F,** *1 month later;* **C, G,** *6 months later;* **D, H,** *12 months later. Findings on the left VADA remained unchanged.*

the VADA resolved spontaneously.[1,4,22] Our study included 18 patients with MRA evidence of spontaneous improvement or gradual healing of the dissection site without surgical intervention. In 5 patients, vascular imaging findings at the site of dissection had normalized; in 3, the pearl-and-string sign was shown and in the other 2, only the string sign was noted.

The natural course of an unruptured VADA has been reported to be relatively benign.[1,6,9,15] On the other hand, Naito et al[14] found that the bleeding rate from unruptured VADAs was higher than expected. They encountered 3 patients with 21 unruptured VADAs who subsequently experienced SAH; in 2 patients, SAH occurred within a day after initial symptom onset. Tsutsumi et al[23] reported 9 patients with unruptured VADAs who later experienced SAH; in 7, it occurred between 9 days and 1 month (mean, 6.7 days) after symptom onset. According to Mizutani et al,[24] two thirds of patients with unruptured VADAs experienced bleeding within 3 days of first experiencing headache. These observations suggest that if 4 or more days pass without bleeding episodes, the prognosis of patients with unruptured VADAs may be good. Our findings also indicate that the natural course of unruptured VADAs is benign. In our series, the interval between initial symptom onset and the first medical examination was 5.8 days, suggesting that the clinical course of many of our patients was excellent.

Patients with aneurysmal enlargement during follow-up should be carefully monitored. Therefore, patients whose VADAs enlarge, especially during the first month after symptom onset, must be carefully monitored.[14] According to Yamaura et al,[25] 3.4% of ischemia-producing VADAs resulted in SAH. Iihara et al[26] recommended follow-up angiography in patients with unruptured VADAs during the early stage (<3 weeks) after presentation and therapeutic intervention to prevent bleeding if aneurysm formation is observed. We detected aneurysmal changes during follow-up in 5 of our VADA patients; all underwent surgical intervention, and none experienced bleeding during the 2 years of follow-up.

In determining the indications for parent artery occlusion, the risk of bleeding in conservatively treated patients and the risk of ischemic complications in patients subjected to parent artery occlusion must be considered. If occlusion is contemplated, test occlusion is necessary.[11] During this procedure, the blood supply from the anterior and posterior spinal arteries and the perforating arteries, as well as the cross-flow from the contralateral side and the relationship between the aneurysm and the PICA, must be assessed. Favorable clinical outcomes were obtained in some patients whose VADAs were addressed by stenting with or without coiling.[27,28] This reconstructive method aims at the preservation of the VA flow. We posit that stent placement will become the primary treatment for patients

with VADAs. However, intracranial stents have not yet been approved for use in Japan.

In patients with VADAs, recurrent ischemia may be attributable to thrombotic occlusion of the stenotic lesion, to distal emboli, or to further occlusion of perforating arteries originating from the VA due to progression of the dissection. Yoshimoto and Wakai[9] reported a low rate of recurrent ischemic attacks and mild symptom exacerbation in patients with unruptured VADAs. Naito et al[14] administered antiplatelet or anticoagulant therapy in patients with VA occlusion or stenosis without aneurysmal dilation. However, the administration of antiplatelet or anticoagulant agents is controversial in patients with ischemic symptoms because although these agents may effectively prevent thrombotic occlusion or distal emboli, they may promote progression of the dissection, resulting in exacerbation of ischemic symptoms or aneurysmal rupture. Our 38 patients with recurrent ischemic events received antiplatelet therapy; none manifested aneurysmal dilation, and in 20, the pearl-and-string sign and in 18, the string sign only were noted. We tend to restrict the use of such agents to patients with angiographic evidence of occlusion or stenosis without aneurysmal dilation.

Disruption of the internal elastic lamina is the primary mechanism underlying arterial dissection.[29,30] In the subsequent healing process, leukocytes and macrophages infiltrate the dissection site, endothelium covers the lesion, synthetic smooth muscle cells appear, followed after several months by neointima formation.[31] Postmortem pathological studies of patients with VADAs suggest that vascular dissection is attributable to rupture of the internal elastic lamina.[32] Most defects were located anterior to the penetration of the dura mater and near the branching of the PICA 10 to 20 mm distal to the site of penetration, a finding in agreement with the report of Sato et al,[5] who suggested that this may be related to the development of dissection at these sites. Ro et al[33] reported that intracranial VADAs tend to induce multiple lesions that affect both intracranial VA iteratively. Their observations point to the importance of monitoring these vessels carefully to prevent SAH. The incidence of prodromal symptoms was significantly higher among patients with earlier intracranial VA dissections, indicating that the early diagnosis of intracranial VA dissection before the manifestation of rupture is necessary to protect against fatal SAH. We plan to investigate further minor dissection signs in patients with unruptured VADAs in an effort to prevent subsequent progression of the dissection.

CONCLUSION

Unruptured VA dissections are not usually aggressive. If the risk of ischemic complications associated with parent artery occlusion is low, early endovascular treatment should be considered in patients with relatively large or growing aneurysmal dilations. After 1 month, the risk of bleeding decreases; none of our 100 patients required additional treatments at 6, 12, or 24 months post-onset. Based on our experience we recommend that patients with unruptured VA dissections be treated conservatively and monitored closely for 6 months.

Disclosure

The authors have no personal financial or institutional interest in any of the drugs, materials, or devices described in this article.

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COMMENTS

Dissecting vertebral artery aneurysms are unusual lesions, but are often seen in the setting of acute subarachnoid hemorrhage or posterior circulation stroke. This study provides a fairly optimistic assessment of the natural history of unruptured vertebral dissections. In 100 patients followed for at least 2 years, there were no episodes of delayed rupture, and very few neurological problems that required invasive treatment. However, the average interval from symptom onset to presentation in these study patients was almost 6 days, and some patients were first seen more than 1 month after initial symptoms. It would appear that the highest risk for patients after vertebral artery dissection is in the first few days after symptoms begin. Therefore, this series may be somewhat preselected for good outcomes because there must be some patients who had rupture or major stroke in the first few days after initial symptom onset, and these patients would not be included in this analysis.

Although this study does not help us in managing patients who present with acute vertebral artery dissection without hemorrhage, the results clearly support conservative treatment in incidentally discovered dissections and those patients who have had minimal symptoms for several days before presentation.

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Kai et al present their findings from a retrospective analysis of their treatment protocol for unruptured vertebral artery dissecting aneurysms (VADAs). They show the results of 100 patients initially presenting with headache, an ischemic event, or mass effect from the lesion. Patients were monitored for persistent symptoms or lesion progression on repeat MRI/MRA. Without symptom progression, repeat MRI/MRA was obtained at 2 weeks and 1, 3, 6, 12, and 24 months. Those patients found to have recurrent symptoms or progression on imaging underwent either surgical or endovascular intervention. In their results, they found that 9% required intervention, whereas 91% were managed conservatively. Because of recurrent ischemia, 38 patients were placed on antiplatelet therapy. Regarding the imaging findings, 5 patients had progression requiring treatment. All these changes occurred within the first 6 months of follow-up. No patients experienced SAH.

From these findings the authors conclude that unruptured VADAs are not usually aggressive. They further recommend that although in the first 6 months close follow-up is necessary, imaging after 6 months is not necessary.

Because of the poor prognosis of ruptured VADAs (rehemorrhage rates >50% and mortality up to 87%), aggressive management has been recommended. A number of retrospective studies, however, have suggested that the hemorrhage rate of unruptured VADAs is low. This study is one of the largest series evaluating unruptured VADAs and gives further evidence of this by showing a relatively unaggressive natural history and 0% hemorrhage rate. However, there are previous studies such as that by Naito et al that suggest that subarachnoid hemorrhage (SAH) occurs more often than previously expected. They found hemorrhage rates greater than 10% in their evaluation of 21 patients and because of this suggest early treatment.

The findings presented are evidence that a management protocol with close follow-up and early aggressive treatment for those lesions undergoing dynamic changes has good outcomes and low occurrence of SAH (0%). Also, important is the finding that these lesions appear to reach some level of stability within 6 months of presentation. Although their 2-year follow-up is notable, given the potential devastating consequences of these lesions, we would be hesitant to stop following these patients at this time.

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The natural history of spontaneous intracranial vertebral artery dissection and its optimal management are incompletely understood. This study adds to our knowledge through a careful analysis and 2-year follow up of 100 patients managed at 1 institution by an established protocol. Of the 100 patients, 72 were male and the mean age was 61.2 years. Presenting symptoms were headache in 66, ischemic symptoms in 30, and mass effect in 4. Diagnosis was established by MRI/MRA, and angiography was performed only if the dissection site enlarged on follow-up MRI/MRA and in patients with progressive symptoms despite medical management. That medical management consisted of antiplatelet therapy for patients presenting with ischemic symptoms and steroids for those presenting with mass effect.

Remarkably, only 9 of the 100 patients required intervention beyond medical therapy. Four experienced symptom progression within the first 2 weeks and underwent endovascular occlusion of the affected vertebral artery (n = 3) or surgical occlusion with a bypass from the occipital to the posterior inferior cerebellar artery (n = 1). The other 5 patients requiring endovascular occlusion had progressive symptoms at 2 weeks (n = 2), symptom progression at 1 month (n = 2), and

a change in the MR appearance at 3 months (n = 1). Thereafter, no additional treatment was required at 6-, 12-, or 24-month follow-up. No patients experienced bleeding, and all had a modified Rankin Scale score of 0 to 1 at 2-year follow-up. On follow-up imaging of the 91 conservatively treated patients, 70 remained stable, 18 had healed, and 3 had disappeared.

This study further establishes MRI/MRA as the primary imaging modality for the diagnosis and re-evaluation of intracranial vertebral artery dissection, suggests that a minimum follow-up of 6 months is required for these patients with none in this study experiencing a progression of symptoms after 3 months, and establishes the safety of antiplatelet therapy for patients with ischemic symptoms. In those requiring intervention, endovascular occlusion was the mainstay. In the future, endovascular stenting with parent artery preservation may likely play a more important role.

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