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AJNR Am J Neuroradiol 2003, 24 (4) 691-699
http://www.ajnr.org/content/24/4/691

AJNR Am J Neuroradiol 24:691–699, April 2003

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BACKGROUNDAND PURPOSE: **Case reports of nontraumatic arterial dissection of the anterior cerebral artery (ACA) have recently increased. The aim of this study was to investigate the neuroradiologic and clinical features of ACA dissection based on a series of collected cases.**

METHODS: **The cases of 18 patients with a diagnosis of ACA dissection based on clinical signs and neuroradiologic findings from 46 stroke centers during a 5-year period were collected. The neuroradiologic and clinical records were analyzed.**

RESULTS: **The mean patient age was 52.8 ± 9.8 years. Five cases presented with subarachnoid hemorrhage, nine with cerebral ischemia, and four with both ischemic symptoms and subarachnoid hemorrhage. In cases presenting with ischemia, the main site of the lesion was the A2 portion and the main angiographic finding was stenosis with or without dilation. Follow-up angiography showed progression of the stenosis in the acute stage and resolution of the stenosis in the chronic stage. Hyperintensity around the flow void due to intramural hematoma on T1-weighted MR images was often seen during the second week. In all cases, the findings of MR angiography corresponded to the findings of cerebral angiography. Eight of nine cases showed a good prognosis. In three of the patients with bleeding, in whom the site of the lesion was at the A1 portion, a diffuse thick subarachnoid hemorrhage was present and surgical treatment was required but resulted in a poor prognosis. In the other patients with bleeding, in whom the site of the lesion was at the distal ACA, the prognosis was good and no rebleeding or need for surgical treatment occurred.**

CONCLUSION: **ACA dissection presenting with ischemia has several identifiable neuroradiologic and clinical characteristics, which suggests that it may be classified as a unique clinical entity.**

Spontaneous arterial dissections or dissecting aneurysms in the intracranial cerebral arteries are rather rare and occur most frequently in the territory of the vertebrobasilar circulation (1–12). In the carotid circulation, arterial dissections have been known to involve the internal carotid artery and the middle cerebral artery, causing cerebral ischemia in young patients (13, 14), whereas dissections of the anterior cerebral artery (ACA) had been considered very rare. However, since 1990, 15 cases of ACA dissection have been reported (15–26). This suggests that with a heightened clinical awareness of its presence and the use of advanced diagnostic criteria and procedures for diagnosis, this disease entity can be recognized more often.

An analysis of previous reports of ACA dissection showed some common characteristics: middle-aged patients were most commonly affected, follow-up angiography often showed serial changes in the lesion, and most of the patients had a good prognosis. Therefore, a dissecting aneurysm of the ACA could be categorized as a definable clinical entity. The aim of this study was to analyze the neuroradiologic and clinical features of ACA dissection by using a series of collected cases and to explore the possibility that this condition could be categorized as a single unique clinical entity.

Methods

The cases of patients with nontraumatic ACA dissection were retrospectively collected from the 46 institutes at which they were treated during a 5-year period from 1995 through 1999. The diagnosis of ACA dissection was made by stroke specialists at each institute, based on the following clinical and

Received June 6, 2002; accepted after revision October 18.

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TABLE 1: Clinical characteristics and neuroradiologic findings in 18 cases of anterior cerebral artery dissection

Case No.	Age (yr)/Sex	Presentation at Onset	Site of Lesion	Initial Angiographic Findings	Confirmation of Dissection	Outcome
1	39/F	Bleeding	A1	S + D	Ope	VS
2	66/F	Bleeding	A1	S + D	Ope	VS
3	66/F	Bleeding	A1	S + D	Ope	MD
4	49/F	Bleeding	A2	S	DL†	GR
5	53/M	Bleeding	A3	S	DL	GR
6	43/F	Ischemia	A1–2	S	IMH	GR
7	50/F	Ischemia	A2	S	DL†	GR
8	65/M	Ischemia	A2	S	DL	GR
9	56/M	Ischemia	A2	S + D	DL†	GR
10	67/F	Ischemia	A1–2	S + D	DL†	GR
11	52/F	Ischemia	A2	S + D	IMH	GR
12	36/M	Ischemia	A2	S	IMH	MD
13	56/F	Ischemia	A2	S + D	DL	GR
14	50/F	Ischemia	A2	S	IMH, DL	GR
15	40/M	Combined*	A2	S	IMH, DL	GR
16	64/F	Combined*	A2	S + D	IMH, DL	GR
17	49/F	Combined*	A2	S + D	Ope	GR
18	49/M	Combined*	A2–3	S	DL	GR

Note.—F indicates female; M, male; S + D, stenosis with dilation; S, stenosis without dilation; Ope, discoloration of affected artery around aneurysmal dilation due to intramural hematoma seen during operation; DL, double lumen sign seen on cerebral angiograms; IMH, hyperintensity around signal void due to intramural hematoma seen on T1-weighted MR images; VS, vegetative state; MD, moderate disability; GR, good recovery.

* Four cases showed ischemic symptoms and CT evidence of slight subarachnoid hemorrhage on initial CT scans.

† In 4 cases, the double lumen sign was seen on follow-up angiography.

TABLE 2: Clinical parameters and angiographic findings in cases of anterior cerebral artery dissection according to clinical presentations

	Total	Bleeding Group	Ischemic Group	Combined Group
No. of cases	18	5	9	4
Age (yr) (mean ± SD)	52.8 ± 9.8	54.6 ± 11.6	52.8 ± 9.8	50.5 ± 9.9
Sex ratio (male:female)	6:12	1:3	3:6	2:2
Main site of lesion (n)				
A1	3	3	0	0
A2	14	1	9	4
A3	1	1	0	0
Initial angiographic findings (n)				
Stenosis without dilation	8	2	4	2
Stenosis with dilation	10	3	5	2
Double lumen or intimal flap	7*	1*	3*	3*
Dilation only	0	0	0	0
Prognosis at 1 year after onset (n)				
Good recovery	14	2	8	4
Moderate disability	2	1	1	0
Severe disability	0	0	0	0
Vegetative state	2	2	0	0
Death	0	0	0	0

* Double lumen or intimal flap was seen accompanied by the other angiographic findings.

neuroradiologic criteria: sudden onset of ischemic or hemorrhagic symptoms; cerebral angiographic findings consistent with clinical symptoms; and cerebral angiographic findings characteristic of arterial dissection (3–6), such as double lumen sign (presence of a false lumen or an intimal flap) (1, 2, 7), stenosis with dilation (pearl and string sign) (2, 5, 7, 8), stenosis (string sign or tapered narrowing), and occlusion (7). However, only the double lumen sign was used as a reliable angiographic finding in this study. Cases without the double lumen sign had to have fulfilled at least one of the following additional criteria to be confirmed as arterial dissection: discoloration of affected artery around aneurysmal dilation, considered to be due to intramural hematoma (1, 5, 8), seen during surgery; or hyperintensity around signal intensity void, considered to be due to intramural hematoma (2, 3), seen on T1-weighted MR images.

Patients were interviewed to assess for the preexistence of head trauma. For patients who were confused or unconscious, family members with full knowledge of the medical and general histories of the patients were interviewed. Patients with histories of head trauma within 6 months before onset were excluded.

Three observers independently judged the data of submitted cases based on the above diagnostic criteria. If all three observers agreed, the patient was enrolled in the study. Consequently, the cases of 18 patients who fulfilled the above criteria were collected and the 18 patients were registered for the study.

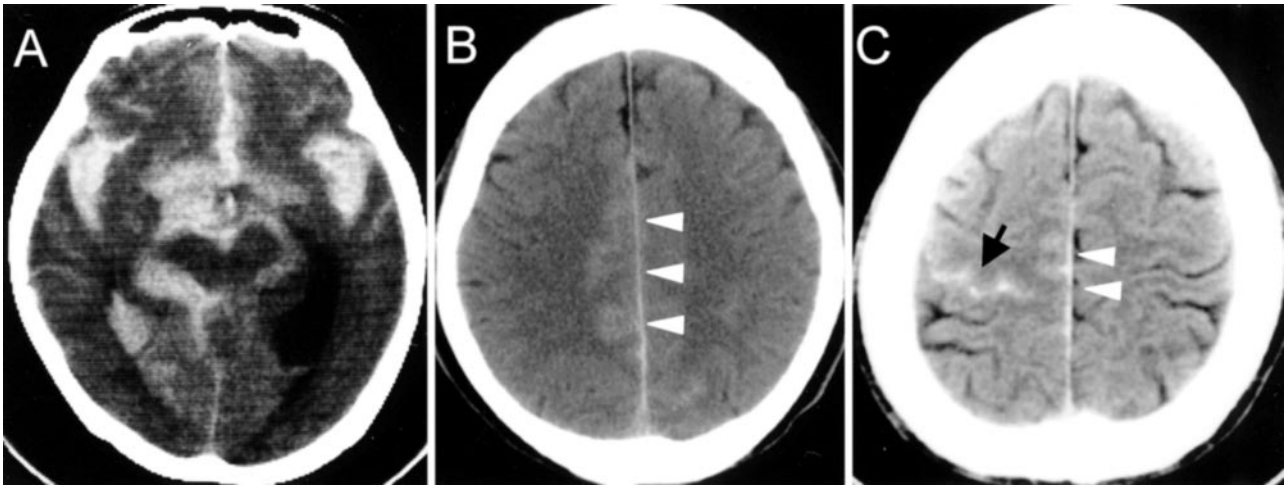

FIG 1. Representative CT findings in patients presenting with SAH.
A, Diffuse thick SAH in the most subarachnoid cisterns due to bleeding from arterial dissection at A1.
B, Thin localized SAH in the interhemispheric fissure (*arrowheads*) due to bleeding from arterial dissection at A3.
C, Very thin SAH in the interhemispheric cistern (*arrowheads*) and in the sulcus of the convexity of the cerebral hemisphere (*arrow*) due to bleeding from arterial dissection at A2 in the combined group.

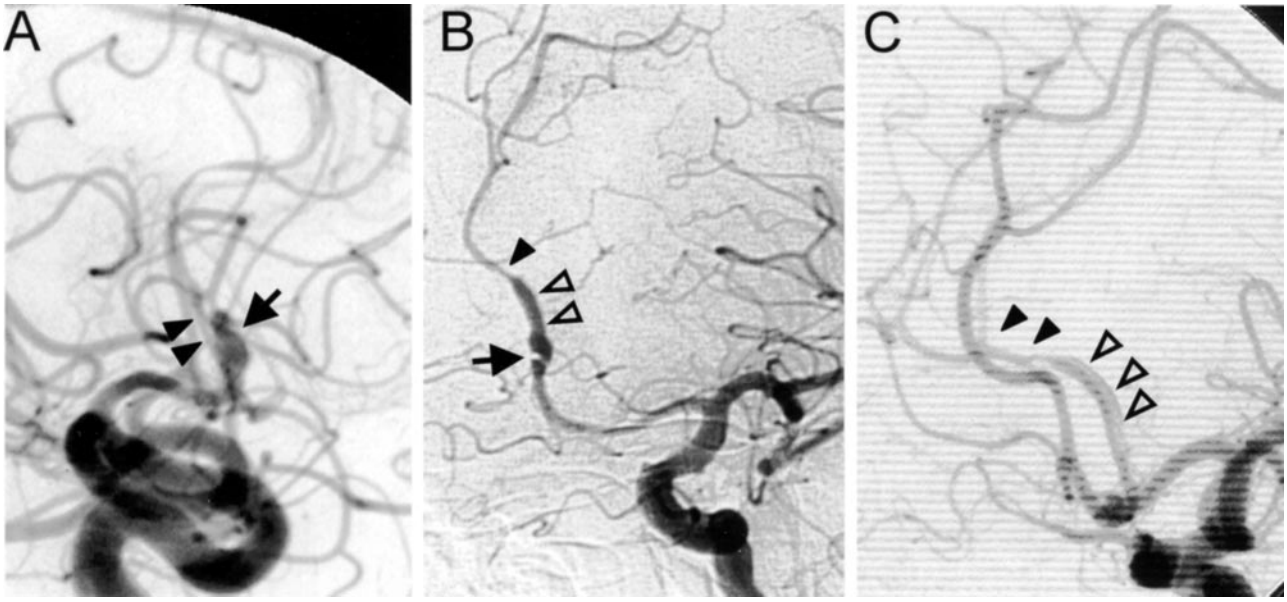

FIG 2. Representative findings of cerebral angiography in the ischemic cases.
A, Lateral view angiogram of the right carotid artery in case 9. Stenosis (*arrowheads*) with dilation (*arrow*) at the A2 portion of the ACA.
B, Oblique view angiogram of the left carotid artery in case 13. Stenosis (*closed arrowhead*) with dilation (*open arrowheads*) at the A2 portion of the ACA accompanied by an intimal flap (*arrow*).
C, Oblique view angiogram of the left carotid artery in case 8. Stenosis (*closed arrowheads*) accompanied by the double lumen sign (*open arrowheads*).

Their ages ranged from 36 to 67 years (mean age, 52.8 years), and 12 of the 18 were female patients.

The following neuroradiologic and clinical parameters were evaluated and compared: site of lesion; type of onset; angiographic findings; other neuroradiologic findings (such as CT, MR imaging, and MR angiographic findings); treatment; clinical course; and prognosis. Plain CT was performed within 24 hr of onset in all cases. Infarction size was evaluated based on plain CT scans obtained ≥4 weeks after onset. Initial MR imaging was performed within 2 weeks after onset in 11 cases. Follow-up MR imaging was performed within 9 months after onset in five cases. One patient underwent follow-up MR imaging twice, for a total of 17 MR imaging studies. At all institutes, MR imaging was performed with 1.5-T MR imaging systems, to obtain spin-echo T1-weighted and T2-weighted images with a 5-mm section thickness. MR angiography was performed in 11 cases within 3 weeks of onset. MR angiography was routinely performed with the time-of-flight technique. Initial cerebral angiography was performed within 18 days after onset in all cases. Follow-up angiography was performed in 10 cases between 13 and 285 days after onset. Five patients underwent follow-up angiography twice. Therefore, cerebral angiography was performed a total of 28 times. Cerebral angiography was performed by using the Seldinger-technique, and digital subtraction angiograms were obtained. Outcome was evaluated at 1 month, 3 months, and 1 year after onset. Using the Glasgow Outcome Scale, the outcome was categorized into five stages: good recovery, moderate disability, severe disabil-

ity, vegetative state, and death. Poor outcome was defined as severe disability, vegetative state, or death based on the Glasgow Outcome Scale.

Results

The clinical parameters of the 18 cases of ACA dissection are shown in Table 1. Neuroradiologic and clinical features were analyzed according to the presentation at onset by dividing the 18 cases into the bleeding group, the ischemic group, and the combined group (Table 2). Four cases from the combined group presented with ischemic symptoms at the time of onset without sudden headache despite very slight subarachnoid hemorrhage (SAH) detected by initial CT.

In the ischemic and combined groups, the admission ischemic symptom was a motor disturbance: paresis of the contralateral lower extremity in six cases, contralateral hemiparesis in four, and paraparesis in two. In all five cases from the bleeding group, the initial symptom was sudden headache, which was accompanied at the time of admission by a disturbance of consciousness in three cases.

In the ischemic and combined groups, the main site of the lesion was the A2 portion on initial angiograms in all cases. In the bleeding group, the site of the lesion was the A1 portion in three cases and the A2 or A3 portion in two cases.

In the bleeding group, CT scans obtained at admission in the three cases with the lesion in the A1 portion showed a diffuse thick layer of SAH in the most subarachnoid cisterns. In the two cases in which the lesion was in the A2 or A3 portion, localized SAH in the interhemispheric cistern with or without intracerebral hematoma was seen on CT scans (Fig 1). In the combined group, CT scans obtained at admission showed a very thin SAH in the interhemispheric cistern or in the sulcus of the convexity of the cerebral hemisphere near the interhemispheric cistern (Fig 1).

In the ischemic and combined groups, the main angiographic finding was stenosis with or without dilation at the A2 portion, which was accompanied by a double lumen or an intimal flap in six cases (Table 2, Fig 2). In the bleeding group, dilation with slight stenosis was seen in the three cases with the A1 lesion (Fig 3), and this was confirmed to be due to arterial dissection during surgery.

Serial changes of the stenotic portion were analyzed by examining 13 follow-up angiograms obtained in seven cases from the ischemic group and three cases from the combined group (Fig 4). All 10 cases showed serial changes of the stenotic portion of the lesion. Although progression of the stenosis was often seen on follow-up angiograms obtained within 1 month after onset, in all cases, the stenosis improved after 2 months (Figs 4 and 5).

MR imaging was performed in eight cases from the ischemic group and in three cases from the combined group. Hyperintensity around the signal intensity void on T1-weighted MR images was seen in six cases (Fig 6), often during the second week after onset (Fig 7). MR angiography was performed in 11 cases from the ischemic and combined groups, and findings compatible with cerebral angiography were obtained for all the cases (Fig 8).

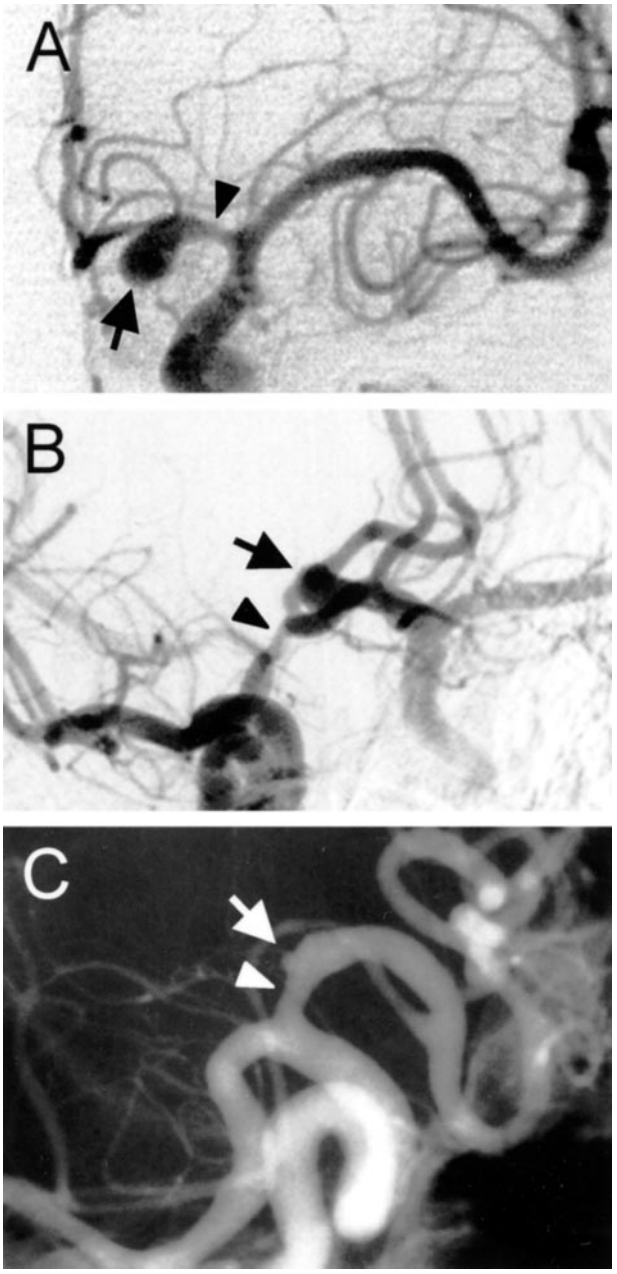

FIG 3. Cerebral angiograms of the cases presenting with bleeding from arterial dissection at A1. Slight stenosis (*arrowhead*) and aneurysmal dilation (*arrow*) are seen in all cases.

A, Anteroposterior view angiogram of the left carotid artery in case 1.

B, Oblique view angiogram of the right carotid artery in case 3.

C, Oblique view angiogram of the right carotid artery in case 2.

In the ischemic and combined groups, the localization of the infarct detected on CT scans or MR images could be divided into three patterns: a parainterhemispheric area in six cases, a watershed area in three, and multiple areas in three (Fig 9). In one case, no infarction was detected on the images. Regarding the distribution of infarction, the supplementary mo-

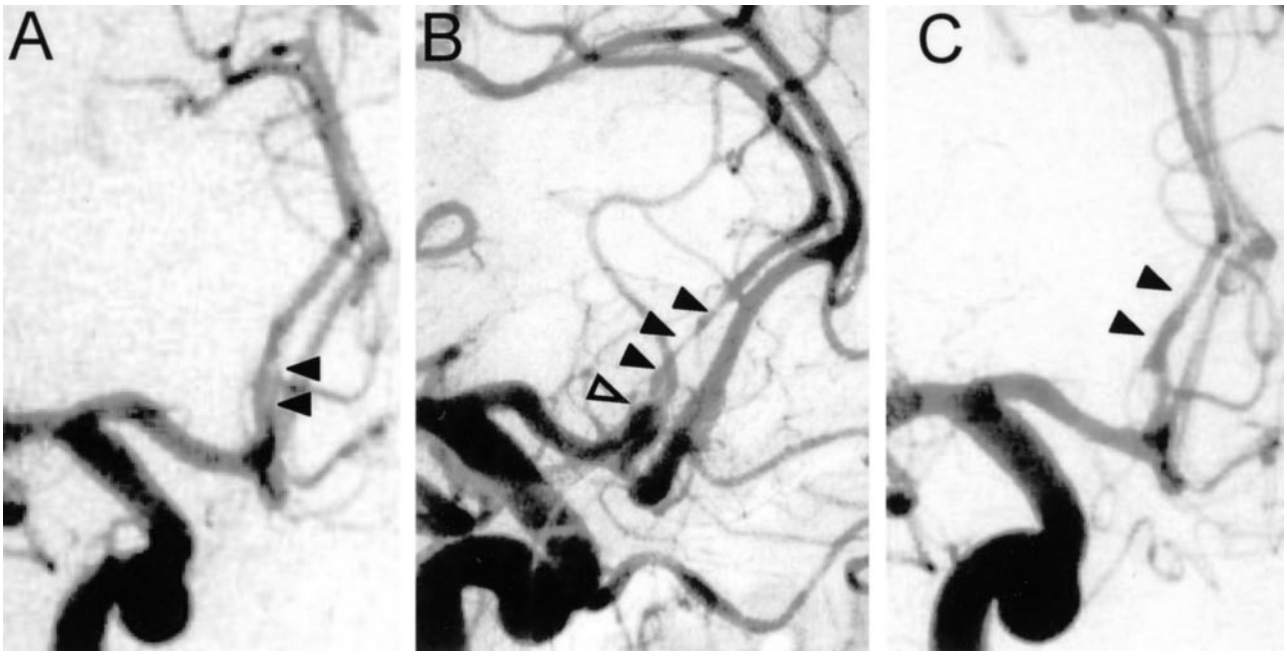

FIG 4. Representative findings of serial changes of the lesion on cerebral angiograms.
A, Oblique view angiogram of the right carotid artery, obtained at admission, shows mild stenosis accompanied by double lumen (*arrowheads*) at A2.
B, Oblique view angiogram of the right carotid artery shows progression to severe stenosis (*closed arrowheads*) with aneurysmal dilation (*open arrowhead*) 2 weeks after onset.
C, Oblique view angiogram of the right carotid artery shows resolution 5 months after onset (*arrowheads*).

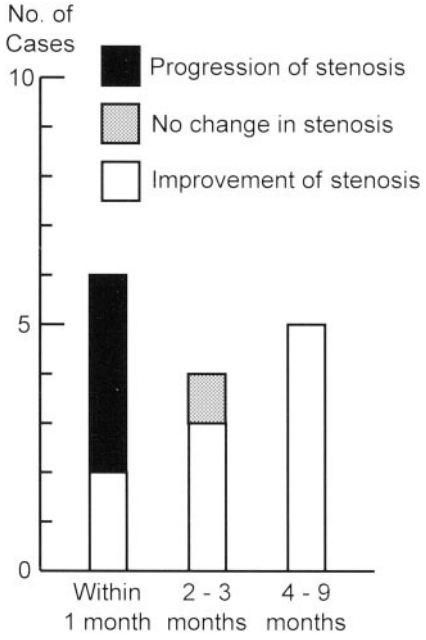

FIG 5. Bar graph shows serial changes of the stenotic portion as seen on cerebral angiograms. The changes were analyzed by using the 15 follow-up angiography studies.

tor cortex was involved in six cases, the motor cortex in four, and the frontal association area in five.

In the ischemic and combined groups, the clinical course corresponded to the serial angiographic findings described above. In three of these cases, the clinical course showed aggravation of ischemic symptoms within 1 month, but all these cases eventually improved. Consequently, this was reflected in the prognosis for these groups. Good recovery was achieved in nine cases at 1 month, 11 cases at 3 months, and 12 cases at 1 year after onset (Tables 1 and 2). Anti-platelet drugs were used in 10 cases. In addition to anti-platelet drugs, anticoagulant or fibrinolytic drugs were used in three cases. However, the prognosis did not seem to be affected by whether a drug was used or which type of drug was used.

In the bleeding group, three cases with the lesion in the A1 portion underwent aneurysm surgery: one case underwent clipping of the aneurysmal bulge, which resulted in a recurrence of SAH, and two cases underwent trapping of the lesion. Of these three cases, two cases developed a vegetative state because of recurrent SAH or cerebral vasospasm. The other two bleeding cases had A2 or A3 lesions and were managed conservatively; in both cases, good recovery was achieved at 1 year after onset without recurrence of bleeding. Of the four cases in the combined group, none showed recurrent bleeding, although operative wrapping of the lesion was performed in one case. In terms of risk factors, hypertension was a factor in eight cases, cigarette smoking in six, and diabetes mellitus in four (Table 2).

Discussion

Since 1990, 15 cases of ACA dissection have been reported in the literature (Table 3) (15–26). The clinical features of the cases that were collected for the present study were analyzed and compared with those of the previously reported cases.

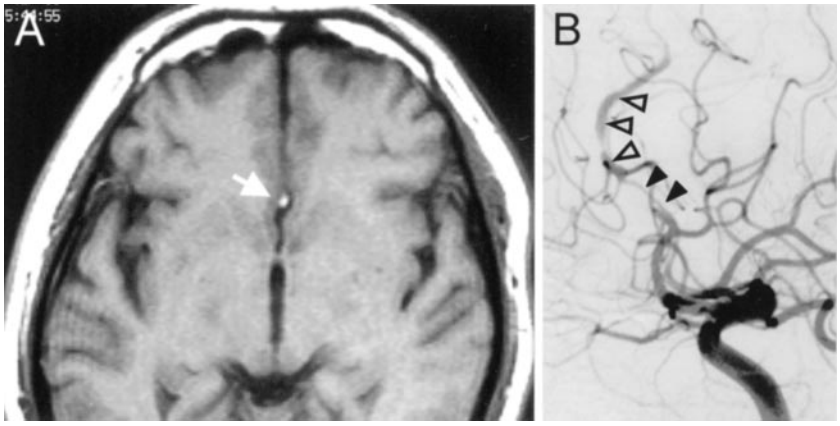

FIG 6. Images from case 12.
A, T1-weighted MR image shows hyperintensity around the signal intensity void.
B, Lateral view angiogram of the left carotid artery, obtained on the same day as the image presented in *A*, shows stenosis (*closed arrowheads*) with dilation (*open arrowheads*).

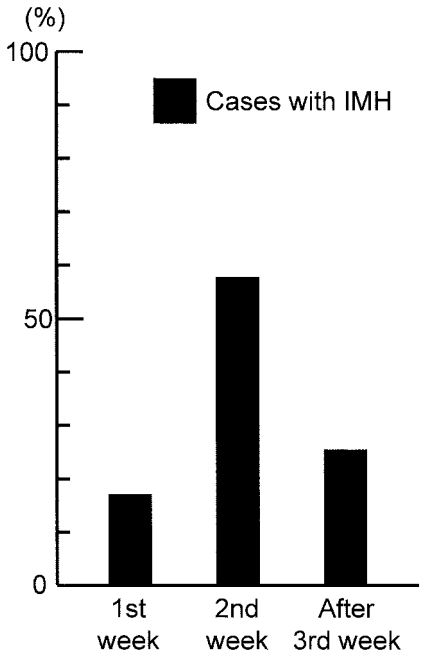

FIG 7. Bar graph shows hyperintensity around the signal intensity void, as seen on T1-weighted MR images, considered to be due to intramural hematoma (*IMH*) based on the timing of the examination.

The analysis showed that this disease frequently occurs in middle-aged persons. The mean patient age in the reported cases was 49.2 ± 10.4 years, which was similar to the patient age range in the present study. However, the sex distribution was different between the reported cases and the cases in the present study; female patients predominated in the present study, whereas male patients predominated in the reported cases. The reason for this difference in sex distribution is unclear. Both in the reported cases and in the present study, ischemia was slightly more dominant than bleeding as a presenting symptom. As risk factors, hypertension was frequently noted and cigarette smoking and diabetes mellitus were sometimes noted. Therefore, increased hemodynamic stress on degenerative arterial walls might have a part in the cause of this disease.

Several further characteristics were documented by angiography (Tables 2 and 3). A2 was the most frequent site of the lesion in the ischemic group, both in this study and in the reported cases. On the other hand, in the bleeding group, the characteristic findings of dissection could be seen at any site of the ACA, both in the reported cases and in the present study. Angiographic findings in the reported cases revealed that ischemic cases often had stenosis with or without dilation and that bleeding cases often had dilation with or without stenosis. However, this study found that any combination of stenosis and dilation could be seen in the ischemic, bleeding, and combined groups.

The most characteristic angiographic findings in the ischemic cases in this study were the serial changes in the lesion often seen on follow-up angiograms. These changes have also been found in cases of vertebrobasilar artery dissection (9, 12) and in reported cases of ACA dissection (15–19, 23–25). However, these articles do not clearly state the time period that elapsed from onset to serial changes. In this study, all 10 patients who underwent follow-up angiography had serial changes shown that could be analyzed according to time from onset. Thus, the present study revealed that stenosis often progressed during the acute stage and resolved during the chronic stage. In addition, stenosis was often accompanied by dilation in the subacute stage. These results indicate the dynamic nature of the pathologic condition (28), with increased narrowing due to intramural hematoma and the formation of an aneurysmal bulge in the acute to subacute stage and then the healing process in the chronic stage. Based on the ischemic symptoms and angiographic findings, making a diagnosis in the ischemic group is not difficult. However, in cases in which diagnosis was not confirmed during the acute stage, serial changes found on follow-up angiograms helped make the diagnosis.

The serial changes in the clinical condition of the ischemic group corresponded to the serial changes seen on angiograms. The conditions in some of the cases in this study deteriorated during the acute stage. However, because all these cases eventually improved, eight of nine cases in the ischemic group showed a good prognosis. This type of clinical course and a good prognosis is confirmed not only by the reported cases of ACA dissection presenting with

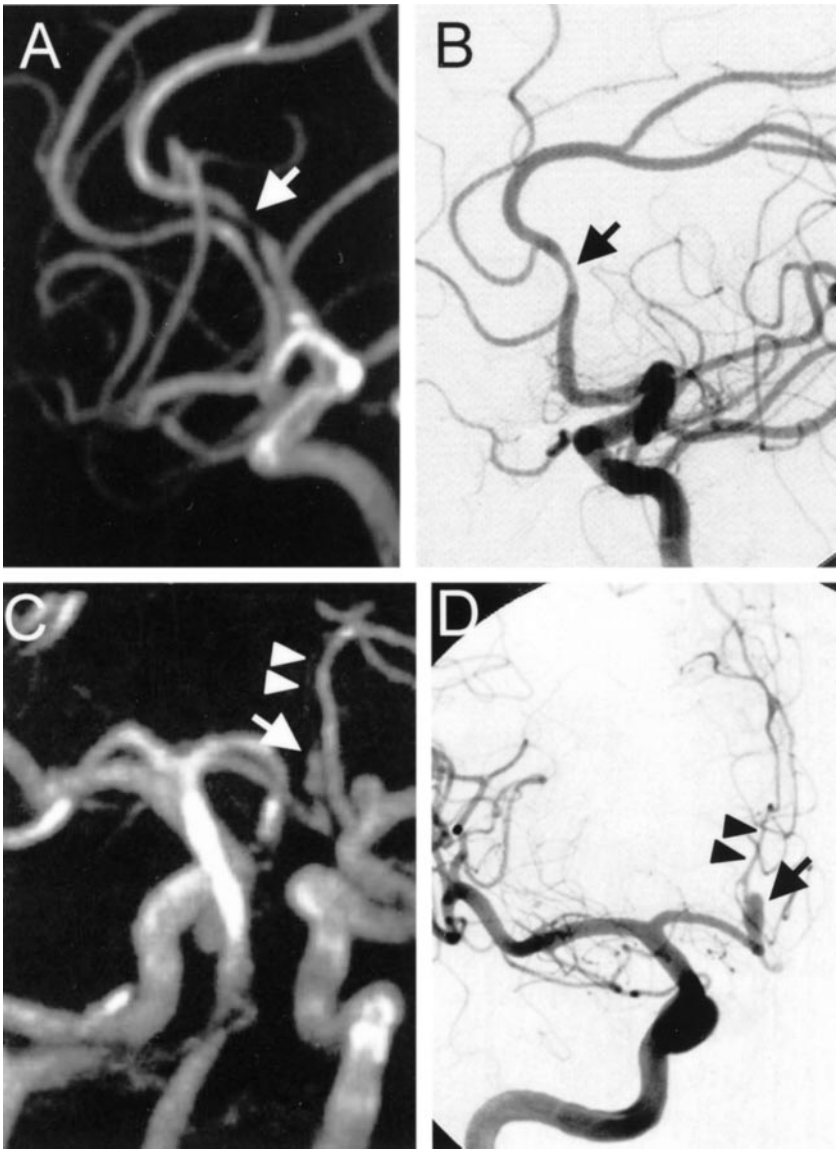

FIG 8. Example comparisons of MR angiograms and cerebral angiograms.

A, MR angiogram from case 7 shows stenosis (*arrow*) in the left ACA.

B, Lateral view angiogram of the left carotid artery shows compatible findings.

C, MR angiogram from case 9 shows stenosis (*arrowheads*) and dilation (*arrow*) in the right ACA.

D, Anteroposterior view angiogram of the right carotid artery shows compatible findings.

ischemia (Table 3) but also by the studies of vertebrobasilar artery dissection presenting with ischemia (9, 12). On the other hand, in cases of cerebral ischemia due to causes other than arterial dissection, clinical deterioration during the acute stage markedly worsens the prognosis (29) and stroke due to large-vessel occlusion or stenosis often shows further deterioration after spontaneous improvement (30). The 1-year prognosis after stroke for all cases of cerebral infarction indicates that only half the patients are functionally independent (31). Thus, cerebral ischemia caused by ACA dissection could be considered a somewhat benign type of stroke. It should, therefore, be treated conservatively with careful follow-up monitoring before contemplating treatments with potentially serious side effects (12). Follow-up monitoring can be performed by MR angiography, because the results of MR angiography correspond to the findings on cerebral angiograms (3, 32). However, hyperintensity around the signal intensity void seen on T1-weighted MR images cannot substitute for cerebral angiography in making a diagnosis, because this study showed that its positive rates are largely affected by the timing of the MR imaging. This was also shown in previous studies of vertebrobasilar dissecting aneurysms (2, 3).

In the bleeding group, the diagnosis was not difficult to make in those cases with A2 or A3 lesions. However, among cases with A1 lesions, one case showed a small aneurysmal bulge and mild stenosis in the A1 lesion. This suggests that a careful inspection of the angiograms may be required in some cases with A1 lesions.

In the bleeding group, three patients each with diffuse SAH due to a dissecting aneurysm at the A1 portion underwent surgical treatment during the acute stage. However, none of the three showed a good prognosis. One of these patients underwent clipping of the aneurysmal bulge, which resulted in postoperative aneurysmal growth and rebleeding. Thus, proximal occlusion or trapping of the parent artery may be a more effective surgical procedure, not only

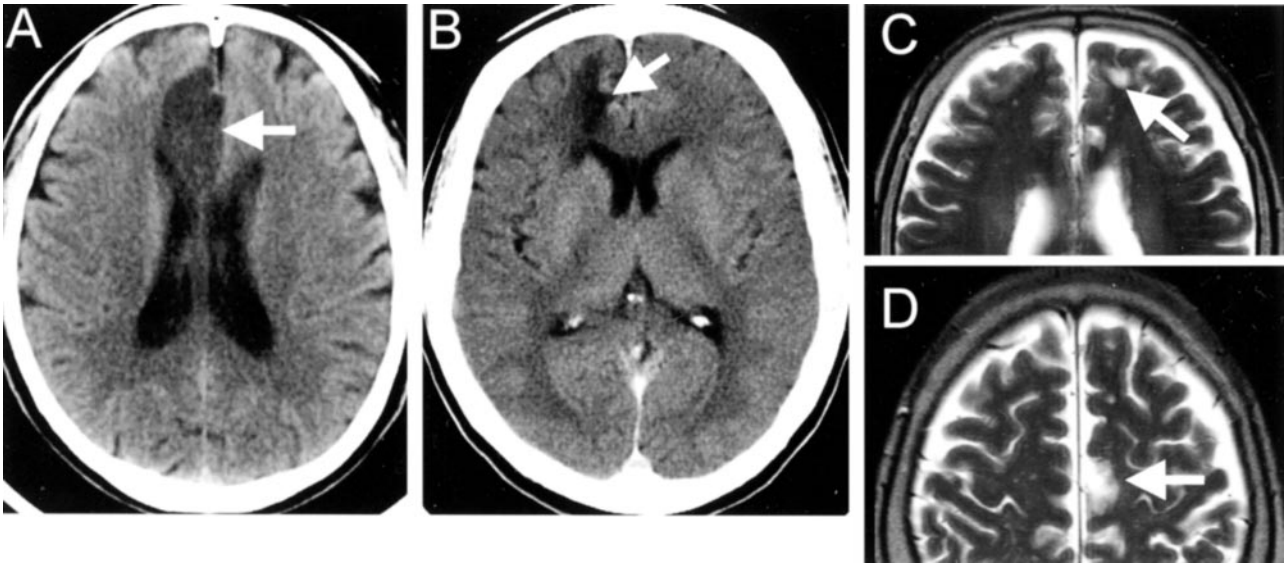

FIG 9. CT scans and MR images show infarction in patients presenting with cerebral ischemia.
A, CT scan shows infarction along the interhemispheric fissure (*arrow*).
B, CT scan shows infarction at the watershed area (*arrow*).
C, MR image shows multiple patchy infarctions in the ACA territory (*arrow*).
D, MR image shows patchy infarctions in the ACA territory (*arrow*).

TABLE 3: Clinical parameters and angiographic findings in reported cases of anterior cerebral artery dissection

	Total	Bleeding Group	Ischemic Group
No. of cases	15	7	8
Age (yr) (mean ± SD)	49.2 ± 10.4	52.6 ± 13.9	46.3 ± 5.3
Sex ratio (male:female)	12:3	5:2	7:1
Site of lesion (n)			
A1	3	2	1
A2	9	2	7
A3, 4	3	3	0
Angiographic findings (n)			
Stenosis without dilation	0	0	0
Stenosis with dilation	8	2	6
Double lumen or intimal flap	5	1	4*
Dilation only	4	4	0
Occlusion	2	0	2
Outcome (n)			
Good recovery	12	5	7
Moderate disability	1	0	1
Severe disability	1	1	0
Vegetative state	0	0	0
Death	1	1	0

* Double lumen or intimal flap was seen in four cases accompanied by the other angiographic findings.

for dissecting aneurysms of the vertebral artery (1, 5, 10) but also for dissecting aneurysms of the ACA. In addition, instead of direct surgical procedures, proximal occlusion, obstruction of the aneurysmal bulge, or stent placement can be achieved by endovascular treatment (33). On the other hand, in the bleeding and combined groups, surgical treatment was performed in only one of six cases with localized SAH at the interhemispheric cisterns due to dissection of the peripheral ACA and no rebleeding was seen. In reported cases, only one of five bleeding cases with the lesion in the peripheral ACA suffered from rebleeding 15 weeks after onset (25). Higher rates of rebleeding were reported in association with SAH due to dissection in the vertebrobasilar circulation (1, 10, 11). Therefore, bleeding from a dissecting aneurysm at the peripheral site of the ACA may be a somewhat benign type of bleeding. Future studies will have to examine the indications for surgical treatment in such cases.

Conclusion

The results of this study show that patients with dissection in the peripheral ACA share several characteristics. They are middle-aged. Fourteen of 15 cases showed a good prognosis because recurrent stroke is less likely. Serial angiographic changes of the lesion are often seen. Dissection in the proximal ACA accompanied by diffuse SAH resulted in a poor prognosis, but its characteristics could not be conclusively determined because of the low number of cases studied. Therefore, although dissection of the peripheral ACA seems to be a unique clinical entity, this will have to be confirmed by further study.

Acknowledgments

We express our gratitude to the members of the following 46 institutes for kind enrollment of cases: Akita University, Iwate Medical University, Tohoku University, Fukushima Medical University, Hirosaki University, Yamagata University, Aomori Rosai Hospital, Hatinohe Municipal Hospital, Towada Municipal Hospital, Kuroishi Municipal Hospital, Noheji Hospital, Iwate Prefectural Central Hospital, Iwate Prefectural Kuji Hospital, Morioka Red Cross Hospital, Iwate Prefectural Miyako Hospital, Iwate Prefectural Iwai Hoswpital, Izumi Hospital Iwate, National Miyagi Hospital, Iwate Prefectural Fukuoka Hospital, National Iwate Hospital, Iwate Prefectural

Daitoh Hospital, Higashi Hatimantai Hospital, Heiwadai Hospital, National Sendai Hospital, Kohnan Hospital, Izumi Hospital Sendai, National Miyagi Hospital, Ishinomaki Red Cross Hospital, Senseki Hospital, Miyagi Prefectural Cancer Research Center, Sendai Tokusukai Hospital, Tohoku Koseinenkin Hospital, Iwaki Kyoritu Hospital, Masukinen Hospital, Watari Hospital, Haramachi Municipal Hospital, Fukushima Medical Clinic, Takeda General Hospital, Shirakawa Kosei Hospital, Yamagata Prefectural Central Hospital, Yamagata Municipal Saiseikan Hospital, Yonezawa Municipal Hospital, Sakata Municipal Hospital, Yamagata Prefectural Kahoku Hospital, Akita Prefectural Cerebrovascular Research Center, Akita Red Cross Hospital.

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