

Traumatic and spontaneous extracranial internal carotid artery dissections*

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Summary. Seventy patients with spontaneous and 21 with traumatic extracranial internal carotid artery dissections were studied clinically and angiographically with mean follow-ups of 64 (spontaneous group) and 40 months (traumatic group). Sixty percent of the patients in the spontaneous group and 71% in the traumatic group also had follow-up angiograms. In traumatic dissections aneurysms were common, significantly fewer aneurysms resolved or became smaller and fewer stenoses resolved or improved, whereas more stenoses progressed to occlusion. Traumatic dissections were more likely to leave the patients with neurological deficits. A significantly higher percentage of the patients with spontaneous dissections were asymptomatic at follow-up compared with the traumatic group. Although both spontaneous and traumatic dissections of extracranial internal carotid arteries mostly carry a good prognosis, the outcome may be somewhat less favorable for the traumatic group.

Key words: Carotid dissection – Spontaneous carotid dissection – Traumatic carotid dissection – Carotid artery trauma

Introduction

In the past two decades there has been an increasing familiarity with the clinical and the angiographic features of spontaneous and traumatic dissections of the extracranial internal carotid arteries (ICAs) [3, 8–10, 12–19, 21, 22, 24].

These dissections are uncommon but are not rare. They occur when the circulating blood penetrates the arterial wall, usually splitting the media and creating a false lumen that dissects the arterial wall for varying distances [2]. If the intramural hematoma expands toward the adventitia, an aneurysmal dilatation may form (a "dissecting aneurysm"). Some dissections occur after a definite head or neck injury (traumatic dissections), and others occur without history of definite trauma (spontaneous dissections), although at times there may be history of minimal or trivial trauma. Practically, there seems to be a difference between the extracranial ICA dissections caused by definite severe head and neck trauma and those occurring spontaneously, with or without a history of minimal or trivial trauma [13].

This study was conducted to compare the clinical and angiographic features as well as the outcome of these two groups.

Materials and methods

The records of patients with a diagnosis of spontaneous or traumatic subintimal dissection, dissecting aneurysm, or dissection of an ICA, seen at Mayo Clinic prior to 1985, were reviewed. Twenty-one patients with traumatic extracranial ICA dissections (15 men, 6 women) and 70 patients with spontaneous ICA dissections (29 men, 41 women) were identified. Age and duration of clinical and angiographic follow-up of these patients are summarized in Table 1. The follow-up evaluation included one or more neurologic examinations in 79 patients (including all of the patients with traumatic ICA dissections), telephone calls in 75, and follow-up letters in 15. Information regarding persistence or resolution of symptoms and signs, resumption of normal activities, and return to work were recorded. On follow-up arteriography, 22 ICAs of the traumatic group and 52 ICAs of the spontaneous group had been re-studied. If a patient had undergone resection of the extracranial ICA dissecting aneurysm, the specimens were subjected to pathology studies.

On initial evaluation, all of the patients had undergone conventional arteriography. Of the 15 patients with traumatic dissections who had follow-up angiograms, this consisted of intravenous (i.v.) digital subtraction angiography (DSA) in 2 patients and transfemoral conventional angiography in 13 patients (2 of whom had also undergone DSA). The interval from the initial to the last angiogram was 2 weeks to 3.5 years. Initial angiograms were done within hours of the accident in 11 cases, within days in 4 cases, within months in 2 cases, and within years in 4 cases (in these delayed cases neurologic manifestations had developed long after the

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 Table 1. Patients with traumatic or spontaneous extracranial internal carotid artery (ICA) dissections

Data	Patient group			
	Spontaneous	Traumatic		
No. of patients	70 (29 M, 41 F)	21 (15 M, 6 F)		
Patient age (years)				
Range	18–76	19–55		
Mean	41	32		
Duration of follow-up				
Range	22 months-9.5 years	4 days-7.5 years		
Mean	64 months	40 months ^a		
No. of patients with follow-up angio-				
graphy (%)	42 (60)	15 (71)		

^a Excluding two patients who died 4 and 6 days after the accident as the result of massive cerebral infarct and edema

accident as the result of embolization from thrombi within the residual dissecting aneurysms). Of the 42 patients with spontaneous dissections who underwent follow-up angiography, this was a conventional arteriogram in 29 and i.v. DSA in 13. These had been carried out within 1 month to 6.5 years after the initial angiogram. The initial angiogram had been done within a few days after the onset of the symptoms in 62 cases and within 1 month in all but 3 cases.

The χ^2 test and Fisher's exact test were used to evaluate the data.

Results

Clinical findings

Spontaneous group. Headache, the most common symptom (Table 2), was often unilateral (73%), and it was usually focal and located in the orbital, periorbital, and frontal regions. The other common locations were mastoid region and deep in the ear, temporal region, angle of the mandible, face, and finally occipital region. Focal cerebral ischemic symptoms [transient ischemic attack (TIA) and stroke] usually were delayed and followed a headache by a period ranging from a few minutes to 2 weeks. Although the clinical picture varied and some of the patients presented with only focal ischemic events (TIA, stroke, amaurosis fugax), or lower cranial nerve palsies, or headache and bruits, many presented with one of the following distinct syndromes: hemicrania and oculosympathetic palsy (OSP) (12 patients), hemicrania and delayed focal cerebral ischemic events (20 patients), and combination of hemicrania, OSP, and focal cerebral ischemic events (15 patients). Forty percent of the patients were hypertensive and 16% had angiographic evidence of fibromuscular dysplasia in carotid, vertebral, or renal arteries.

Traumatic group. Focal cerebral ischemic symptoms were the most common of the clinical manifestations and often occurred at some time after the accident, sometimes months or years (see the footnote to Table 2). These de**Table 2.** Frequency of symptoms and signs in 70 patients with spontaneous and 21 patients with traumatic dissections of extracranial ICA

Symptom or sign	Patient group					
	Sponta	Traumatic				
	No.	%	No.	%		
Headache	59	84	8	38		
Focal cerebral ischemic symptoms TIAs	43 24	61	15ª 2	71		
Stroke	13		11			
TIAS and stroke	6		2			
Oculosympathetic paresis	37	53	7	33		
Bruits (subjective, objective, or both)	32	46	8	38		
Neck pain	16	23	1^{b}			
Light-headedness	15	21				
Syncope	8		2			
Amaurosis fugax	8		1			
Scalp tenderness	5					
Neck swelling	3					
Dysgeusia	2					
Lower cranial nerve palsies	2					
Asymptomatic ^c	1					
Sensation of pulsation in neck	1		1			

^a Often delayed after the accident: within 30 min-12 h, 6; within

4-20 days, 3; in 2 months, 1; after 6-10 years, 5

^b Excluding any neck pain in connection with the original trauma

^c Symptoms related to a concomitant vertebral artery dissection

TIA, Transient ischemic attack

layed ischemic symptoms were caused by embolization from thrombi within the residual post-traumatic dissecting aneurysms as demonstrated angiographically or by pathologic examination of the excised dissecting aneurysms (Fig. 1). All patients had suffered moderate to marked blunt trauma, with motor vehicle accident as the leading cause (62%). All but 3 patients had suffered other injury in addition to the ICA injury.

Angiographic findings

These abnormalities (Table 3) have been previously described by us and by others [5, 8, 10-14, 16, 19, 20]. The luminal stenosis was frequently irregular and often elongated and tapered. Aneurysms were saccular or were elongated, ovoid, or finger-like. The upper third of the extracranial ICA and subcranial region was the most common site, but the aneurysms also were seen frequently in the mid-third of the ICA and with a lesser frequency in the lower third of the ICA. Occlusions were often 2-3 cm distal to the carotid bifurcation and were usually tapered to a point with a flame-like configuration. ICA dissection was bilateral in 20 patients (29%) in the spontaneous group and in 8 patients (38%) in the traumatic group. Concomitant vertebral artery dissection was noted in 3 patients in the spontaneous group and in 2 patients in the traumatic group.





Fig. 1. a Right carotid arteriogram demonstrates an elongated dissecting aneurysm (post-traumatic in this case). Contrast-filled true lumen is irregular due to filling of false lumen by thrombus. Also noted is a long intimal flap (*arrows*). **b** Cross section of a segment of the lesion demonstrates section through the internal carotid artery (ICA) and the related dissecting aneurysm. Note that the aneurysm is formed within the media. The external elastic lamina (*thin arrows*) surrounds both the true lumen of the ICA (*asterisk*) and the thrombus-filled false lumen of the aneurysm (*FL*). The false lumen has compressed the true lumen (*thick arrows*)

Table 3. Angiographic findings in 90 ICAs (of 70 patients) with spontaneous dissection and 29 ICAs (of 21 patients) with traumatic dissection

Angiographic findings	Patient group					
	Spont	Traumatic				
	No.	%	No.	%		
Luminal stenosis	69	77	13	45		
Aneurysm	35	39	17	59		
Intimal flaps	25	28	7	24		
Slow ICA-MCA flow	22	24	3	10		
Occlusion	15	17	8	28		
Distal branch occlusions (emboli)	11	12	3	10		

MCA, Middle cerebral artery

Follow-up

Clinical follow-up. Of the spontaneous group, 76% were asymptomatic, 1 died of unrelated cause (colon carcinoma), and only 4 patients had marked or moderate deficits (Table 4). Mild deficits or residual recurrent head-aches were present in 12% of the patients. The head-aches were severe in 2 patients. Overall, 59 patients (85%) made complete or excellent recoveries. Of the traumatic group, 2 died of massive cerebral infarct and edema and 4 had moderate or marked deficits. Overall, 71% had made an excellent or a complete recovery, al-

though only 23% of the patients were completely asymptomatic.

Angiographic follow-up. In the spontaneous group, 52 dissected ICAs (of 42 patients) were restudied. Of 20 aneurysms noted in the initial angiograms, 6 remained unchanged, and 12 either decreased in size or resolved (obliterated). Of the 40 stenoses, more than 87% either completely or partially resolved. Of 7 occlusions, only 1 recanalized, and the rest remained unchanged.

Twenty-two dissected ICAs (of 15 patients) in the traumatic group were restudied angiographically. Only 3 of the 14 aneurysms noted in the initial angiogram decreased in size or resolved. Also, only 6 of the 11 stenoses completely or partially resolved, and 3 stenoses progressed to occlusion. None of the 5 occlusions noted initially recanalized.

Treatment

Different treatment modalities had been used (Table 5) depending upon the clinical manifestations, judgment of the clinicians involved in the case, and the amount of information available at the time regarding the natural history of the disease. In the spontaneous group, about one-third received no specific treatment. Seven patients had received corticosteroids prior to their referral to us with the impression of "arteritis" on the basis of angiographic findings of an irregular elongated segment of ICA. This treatment was terminated in all 7. A consider-

Table 4. Follow-up in spontaneous and traumatic dissections of ICA

	Patient group					
	Spontaneous		Traumatic			
	No.		%	No		%
Clinical follow-up						
Died	0		0	2		10
Marked or moderate deficits	4		6	4		19
Mild deficits ^a	7		10	6		29
Mild or no deficits but residual headaches	5		7	4		19
Asymptomatic	53		76	5		24
Died of unrelated cause	1			0		
Angiographic follow-up	(52 ICAs of 42 patients)		(22 15	(22 ICAs of 15 patients)		
Aneurysm	20			14		
Eliminated by resection		0			4	29
Unchanged		6	30		5	36
Decreased in size		8	40		2	14
Resolved		4	20		1	7
Not visualized ^b		2			2	
Stenosis	40			11		
Resolution, complete	2	23	58		4	36
Resolution, partial	1	2	30		2	18
Progression ^c		2	5		1	9
Progression to occlusion		2	5		3	27
Elimination by resection		0			1	9
Unchanged		1			0	
Occlusion	7			5		
Recanalization		1			0	
No change		6			5	

^a Not interfering with work or daily activities (i.e., slight paresis, mild sensory deficits, reflex asymmetries, slight dyscalculia, oculosympathetic palsy, bruits)

^b As the result of ligation of parent vessel in one case and as the result of progression of stenosis to occlusion in the other cases ^c With additional appearance of a small aneurysm in the midseg-

ment of extracranial ICA

ably larger proportion of the traumatic group had undergone operation, mostly consisting of resection of the residual aneurysm (considered to be the source of embolization) (Fig. 1). We now rarely operate on a patient with spontaneous ICA dissection. The indications and the rationale for surgical treatment in the traumatic group have been discussed in a previous publication [19].

Discussion

The clinical and angiographic features, as well as the outcome of spontaneous and traumatic ICA dissections, have been discussed in many papers [4, 10, 11, 13, 14, 18, 19, 24]. Traumatic dissections of the ICA resulting from blunt injuries are less common. Their clinical manifestations vary. In severe traumas, especially when the

 Table 5. Treatment modalities implemented in 70 patients with spontaneous and 21 patients with traumatic ICA dissections

Treatment modality	No. of patients ^a			
	Spontaneous	Traumatic		
Supportive only	22	3		
Anticoagulant therapy	18	6		
Antiplatelet therapy	40	9		
Surgery	9 ^b	10°		

Some patients received more than one of these modalities

^b Superficial temporal artery (STA)-middle cerebral artery (MCA) bypass in 8 patients, ICA exploration in 1 patient

^c Resection and interposition graft in 5 patients, STA-MCA bypass ICA ligation in 1 patient, removal of MCA embolus and STA-MCA bypass in 1 patient

patient is initially comatose or has suffered multiple organ injuries, traumatic ICA dissections may go undiagnosed. Patients who survive these severe traumas may have no neurologic deficits related to their undiagnosed ICA dissection or may have deficits that are often interpreted as related to "head injury" and thus angiography is not carried out. Some of these patients have residual traumatic dissecting aneurysms that may become symptomatic even years later by causing focal cerebral ischemic symptoms as the result of embolization from a thrombus within the aneurysm [19].

Spontaneous ICA dissections also may sometimes go undiagnosed because at times they may cause only mild or trivial symptoms or be entirely asymptomatic (for example, diagnosed incidentally during work-up of a patient with spontaneous vertebral artery dissection or the opposite ICA dissection). Symptoms delayed for years in spontaneous dissections, however, have been most unusual in our experience. Among more than 100 cases of spontaneous ICA dissections seen by us, only 1 patient presented with symptoms due to embolization from a thrombus within a dissecting aneurysm, which was thought to have existed for years as the result of an undiagnosed spontaneous dissection (B. Mokri, unpublished data).

Clinically, in the traumatic group the most common picture was appearance of focal cerebral ischemic manifestations (TIAs or strokes) delayed from the accident for periods ranging from hours to years (Table 2). The most common clinical pictures in the spontaneous group were hemicrania and Horner's syndrome (17%), or hemicrania and delayed focal cerebral ischemic manifestations (29%), or a combination of both (21%). Major clinical and angiographic features of the traumatic and spontaneous groups are compared in Table 6 and listed in decreasing order of statistical significance. However, it should be pointed out that, from the statistical point of view, the numbers, particularly of the traumatic group, are not large. It is possible that larger numbers could establish more or other significant differences. Cerebral ischemic events appeared to be somewhat more common in the traumatic group, but not significantly so. Headaches (often unilateral) were significantly more common

Table 6. Major	clinical and	angiographic f	eatures a	nd follow-up of
traumatic and s	pontaneous	extracranial IC	CA dissec	tions

Feature	Patients or ICAs, % (no. involved/total no.)					
	Spontaneous	Traumatic	Р			
Death	0 (0/70)	10 (2/21)				
Headaches	84 (59/70)	38 (8/21)	$< 0.001^{a}$			
Asymptomatic at follow-up	76 (53/70)	24 (5/21)	$< 0.001^{a}$			
Stenosis	77 (69/90)	45 (13/29)	0.001^{a}			
Decrease in size or resolution of aneurysm at follow-up	60 (12/20)	21 (3/14)	0.026 ^a			
Partial or complete resolution of stenosis at follow-up	88 (35/40)	55 (6/11)	0.027 ^b			
Progression of stenosis to occlusion	4 (2/50)	27 (3/11)	0.061 ^b			
Aneurysm	39 (35/90)	59 (17/29)	0.062 ^a			
Marked or moderate deficit at follow-up	6 (4/70)	19 (4/21)	0.079 ^b			
Oculosympathetic palsy	53 (37/70)	33 (7/21)	0.116^{a}			
Occlusion	17 (15/90)	28 (8/29)	0.195 ^a			
Residual recurrent hemicrania at follow-up	7 (5/70)	19 (4/21)	0.203 ^b			
Concomitant dissection of vertebral artery	4 (3/70)	10 (2/21)	0.326 ^b			
Focal cerebral ischemic events	61 (43/70)	71 (15/21)	0.403 ^a			
Bilaterality	29 (20/70)	38 (8/21)	0.407^{a}			
Bruits	46 (32/70)	38 (8/21)	0.537ª			

^a χ^2 test

^b Fisher's exact test (two-tail)

in the spontaneous group. However, in about one-fourth of the patients in the traumatic group, cerebral ischemic symptoms that led to the detection of ICA dissection appeared months or years after the accident. Therefore, an initial headache or neck pain might have gone unreported or have been attributed to the direct effect of trauma.

Oculosympathetic palsy was somewhat more common in the spontaneous group. However, because of delayed diagnosis in many cases of the traumatic group, it is likely the oculosympathetic palsy might have resolved in some of the patients during the delay interval. Resolution of oculosympathetic palsy has been documented in about one-third of ICA dissections [18].

Angiographically, occlusions and particularly aneurysms appeared to be more common in the traumatic group, although with the available numbers this reached borderline statistical significance only for the aneurysms. The stenoses were significantly more common in the spontaneous group. Again, since the diagnosis of dissection in about one-fourth of the traumatic group was made years after the accident, it is possible that in some of the patients the stenosis might have resolved in the interval, leaving the residual aneurysm as the only angiographic finding. Besides, occlusions were more common in the traumatic group, and this higher incidence of occlusion would subtract from the incidence of other angiographic abnormalities, including the stenosis.

Most of the patients with traumatic ICA dissections who survive the initial injury do well. Overall, more than 70% made excellent or complete clinical recoveries. Fifty-five percent of the stenoses and 20% of the aneurysms partially or completely resolved.

There was a somewhat higher incidence of residual marked or moderate deficits in the traumatic group. The incidence of residual hemicrania appeared to be higher in the traumatic group, but not significantly. A significantly higher percentage of the patients in the spontaneous group were asymptomatic at follow-up (P < 0.001).

Angiographically, in the spontaneous group a higher percentage of aneurysms decreased in size or disappeared, and significantly more stenoses partially or completely resolved. Progression of stenosis to occlusion was more common in the traumatic group. There were two deaths in the traumatic group; no dissection-related deaths were noted in the spontaneous group. Deaths in connection with the spontaneous ICA dissections, however, have been reported [1, 6, 7, 23].

Because we saw fewer traumatic dissections and some of them were delayed cases rather than early and acute cases, and particularly if the prognosis at least in terms of mortality is more guarded in the early stages of the disease, then the relative risk of impairment and death related to traumatic dissection may even have been underestimated in this study.

In conclusion, compared with spontaneous dissections of the ICAs, traumatic dissections are more likely to leave the patients with significant neurologic deficits. In spontaneous dissections, a significantly higher proportion of the patients were asymptomatic at follow-up. Angiographically, aneurysms are more frequent in traumatic dissections, significantly fewer aneurysms resolve or become smaller, and fewer stenoses resolve or improve, whereas more stenoses progress to occlusion. Overall, although dissections of the extracranial ICAs mostly carry a good prognosis, the prognosis may be somewhat less favorable for the traumatic group compared with the spontaneous group.

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References

- Anderson RM, Schechter MM (1959) A case of spontaneous dissecting aneurysm of the internal carotid artery. J Neurol Neurosurg Psychiatry 22:195–201
- Anderson WAD, Scotti TM (1980) Synopsis of pathology, 10th edn. Mosby, St. Louis, pp 290–293
- 3. Batzdorf U, Bentson JR. Machleder HI (1979) Blunt trauma to the high cervical carotid artery. Neurosurgery 5:195–201
- 4. Bogousslavsky J, Despland P-A, Regli F (1987) Spontaneous carotid dissection with acute stroke. Arch Neurol 44:137–140
- 5. Bradac GB, Kaernbach A, Bolk-Weischedel D, Finck GA (1981) Spontaneous dissecting aneurysm of cervical cerebral arteries: report of six cases and review of the literature. Neuro-radiology 21:149–154

- Brice JG, Crompton MR (1964) Spontaneous dissecting aneurysms of the cervical internal carotid artery. Br Med J 2:790– 792
- Brown OL, Armitage JL (1973) Spontaneous dissecting aneurysms of the cervical internal carotid artery: two case reports and a survey of the literature. Am J Roentgenol 118:648–653
- Davis JM, Zimmerman RA (1983) Injury of the carotid and vertebral arteries. Neuroradiology 25:55–69
- Dragon R, Saranchak H, Lakin P, Strauch G (1981) Blunt injuries to the carotid and vertebral arteries. Am J Surg 141: 497-500
- Ehrenfeld WK, Wylie EJ (1976) Spontaneous dissection of the internal carotid artery. Arch Surg 111:1294–1301
- Fisher CM, Ojemann RG, Roberson GH (1978) Spontaneous dissection of cervico-cerebral arteries. Can J Neurol Sci 5:9– 19
- Friedman WA, Day AL, Quisling RG, Sypert GW, Rhoton AL Jr (1980) Cervical carotid dissecting aneurysms. Neurosurgery 7:207-214
- Hart RG, Easton JD (1983) Dissections of cervical and cerebral arteries. Neurol Clin 1:155–182
- Houser OW, Mokri B, Sundt TM Jr, Baker HL Jr, Reese DF (1984) Spontaneous cervical cephalic arterial dissection and its residuum: angiographic spectrum. AJNR 5:27-34
- Krajewski LP, Hertzer NR (1980) Blunt carotid artery trauma: report of two cases and review of the literature. Ann Surg 191: 341–346
- Mokri B (1987) Dissections of cervical and cephalic arteries. In: Sundt TM Jr (ed) Occlusive cerebrovascular disease: diag-

nosis and surgical management. Saunders, Philadelphia, pp $38{-}59$

- Mokri B, Piepgras DG, Sundt TM Jr, Pearson BW (1982) Extracranial internal carotid artery aneurysms. Mayo Clin Proc 57:310-321
- Mokri B, Sundt TM Jr, Houser OW, Piepgras DG (1986) Spontaneous dissection of the cervical internal carotid artery. Ann Neurol 19:126–138
- Mokri B, Piepgras DG, Houser OW (1988) Traumatic dissections of the extracranial internal carotid artery. J Neurosurg 68:189-197
- Momose KJ, New PFJ (1973) Non-atheromatous stenosis and occlusion of the internal carotid artery and its main branches. Am J Roentgenol 118:550-566
- Ojemann RG, Fisher CM, Rich JC (1972) Spontaneous dissecting aneurysm of the internal carotid artery. Stroke 3:434– 440
- 22. Stringer WL, Kelly DL Jr (1980) Traumatic dissection of the extracranial internal carotid artery. Neurosurgery 6:123-130
- 23. Thapedi IM, Ashenhurst EM, Rozdilsky B (1970) Spontaneous dissecting aneurysm of the internal carotid artery in the neck: report of a case and review of the literature. Arch Neurol 23:549-554
- Zelenock GB, Kazmers A, Whitehouse WM Jr, Graham LM, Erlandson EE, Cronenwett JL, Lindenauer SM, Stanley JC (1982) Extracranial internal carotid artery dissections: noniatrogenic traumatic lesions. Arch Surg 117:425–432