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BACKGROUND AND PURPOSE: **An effective intervention has not yet been established for patients with acute ischemic stroke who present with serious neurologic symptoms due to occlusion or a high-grade stenosis of the internal carotid artery (ICA). The aim of our retrospective study was to investigate the feasibility, safety, and efficacy of emergency carotid artery stent placement to improve neurologic symptoms and clinical outcome.**

METHODS: **Of 896 consecutive patients with acute ischemic stroke who were admitted to our institution within 7 days of onset from July 2000 to June 2003, 17 patients (1.9%) with occlusion or a high-grade stenosis of the ICA underwent emergency carotid artery stent placement. We reviewed their records for neurologic outcome, per the National Institutes of Health Stroke Scale (NIHSS) score, before and at 7 days after stent placement; clinical outcome, per the modified Rankin Scale score (mRS), at 90 days; frequency of procedure-related complications within 30 days; and recurrence rate of ipsilateral ischemic stroke within 90 days.**

RESULTS: **Carotid lesions were dilated completely in all patients. Median NIHSS scores before emergency stent placement and at 7 days were 12 and 5, respectively, showing significant improvement ($P < .01$, Wilcoxon rank sum test). Ten patients (59%) had favorable outcomes (mRS score 0–1) at 90 days. Irreversible complications occurred in two patients (12%): distal embolism in one and intracerebral hemorrhage in the other. No ipsilateral ischemic stroke recurred.**

CONCLUSION: **Emergency carotid artery stent placement can improve the 7-day neurologic outcome and may improve the 90-day clinical outcome in selected patients with ischemic stroke.**

For patients with acute ischemic stroke who present with serious neurologic symptoms on admission or continue to deteriorate neurologically due to total occlusion or a high-grade stenosis of the internal carotid artery (ICA) despite maximal medical treatment, an effective intervention to improve their neurologic symptoms and clinical outcome has not yet been established. Although carotid endarterectomy (CEA) has proved to be beneficial in the prevention of stroke recurrence in patients with a high-grade stenosis of the ICA (1, 2), there have been few studies of emergency CEA to improve their neurologic symptoms (3–7) or no rigorous clinical trials regarding the efficacy of CEA in an acute stroke stage. Some neurosurgeons have tried to perform CEA for a critical stenosis of the ICA in patients with acute stroke (8), since there is a 4.9–12.1% risk of recurrent stroke if one waits 4–6 weeks for therapy (9–12). However, emergency CEA carries a risk that decreases cerebral blood flow (CBF) in the cerebral hemisphere during the operation and may render the affected hemisphere more vulnerable to ischemia. Previous studies (13, 14) have supported carotid artery stent placement as a technique of secondary prevention in the chronic stroke stage. Endovascular specialists are becoming familiar with this stent placement procedure, which may be safe and effective even in the acute stage of ischemic stroke (15–18). As emergency carotid artery stent placement is expected to reopen a carotid occlusion or a critical stenosis with rare reduction in CBF in the affected hemisphere during the procedure, we have performed this emergency procedure in patients with acute stroke to improve their neurologic symptoms and clinical outcome.

The purpose of our retrospective study was to investigate the feasibility, safety, and efficacy of emergency carotid artery stent placement.

Methods

Patient Population

Of 896 consecutive patients with acute ischemic stroke who were admitted to our institution within 7 days of onset from July 2000 to June 2003, 17 (1.9%) who fulfilled our inclusion

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From the Department of Stroke Treatment (K.I., T.M., H.I., K.M.), Shonan Kamakura General Hospital, Kamakura, Japan and Department of Neurology (M.W.), Kumamoto University School of Medicine, Kumamoto, Japan.

Address correspondence to Takahisa Mori, MD, PhD, Department of Stroke Treatment, Shonan Kamakura General Hospital, 1202-1 Yamazaki Kamakura, Kanagawa 247-8533, Japan.

criteria underwent emergency carotid artery stent placement. A retrospective review was conducted of these cases.

Inclusion Criteria

Our inclusion criteria for emergency carotid artery stent placement were patients with acute ischemic stroke who were admitted to our institution within 7 days of the stroke onset and who had serious neurologic symptoms defined as a National Institutes of Health Stroke Scale (NIHSS) score of 5 or more just before stent placement, occlusion or a high-grade stenosis of the ICA related to their neurologic symptoms, and a perfusion-diffusion mismatch on MR images.

All patients had either sudden onset of stroke with severe hemispheric neurologic deficits including disturbance of consciousness, aphasia or agnosia, or hemiplegia or hemiparesis, sometimes associated with conjugate deviation of the eyes; or progressing stroke with the neurologic deficits worsening over a 24-hour period despite maximal medical treatment (19, 20), including both fluctuating stroke and stroke in evolution (5). In all patients, a high-grade stenosis (≥70% by North American Symptomatic Carotid Endarterectomy Trial criteria) or an occlusion suspected to be acute was demonstrable angiographically in the ICA and was associated with symptoms. Additional radiologic inclusion criteria based on MR imaging (Signa EchoSpeed 1.5T, GE Yokogawa Medical Systems) were absence of or a minimal area showing high signal intensity on diffusion-weighted (DW) images and decreased CBF ipsilateral to the ICA lesion demonstrated on perfusion-weighted (PW) images. These findings are regarded as a perfusion-diffusion mismatch. In PW imaging, relative mean transit time (rMTT) maps, relative cerebral blood volume (rCBV) maps, rCBV divided by rMTT calculated as relative CBF (rCBF) maps were created with Func Tool (Signa EchoSpeed 1.5 T). Then, rMTT, rCBV, and rCBF were measured on regions of interest positioned in the bilateral middle cerebral artery (MCA) territory. In every patient, 3D time-of-flight MR angiography (28/6.9 TR/TE, 20° flip angle) was also performed.

Patients were excluded if they had embolic ICA occlusion from cardiogenic or aortogenic embolic sources, they had a medical contraindication for angiography, or the patient or family did not give informed consent. Patients with definite loss of sensorium were not excluded from undergoing emergency carotid artery stent placement.

Techniques of Stent Placement Procedure

Use of carotid stents in patients with ischemic stroke was approved by the institutional ethics committee, and patient or proxy informed consent was obtained before initiation of the procedure. Emergency carotid artery stent placement was performed as soon as possible after admission in patients with sudden onset of severe stroke or after a diagnosis of progressing stroke. Transfemoral catheterization was performed, and a guide catheter was advanced by exchange technique to the common carotid artery proximal to the ICA stenosis or occlusion. Angulated angiographic views were recorded to fully display the lesion. To facilitate sizing of balloons and stents, the vessel diameter was measured after calibration of the system with reference to the known length of the lead marker (21). A microguidewire (0.016-inch GT wire; Terumo, Tokyo, Japan) was inserted carefully to the carotid stenosis and advanced up to the distal ICA, and then a microcatheter (Transit 2; Cordis Endovascular, Johnson & Johnson, New Brunswick, NJ) was navigated across the carotid stenosis over the wire. After the 180-cm microguidewire was exchanged with a 300-cm-long microguidewire (0.014 inch, Right Away Super Hard; Piolax, Kanagawa, Japan), the microcatheter was replaced by a low-profile 3.0- or 3.5-mm balloon catheter (Ranger or Gateway; Boston Scientific, Natick, MA), which was used to moderately dilate the carotid stenosis. A self-expandable stent (Easy Wallstent; Boston Scientific) or a balloon-expandable stent (S670; Medtronic Inc., Minneapolis, MN, or NIR Elite; Boston Scientific) was deployed over the residual stenosis. Stents were selected by referring to the diameter of the ICA just distal to the carotid stenosis. In case of a narrow distal ICA or tortuosity of the proximal carotid artery, a balloon-expandable stent with a small and flexible shaft was inserted. Then a balloon catheter with a larger diameter was positioned in the stent site and inflated large enough to dilate stents and the carotid artery. After deployment of stents, the stent site as well as the distal ICA and the MCA were evaluated arteriographically. Systemic anticoagulation was provided by intravenous administration of a bolus of heparin to maintain an activated clotting time of greater than 250 seconds during the procedure.

In case of carotid occlusion, the most critical point is to find the most high-grade atherosclerotic stenotic site in the ICA that was not opacified with contrast material. A microguidewire (0.016-inch GT wire) was inserted carefully to the carotid occlusion while feeling for a high-grade atherosclerotic stenosis and advanced up to the distal ICA, and then a microcatheter (Transit 2) was navigated across the probable high-grade stenosis over the wire. Contrast material was injected slowly through the microcatheter to identify the patent ICA distal to the probable high-grade stenosis. The probable high-grade stenosis was dilated with a balloon catheter by an exchange-wire technique, same as that for a carotid stenosis. After partial recanalization of the ICA, stents were deployed on the most atherosclerotic site dilated by the balloon. Stents were selected as they were for a carotid stenosis. In case of poor opacification of the distal ICA following initial balloon angioplasty, the probable diameter of the ICA was regarded as 4–6 mm.

Management During and After Emergency Stent Placement

Heparin 5000 U was administered intraarterially through the introducing sheath just after insertion into the artery. After the procedure, each patient was monitored closely in the intensive care unit for 24 hours, with strict blood pressure control. Heparin therapy 10,000 U/day was continued for 3 days. Every patient received two antiplatelet agents orally or through a gastrointestinal tube: aspirin 100 mg/day and ticlopidine 100 mg/day. Aspirin was continued indefinitely, and ticlopidine was continued for 90 days after the procedure. Cranial CT was performed immediately, 24 hours, 7 days, and 90 days after completion of the procedure. Although there are few reports concerning the hyperperfusion phenomenon after emergency carotid stent placement in an acute stroke stage, this phenomenon does occur in some patients who undergo elective CEA or elective carotid stent placement (22–25). Probable hyperperfusion phenomenon must be detected immediately after emergency stent placement, since it may lead to cerebral hemorrhage or edema and result in poor clinical outcome. CBF should be examined to evaluate whether or not it is hyperperfusion, and single-photon emission CT (SPECT) was performed within 24 hours of the procedure to confirm occurrence of a hyperperfusion phenomenon, defined as greater flow in the ipsilateral hemisphere than on the contralateral side. Angiography was scheduled 90 days after the procedure to confirm patency of the vessel with stent.

Outcome Measures and Statistical Analysis

For all 17 patients who underwent emergency stent placement, pertinent medical records and imaging studies were reviewed to evaluate neurologic outcome per the NIHSS score at 7 days, clinical outcome per the modified Rankin Scale (mRS) score at 90 days, frequency of procedure-related complications within 30 days, and recurrence rate of ipsilateral ischemic stroke within 90 days of the procedure. Clinical outcome at 90 days was evaluated in the outpatient clinic. In patients with severe disability (mRS score 4–5) or death, the patients' families were interviewed. Change in the NIHSS score during the 7 days was estimated with the Wilcoxon rank sum test. A value

TABLE 1: Patient characteristics and clinical information before emergency carotid artery stent placement

Case No./Sex/ Age (y)	Type of Stroke	Clinical Symptoms	NIHSS Score	Treatment	Onset to Procedure (hrs)
1/F/83	SO	Deep coma, CD, QP	28	Aspirin, ticlopidine	2
2/M/65	SO	Somnolence, MA, FP, HP	9	Aspirin	10
3/M/69	SO	Coma, CD, HP	19	None	24
4/M/60	SO	Coma, HP	18	None	8
5/M/68	SO	Somnolence, HP	7	Aspirin	12
6/M/28	SO	Somnolence, FP, HP	13	None	2
7/M/78	SO	Stupor, AG, CD, FP, HP	15	None	17
8/M/83	SO	Somnolence, FP, HP	12	Aspirin	18
9/M/66	SO	Somnolence, FP, HP	11	None	6
10/F/86	PR	Somnolence, SA, HP	12	Heparin	125
11/M/69	PR	Somnolence, AG, HP	11	Heparin, ozagrel sodium	72
12/M/68	PR	AG, HP	5	Heparin, ozagrel sodium	149
13/F/74	PR	Stupor, AG, FP, HP	15	Argatroban	54
14/F/69	PR	Somnolence, FP, HP	7	Argatroban, aspirin	120
15/M/82	PR	Coma, CD, HP	24	Heparin	26
16/M/62	PR	FP, HP	5	Heparin, aspirin	120
17/M/79	PR	FP, HP	5	Argatroban, ticlopidine	168

Note.—SO indicates sudden onset of severe stroke; PR, progressing stroke; CD, conjugate deviation of the eyes; QP, quadriparesis; MA, motor-dominant aphasia; FP, facial palsy; HP, hemiplegia or hemiparesis; AG, agnosia; SA, sensory-dominant aphasia.

TABLE 2: Preprocedure MR findings

Case No./Sex/ Age (y)	Side of Lesion	Area of High Signal Intensity	DW Imaging	PW Imaging			Perfusion-Diffusion Mismatch
				rMTT	rCBV	rCBF	
1/F/83	L	CP	Small abnormality	Delay	Small decrease	Small reduction	Small
2/M/65	L	DBZ, CF	Small abnormality	Delay	Small decrease	Small reduction	Small
3/M/69	L	BG	Small abnormality	Delay	Large decrease	Large reduction	Large
4/M/60	L	None	Normal	Delay	Large decrease	Large reduction	Large
5/M/68	R	None	Normal	Delay	Large decrease	Large reduction	Large
6/M/28	R	DBZ, CF	Small abnormality	Delay	Large decrease	Large reduction	Large
7/M/78	R	SBZ, CF	Small abnormality	Delay	Small decrease	Small reduction	Small
8/M/83	R	None	Normal	Delay	Large decrease	Large reduction	Large
9/M/66	R	DBZ, CF	Small abnormality	Delay	Large decrease	Large reduction	Large
10/F/86	L	SBZ, DBZ	Small abnormality	Delay	Large decrease	Large reduction	Large
11/M/69	R	DBZ, CP	Small abnormality	Delay	Small decrease	Small reduction	Small
12/M/68	R	SBZ	Small abnormality	Delay	Small decrease	Small reduction*	Small
13/F/74	R	DBZ, CF	Small abnormality	Delay	Small decrease	Small reduction	Small
14/F/69	L	DBZ	Small abnormality	Delay	Small decrease	Small reduction	Small
15/M/82	L	CF	Small abnormality	Delay	Small decrease	Small reduction	Small
16/M/62	L	DBZ	Small abnormality	Delay	Large decrease	Large reduction	Large
17/M/79	R	DBZ	Small abnormality	Delay	Small decrease	Small reduction	Small

Note.—CP indicates cortex of the parietal lobe; DBZ, deep border zone; CF, cortex of the frontal lobe; BG, basal ganglia; SBZ, superficial border zone.

* Contralateral ICA stenosis.

of *P* less than .05 was considered indicative of statistical significance. A commercially available software package (StatView, version 5; SAS Institute Inc., Cary, NC) was used to perform the statistical analysis.

Results

Among the 17 patients who underwent emergency carotid artery stent placement at the our institution, 13 were male and four were female; ages ranged from 28 to 86 years (mean, 69.9 years). Nine patients demonstrated sudden onset of severe stroke, whereas eight demonstrated progressing stroke. Oral antiplatelete drugs had been started more than 30 days before admission in four patients with sudden onset of severe stroke (Table 1) and in one patient with progressing stroke (case 16), whereas these drugs had been started several days before stent placement in two patients with progressing stroke (5 and 7 days in cases 14 and 17, respectively). All patients with progressing stroke had received an intravenous antithrombotic agent immediately after admission. Mean elapsed time from onset to emergency stent placement was 54.9 ± 58.1 hours (Table 1).

Preprocedure DW images depicted no acute infarction in three patients, and a perfusion-diffusion mismatch was noted in all patients (Table 2). On angiograms, mean degree of ICA stenosis was 93.5 ± 10.0%, including four occlusions (Table 3). ICA pseu-

TABLE 3: Preprocedure angiographic findings and devices used for emergency carotid artery stent placement

Case No./Sex/ Age (y)	Preprocedure Stenosis (%)	Cause of Lesion	Stent Devices	Protection Technique
1/F/83	99	Atherothrombotic	S670	No
2/M/65	99	Atherothrombotic	S670	No
3/M/69	94	Atherothrombotic	NIR Elite	No
4/M/60	100	Atherothrombotic	S670	No
5/M/68	93	Atherothrombotic	S670	No
6/M/28	100	Dissection	Easy Wallstent	No
7/M/78	99	Atherothrombotic	NIR Elite	No
8/M/83	100	Atherothrombotic	Easy Wallstent	Yes
9/M/66	99	Atherothrombotic	Easy Wallstent	Yes
10/F/86	100	Atherothrombotic	S670	No
11/M/69	95	Atherothrombotic	S670	No
12/M/68	75	Atherothrombotic	NIR Elite	No
13/F/74	99	Atherothrombotic	S670	No
14/F/69	99	Atherothrombotic	NIR Elite	No
15/M/82	70	Atherothrombotic	Easy Wallstent	No
16/M/62	95	Atherothrombotic	Easy Wallstent	No
17/M/79	75	Atherothrombotic	Easy Wallstent	No

do-occlusion (26) was judged to represent 99% stenosis. In all patients except one with dissection, the cause of the ICA stenosis or occlusion was considered to be atherothrombosis. Ipsilateral intracranial ICA stenosis representing a tandem lesion was detected in one patient (case 15); ipsilateral intracranial ICA-MCA occlusion representing coincidental occlusion or distal embolism was seen in another (case 6). Contralateral ICA stenosis was present in one patient (case 12), bilateral vertebral artery severe stenosis was seen in one patient (case 1), and basilar artery occlusion was seen in another (case 5).

The technical success rate was 100%, with 17 carotid arteries treated (Figs 1–4). Balloon-expandable stents were used in 11 arteries. No protective device was used during the procedure in 15 patients, whereas distal protection was used in two (Table 3). The mean degree of stenosis in the treated ICA just after stent placement was 8.5 ± 6.3%, and no restenosis was seen in the 13 patients who underwent follow-up angiography at 90 days (Table 4).

SPECT performed within 24 hours after stent placement revealed hyperperfusion phenomenon in four patients, all of whom received additional treatment with an antihypertensive drug, a sedative, an anticonvulsant, and edaravone (27) as prophylaxis against cerebral hyperperfusion syndrome. CT just after stent placement revealed no abnormal findings. CT 24 hours after stent placement disclosed new findings in three patients. Ipsilateral subarachnoid hemorrhage with mild cerebral edema was found in two patients, but remained subclinical and disappeared within 7 days (Table 4). New putaminal infarction occurred in one patient (case 7).

Median NIHSS scores before and at 7 days after stent placement were 12 and 5, respectively, representing rapid significant improvement ($P < .01$, Wilcoxon rank sum test; Fig 5). Ten patients (59%) had favorable outcomes (mRS score 0–1), three patients (18%) sustained moderate disability (mRS score 2–3), and three patients (18%) had severe disability (mRS score 4–5) at 90 days. The other patient (6%) died of congestive heart failure 10 days after the procedure (Table 5).

Hypotension and bradycardia occurred during or after angioplasty in four patients, who were treated with intravenous atropine and transvenous temporary pacing; hypertension occurred in five patients, who were treated with antihypertensive agents (Table 5). All of these occurrences were reversible, with complete recovery within 24 hours of the procedure. However, two patients (12%) had irreversible neurologic deficits related to stent placement at 30 days; these resulted from distal embolism during the procedure in one and from intracerebral hemorrhage 5 days after the procedure in the other. The first patient (case 7) suddenly developed left hemiplegia immediately after the procedure, and angiography revealed MCA occlusion. Thrombolysis was performed with urokinase, administered locally into the MCA through a microcatheter, achieving partial MCA recanalization. However, his symptoms—hemisomatognosia and left hemiparesis—did not resolve, and follow-up CT disclosed a right putaminal infarction not present before the procedure. The second patient (case 16) developed headache, vomiting, and arterial hypertension 5 days after the procedure, rapidly followed by global aphasia and right hemiplegia. CT disclosed a large left putaminal intracerebral hemorrhage; aggressive treatment of elevated intracranial pressure failed to improve his neurologic deficit. Except for the one embolic complication, no ipsilateral ischemic stroke recurrence was seen within 90 days of the emergency stent placement procedure.

Discussion

The natural history of carotid-related ischemic stroke is unclear. According to a review by Meyer et al (4), the prognosis for patients with acute stroke with carotid occlusion is poor: 40–69% of these patients were permanently disabled, 16–55% died of the

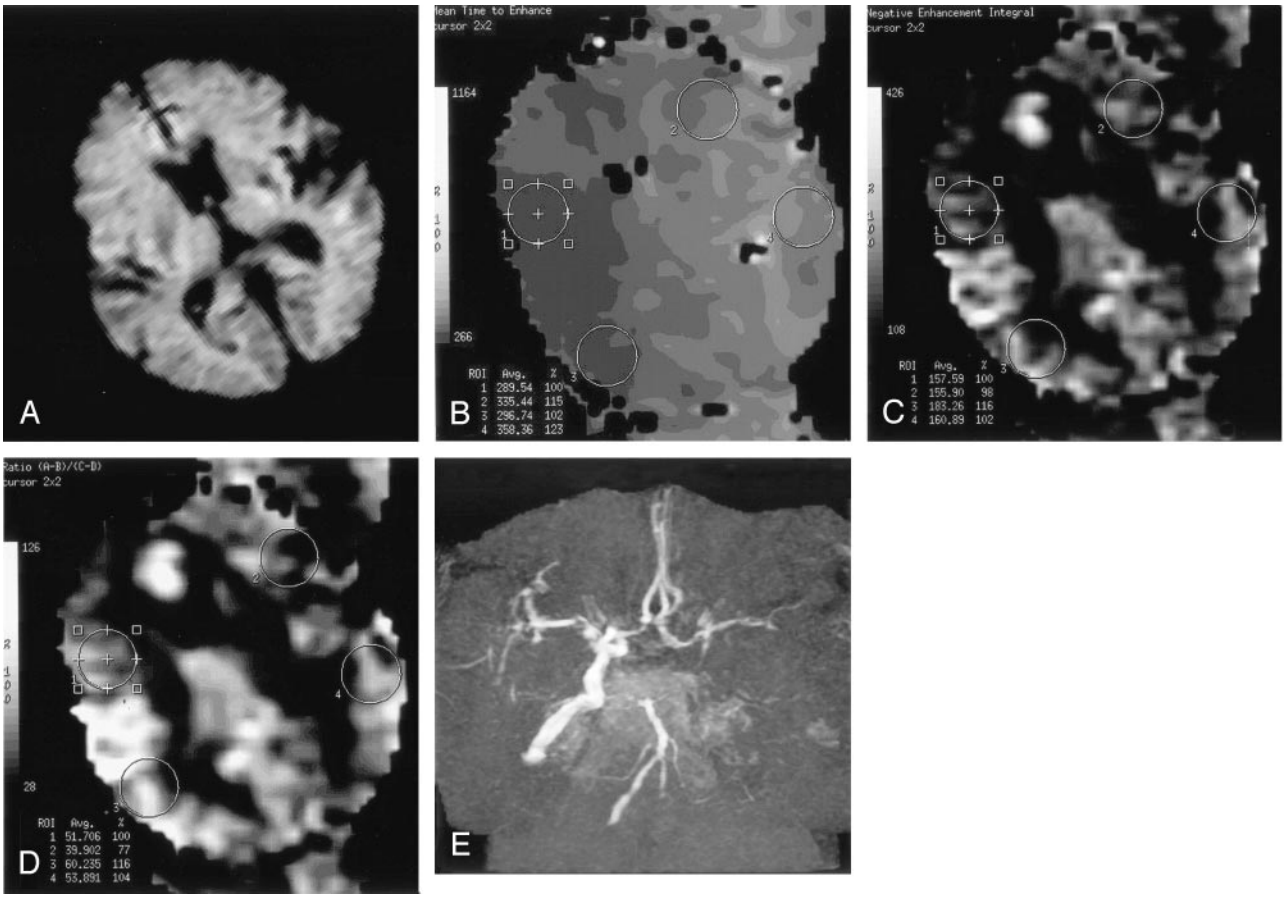

FIG 1. Images obtained on admission in case 1.
A, DW image reveals slightly high signal intensity in the cortex of the left parietal lobe.
B, The rMTT map of PW image shows delay of 15–25% in the territory of the left MCA (ROI 2 and 4).
C, The rCBV map of PW image shows slight decrease of 2–12% in the territory of the left MCA (ROI 2 and 4).
D, The rCBF map of PW image reveals reduction of 11–23% in the territory of the left MCA (ROI 2 and 4).
E, MR angiogram shows poor visualization of the left ICA.

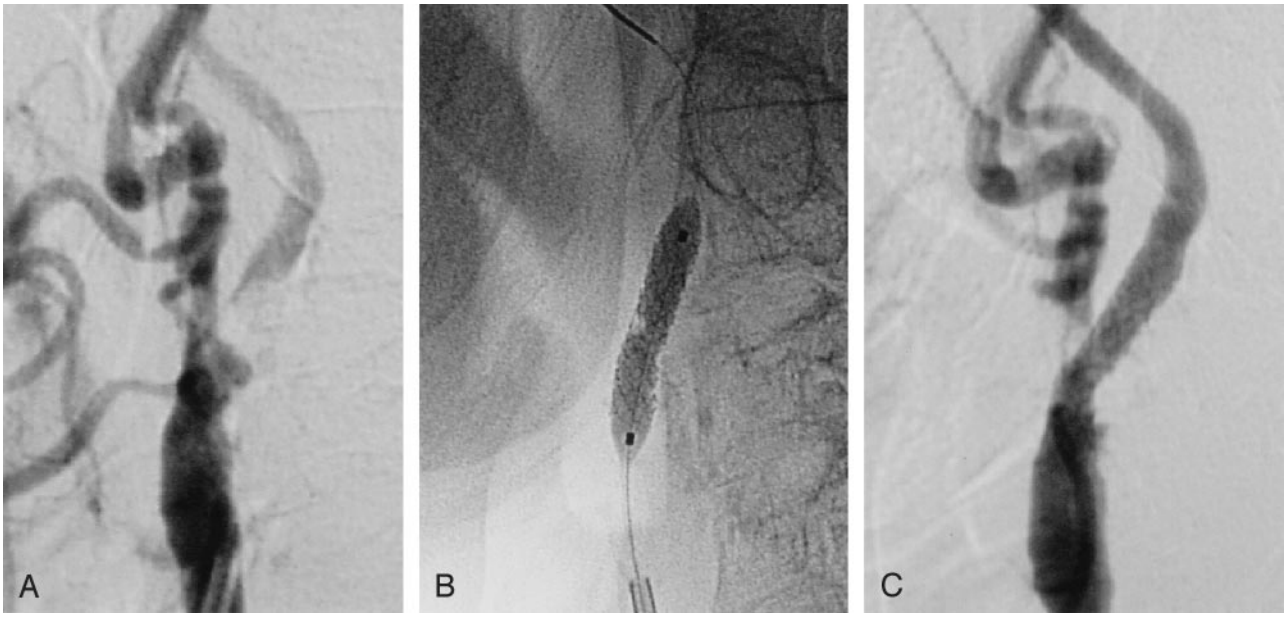

FIG 2. Periprocedural images obtained in case 1.
A, Lateral left carotid angiogram obtained before the procedure shows severe stenosis at the origin of the ICA.
B, Lateral left carotid angiogram obtained during the procedure demonstrates stent implantation and postdilation.
C, Lateral left carotid angiogram obtained after the procedure shows an excellent angiographic result.

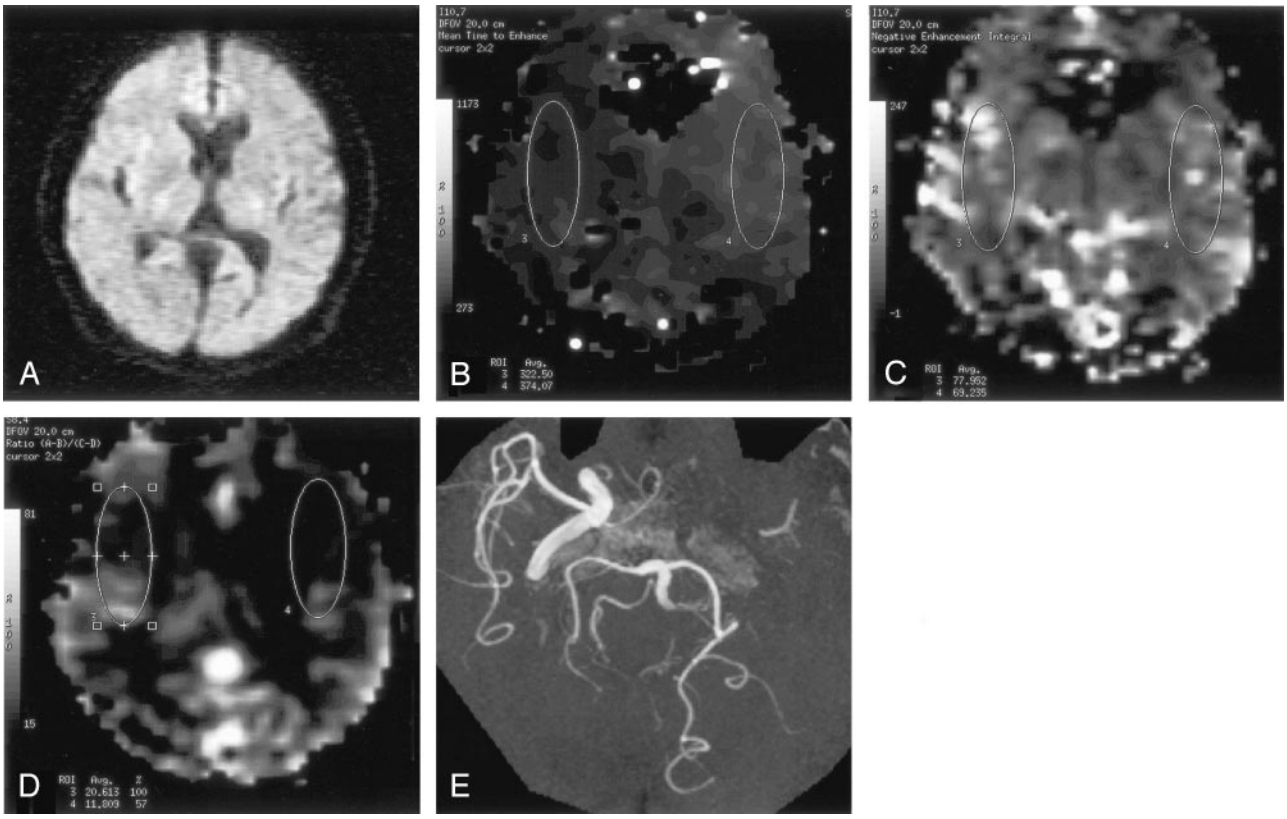

FIG 3. Images obtained on admission in case 4.
A, DW image reveals no area of high signal intensity in the left cerebral hemisphere.
B, The rMTT map of PW image shows delay of 15% in the territory of the left MCA (ROI 4).
C, The rCBV map of PW image shows slight decrease of 11.2% in the territory of the left MCA (ROI 4).
D, The rCBF map of PW image reveals reduction of 43% in the territory of the left MCA (ROI 4).
E, MR angiogram shows no visualization of the left ICA.

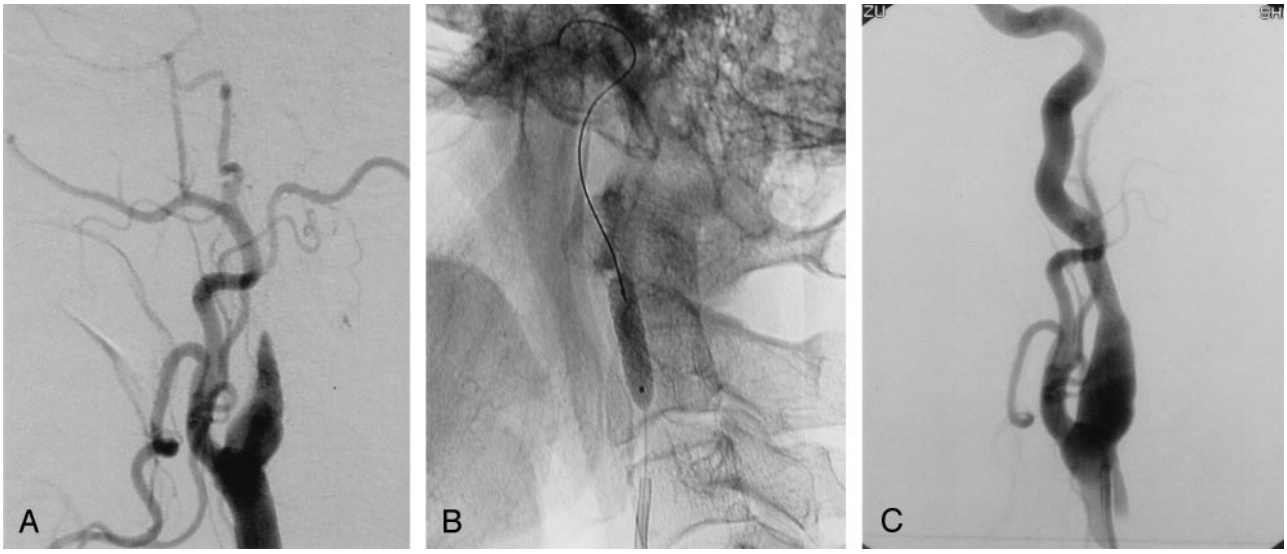

FIG 4. Periprocedural angiograms obtained in case 4.
A, Lateral left carotid angiogram obtained before the procedure shows occlusion at the origin of the ICA.
B, Lateral left carotid angiogram obtained during the procedure demonstrates stent implantation and postdilation.
C, Lateral left carotid angiogram obtained after the procedure shows essentially complete recanalization with 14% residual stenosis of the ICA.

stroke, and only 2–12% had a good recovery. The natural history of progressing stroke also is unfavorable, with mortality rates of 14–36% and disability rates of 54–60% (28, 29). Of 17 patients in the present study, 10 (59%) recovered or were considered to have a nondisabling stroke at 90 days after emer-

TABLE 4: Postprocedure findings

Case No./Sex/ Age (y)	Postprocedure Stenosis (%)*	Stenosis at 90 days (%)*	HP at SPECT	CT Findings within 24 hrs after Procedure
1/F/83	5	20	Negative	SLA
2/M/65	10	20	Negative	SLA
3/M/69	12	12	Negative	SLA
4/M/60	14	40	Positive	NLA + SAH
5/M/68	0	0	Positive	NLA
6/M/28	8	8	Negative	LLA
7/M/78	12	20	Negative	LLA
8/M/83	5	5	Negative	SLA
9/M/66	15	0	Positive	SLA
10/F/86	20	No evaluation	Positive	SLA + SAH
11/M/69	0	No evaluation	Negative	SLA
12/M/68	0	10	Negative	SLA
13/F/74	18	20	Negative	SLA
14/F/69	8	8	Negative	NLA
15/M/82	10	No evaluation	Negative	SLA
16/M/62	8	0	Negative	SLA
17/M/79	0	No evaluation	Negative	SLA

Note.—HP indicates hyperperfusion phenomenon; SLA, small low-attenuation area; LLA, large low-attenuation area; NLA, no low-attenuation area; SAH, subarachnoid hemorrhage.

* Evaluation by angiography.

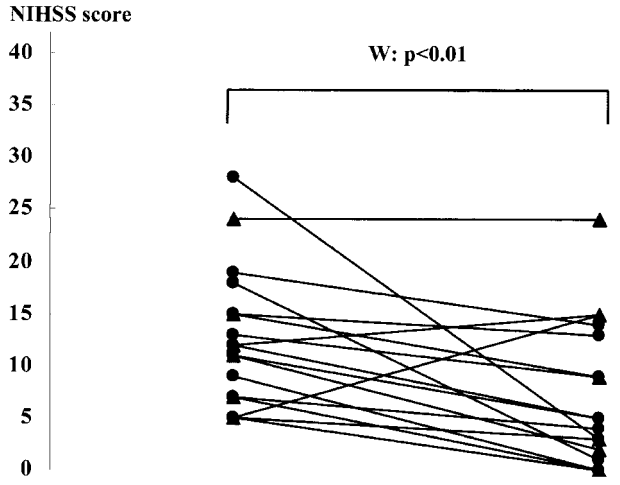

FIG 5. NIHSS scores at 7 days after emergency carotid stent placement (symbols on right) improved significantly ($P < .01$) compared with baseline scores (symbols on left). *W* indicates Wilcoxon rank sum test; ●, patients with sudden onset of severe stroke; ▲, patients with progressing stroke.

gency carotid artery stent placement. When patients were grouped by time course, six (67%) of nine patients with sudden onset of severe stroke and four (50%) of eight patients with progressing stroke were nondisabled at 90 days. These results are better than those reported previously, and NIHSS scores at 7 days after emergency stent placement had improved significantly from baseline scores. Thus, in this small study, emergency carotid artery stent placement compared with historical control subjects treated with medical management was associated with a favorable outcome. More study is required to determine the role of this therapy in the treatment of patients with acute ischemic stroke and an associated significant ipsilateral carotid artery stenosis or occlusion.

Some studies have reported efficacy of emergency CEA in carotid-related stroke (3–8). A previous study (6) of emergency CEA reported results comparable to those of the present emergency stent placement study, which, however, included more serious cases. As for safety of emergency carotid stent placement, two permanent procedure-related complications occurred in the present study (12%), whereas morbidity rates of 6.3–9.9% have been reported in studies of emergency CEA (4, 6, 7), with rates in elective carotid stent placement ranging from 2.4% to 4.4% (13, 14). Thus, emergency carotid stent placement has safety at least comparable to that of emergency CEA, but is not as safe as elective carotid stent placement.

Artery-to-artery embolism, hemodynamic insufficiency, acute circulatory failure, or a combination of these (30, 31), perhaps triggered by plaque rupture or hemorrhage into a plaque (32), might account for progression in carotid-related stroke. The main therapeutic aim of emergency carotid stent placement is not removal of an ongoing embolic source, but restoration of blood flow to rescue the ischemic penumbra in the affected hemisphere. The present study therefore included patients with sudden onset of severe stroke or with progressing stroke and excluded patients with crescendo transient ischemic attacks; the latter are believed largely to result from repeated artery-to-artery carotid embolism, since such patients' carotid plaques commonly show surface irregularity and ulceration at pathologic examination (33). In addition, all of our patients who underwent emergency carotid stent placement were examined with PW imaging before the procedure to confirm a decrease of CBF in the affected hemisphere.

Some smaller nonrandomized studies demonstrated efficacy of emergency CEA for patients with carotid territory stroke, but they excluded patients in coma (5–8); these patients were included in our

TABLE 5: Clinical results

Case No./Sex/ Age (y)	NIHSS Score			mRS Score at 90 Days after Procedure	Procedure-Related Complications
	On Admission	Just Before Procedure	At 7 Days after Procedure		
1/F/83	28	28	3	1	Hypotension, bradycardia
2/M/65	9	9	0	0	None
3/M/69	19	19	14	4	None
4/M/60	18	18	1	0	Hypertension
5/M/68	7	7	4	0	Hypotension, bradycardia
6/M/28	13	13	9	3	None
7/M/78	15	15	13	4	Hypertension, distal embolism
8/M/83	12	12	5	1	Hypertension
9/M/66	11	11	5	1	Hypertension
10/F/86	4	12	15	4	Hypertension
11/M/69	11*	11	2	2	Hypotension, bradycardia
12/M/68	2	5	0	0	None
13/F/74	15*	15	9	1	None
14/F/69	4	7	0	0	None
15/M/82	3	24	24	6	None
16/M/62	1	5	15	3	Putaminal hemorrhage
17/M/79	2	5	3	0	Hypotension, bradycardia

* Scores of patients transferred from another hospital.

present study unless a large area of high signal intensity was detectable on DW images in the ipsilateral hemisphere. Estimation of degree of viability in the affected hemisphere by DW imaging may be essential for attempting emergency carotid stent placement. By using DW imaging to exclude high-risk patients from undergoing emergency stent placement who had radiologic evidence of large irreversible ischemic stroke, we could minimize the chance that the patient would have massive hemorrhagic transformation of the infarct after recanalization. Since reversibility of the brain tissue ischemia is associated strongly with collateral flow, the time window for recanalization by emergency carotid stent placement is variable in patients with carotid-related stroke.

Whether to perform emergency intervention, either CEA or carotid stent placement, in patients with acute stroke who have symptom-related ICA occlusion has been controversial (4–7, 32, 34–37). When acute neurologic deficits are ascribed to ICA occlusion, they can be reduced if the ICA is recanalized without delay, and emergency carotid stent placement was attempted in such patients. No definitive indication for emergency stent placement has been demonstrated in patients with an occluded ICA. A significantly higher risk of complications such as embolic events or even perforation of the arterial wall can be anticipated in such patients, since clot invariably forms distal to an occlusion site and propagates to the first main collateral, while remaining fixed to the arterial wall. Particular care may be necessary if emergency carotid stent placement is to be performed safely in such a situation.

Neither localized thrombolysis nor angioplasty for MCA occlusion coupled with ipsilateral ICA occlusion (case 6) was performed, because DW imaging showed irreversible infarction of a large territory in the ipsilateral hemisphere. Eckstein et al (6) reported that neurologic outcome following emergency CEA was strongly influenced by coexistent MCA embolism. Mori et al (18) reported emergency angioplasty and stent placement for both embolic MCA occlusion and ipsilateral ICA stenosis at the acute stroke stage. In the present study, one patient (case 7) had a diagnosis of embolic MCA occlusion immediately after emergency carotid stent placement, which likely occurred in association with the procedure; additional thrombolysis with urokinase was then performed. A combination of thrombolysis and emergency stent placement may offer treatment for both the embolus and the focus of ICA stenosis (32, 34).

Other procedure-related complications, including hemodynamic instability such as hypotension, hypertension, or bradycardia, occurred in nine (53%) of 17 patients. The frequency of these complications in the present study did not differ from that seen in a study on elective carotid stent placement (38), nonetheless, hemodynamic instability may increase damage to ischemic penumbra in the acute stage, so rigorous management of vital signs is required in patients undergoing emergency carotid stent placement. In one patient (case 15) who died of congestive heart failure 10 days after the procedure, emergency stent placement might have aggravated his condition, although he had manifested congestive heart failure before the procedure and hemodynamic instability did not deteriorate during the procedure.

In another patient (case 16), putaminal hemorrhage occurred 5 days after the procedure and probably was related to the cerebral hyperperfusion syndrome. His small infarct in the deep border zone on DW images before the procedure showed no change on postprocedure CT scans; his intracerebral hemorrhage did not represent hemorrhagic transformation of the infarct, but probably resulted from delayed cerebral hyperperfusion syndrome. Although post-

procedure SPECT disclosed no hyperperfusion phenomenon in this patient, delayed hyperperfusion might have occurred in relation to increased ipsilateral cerebral perfusion, as parasympathetic discharge after stent placement in the carotid sinus was restored. After angioplasty of an extracranial artery, the cerebral hyperperfusion syndrome has been reported to occur in 5% of patients even during the chronic stage (25). One mechanism of hyperperfusion syndrome involves impairment of cerebrovascular autoregulation, which is more severe in the acute stroke stage; preocclusive stenosis of 90–99% is reported to carry an even greater risk for its development in patients undergoing CEA (22). As for occurrence of either hyperperfusion phenomena or delayed cerebral hyperperfusion syndrome in patients undergoing emergency carotid stent placement, one of these conditions developed in five (29%) of our 17 patients, who overall had a mean degree of stenosis of 93.5%. This occurrence is much higher than that reported in elective carotid stent placement or in emergency CEA (7). Mori et al (24) reported intraventricular hemorrhage 4 hours after carotid stent placement in a patient who had shown severely reduced regional vasoreactivity ipsilateral to the ICA lesion on preprocedure SPECT images. A great reduction in regional vasoreactivity can be expected in all patients with sudden onset of a major stroke or with progressing stroke. However, our patients included two who developed subclinical subarachnoid hemorrhage as a reperfusion injury, which has not been reported after CEA, although Schoser et al (23) reported one patient developing severe subarachnoid hemorrhage 16 hours after elective angioplasty. Therefore, cerebral hyperperfusion syndrome may occur occasionally in patients undergoing emergency carotid stent placement. As clinical findings in cerebral hyperperfusion may be difficult to differentiate from those in cerebral ischemia, strict monitoring for evidence of hyperperfusion is necessary for at least the first 7 days after the procedure. Signs suggestive of hyperperfusion are an indication for aggressive blood pressure control even in the acute ischemic stage. Not only impaired cerebral autoregulation, but also hypertension associated with carotid baroreceptor or microvascular failure at the level of the blood-brain barrier reflecting perfusion breakthrough into a recent infarct or spontaneous hemorrhagic conversion by showers of microemboli may occur after stent placement (39); these may contribute to cereberal hyperperfusion syndrome in emergency carotid stent placement. Further investigation is needed to better define underlying mechanisms.

Since an antithrombotic agent had not been in use before emergency stent placement in five of nine patients with sudden onset of severe stroke in the present study, intravenous heparin was administered continuously and daily oral antiplatelet agents were prescribed to avoid subacute thrombosis of vessels with stents and consequent thromboembolism. However, we remained vigilant since antithrombotic therapy carries risks of hemorrhagic complications such as hemorrhagic infarction or cerebral hyperperfusion syndrome.

Conclusion

Our results suggest that emergency carotid artery stent placement can improve 7-day neurologic outcome and may improve clinical outcome at 90 days in selected patients with ischemic stroke and total occlusion or a high-grade stenosis of the ICA.

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