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Symptomatic Patients with Intraluminal Carotid Thrombus: Outcome with a Strategy of Initial Anticoagulation

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Abstract

Background—Optimal treatment for patients with symptomatic intraluminal carotid thrombus (ICT) remains poorly defined.

Methods—We performed a retrospective chart review of patients presenting to our institution between 2001 and 2011 with symptomatic ICT.

Results—Twenty-four patients (16 males, 8 females) with ICT presented with ischemic stroke (n=18) or TIA (n=6). All were treated initially with anticoagulation ± antiplatelet drugs. Eight of these patients had no or mild carotid stenosis on initial angiography and were treated with medical management alone. The remaining 16 patients had moderate or severe carotid stenosis on initial angiography. Of these, 10 underwent delayed revascularization (endarterectomy, n=8; angioplasty and stenting, n=2), 2 refused revascularization, and 4 were treated with medical therapy alone. One patient had multiple TIAs despite medical therapy and eventually underwent CEA; the remaining 23 patients had no TIAs. No patient suffered ischemic or hemorrhagic stroke while on anticoagulation, during the perioperative period or in long-term follow up; one patient died of an unrelated condition (mean follow-up = 16.4 months).

Conclusion—Our results suggest that initial anticoagulation of symptomatic ICT results in a low rate of recurrent ischemic events and that carotid revascularization, if indicated, can be safely performed in a delayed manner.

Keywords

Carotid thrombus; Anticoagulation; Carotid endarterectomy; Carotid stenting

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INTRODUCTION

Intraluminal carotid artery thrombus (ICT) is an infrequent finding in patients presenting with transient ischemic attack (TIA) or ischemic stroke. It can be a focal process limited to the cervical internal carotid artery (ICA), or a more extensive condition that involves both the extracranial and intracranial ICA.⁶ ICT can be found in the presence or absence of carotid stenosis. When identified in patients without underlying carotid stenosis, it is often associated with a hypercoagulable state.^{3,6} When identified in patients with underlying carotid stenosis, its incidence is correlated to the severity of atherosclerotic disease. In the North American Symptomatic Carotid Endarterectomy Trial (NASCET), the frequency of ICT was 1.1% in patients with <70% stenosis vs. 4.3% in patients with >70% stenosis and 5.5% in patients with >85% stenosis.^{4,18}

Once ICT is identified, its presence merits special consideration due a high risk of recurrent ischemic events. In NASCET, patients randomized to antiplatelet medical therapy who had ICT on initial angiography had a significantly higher risk of stroke and death at 1 month as compared to medically treated patients without ICT (11% vs. 2%, respectively).^{4,18} Due to this aggressive clinical course, some consider ICT an urgent or emergent condition that requires early carotid endarterectomy (CEA);^{7,19} however, the risks associated with this strategy have been considerable.⁶ In NASCET, patients undergoing CEA in the presence of ICT had twice the risk of perioperative stroke and death as compared to patients undergoing CEA without ICT (12% vs. 6%, respectively).^{4,18} Similarly, in a multicenter retrospective review of symptomatic patients undergoing CEA, patients with ICT had a nearly threefold higher risk of perioperative stroke as compared to patients without ICT (14% vs. 5%, respectively).¹⁰

Optimal management of patients with symptomatic ICT, therefore, remains poorly defined. Reported treatment options include- 1) medical management with anticoagulation ± antiplatelet drugs,⁶ 2) early carotid revascularization with CEA^{5,6} or carotid artery stenting (CAS),^{13,14} and 3) initial medical management with anticoagulation ± antiplatelet drugs followed by delayed revascularization via CEA^{6,8} or CAS.¹¹ However, results with each of these therapeutic strategies are based on a few small case series, and available evidence does not provide clarity as to best practice for this patient population.

At our institution, patients with symptomatic ICT are treated with initial anticoagulation followed by delayed revascularization via CEA or CAS, if indicated (Figure 1). In this study, we retrospectively evaluated our experience in the management of patients presenting with TIA or acute ischemic stroke due to ICT, and compared our results to the published literature.

PATIENTS AND METHODS

This study was approved by the Human Research Protection Office.

Patient Identification

In our practice, digital subtraction angiography (DSA) is obtained as part of routine care in patients presenting with ischemic symptoms and Doppler ultrasound evidence of 50% stenosis of the ipsilateral carotid artery, in order to determine whether they are candidates for surgical or endovascular revascularization. Patients that are not good candidates for either procedure owing to their health or the severity of their stroke do not undergo DSA. A computerized database of adult patients who underwent DSA at our institution between January 2001 and February 2011 was queried and patients with a diagnosis of ICT, defined as a filling defect in the arterial lumen that was visible on at least two planes, were

identified. All studies were performed by one of three senior interventional neuroradiologists.

Eligibility Criteria

Patients who were symptomatic (TIAs or stroke) with intraluminal thrombus in the common carotid artery (CCA) or extracranial internal carotid arteries (ICA) were included in this study. Exclusion criteria were- 1) Thrombus confined to intracranial ICA, 2) Thrombus associated with trauma and/or dissection, 3) History of CAS ipsilateral to the thrombus (i.e. in-stent thrombosis), 4) Ischemic stroke with hemorrhagic transformation, and 5) Extensive medical co-morbidities at the time of presentation that limited life expectancy.

Chart Review

Medical records of identified patients were retrospectively reviewed and the following data collected- age, sex, comorbidities (hypertension, diabetes mellitus, hyperlipidemia, coronary artery disease, chronic heart failure), history of previous stroke or TIA, current medications (especially antiplatelet agents or anticoagulants), smoking history, clinical presentation (TIA or stroke, number of events prior to diagnosis, time from first event to diagnosis, NIHSS at presentation), admission laboratory values (including hemoglobin, hematocrit, and platelet count), angiographic findings at presentation (degree of stenosis bilaterally, location of thrombus relative to stenosis if present, and hemodynamic effect of the thrombus), treatment (use of anticoagulant and antiplatelet agents, duration of anticoagulation), and follow up (clinical and radiographic). Comorbidities and current medications were identified from self reported history. Clinical presentation was considered to be TIA if the patient presented with an acute focal neurological deficit(s) lasting less than 24 hours, and stroke if there was acute onset focal neurological deficit(s) lasting greater than 24 hours. The duration of symptoms was recorded as 1 day if symptoms were present for 24 hours or less. NIHSS score was assessed retrospectively from examination findings at admission if not directly available in the medical records.¹⁵ Outcome was assessed using radiographic and clinical evaluation by a treating neurologist or neurosurgeon at follow up. The degree of stenosis was classified per NASCET criteria.² For patients who were receiving anticoagulant drugs at last follow up, the interval from initiation of treatment to last follow up was considered to be the duration of anticoagulation.

In our practice, patients with ICT who have minimal atherosclerotic disease on initial angiography undergo a diagnostic workup for underlying hypercoagulable conditions including factor V Leiden mutation, prothrombin G20210A mutation, protein C deficiency, protein S deficiency, antithrombin deficiency, antiphospholipid antibody syndrome and hyperhomocysteinemia. In addition, we have previously identified coexisting anemia and thrombocytosis as a risk factor for ICT in patients without significant underlying carotid stenosis.³ These data were also extracted from the medical records.

RESULTS

Patient Characteristics

Twenty-four patients (16 males, 8 females) with ICT satisfying the inclusion and exclusion criteria were identified. The median age at presentation was 55 years (IQR: 46 to 68 yrs). The majority of patients had known risk factors for stroke: hypertension (54%), hyperlipidemia (50%), smoking (33%), history of stroke/TIA (25%), coronary artery disease (25%), diabetes mellitus (21%), and atrial fibrillation (4.2%). There was a history of use of oral contraceptives in one female patient (12.5%). At presentation, 7 patients (29%) were on antiplatelet drugs, and 1 patient (4.2%) was on warfarin for atrial fibrillation with a therapeutic INR (2.2).

Among the 24 patients, 7 patients had a hypercoagulable state – 1 with antiphospholipid antibody syndrome, 1 with systemic lupus erythematosus and thrombocytosis, 1 with essential thrombocytosis, and 4 with iron deficiency anemia with thrombocytosis. A diagnostic work up for other hypercoagulable conditions including factor V Leiden mutation, prothrombin G20210A mutation, protein C deficiency, protein S deficiency, antithrombin deficiency, antiphospholipid antibody syndrome and hyperhomocysteinemia was negative in patients with iron deficiency anemia and thrombocytosis. The median age of patients presenting with hypercoagulability was significantly lower than those without a hypercoagulable state (43 vs. 59 years, $p=0.006$ by Mann Whitney test). Patient characteristics are further outlined in Tables 1 and 2.

Clinical and Radiographic Presentation

Most patients (75%) presented with ischemic stroke; the remainder presented with TIA (25%). Duration of symptoms ranged from <3 hours to 4.5 months (median: 1 day; interquartile range (IQR): 1 to 2 days). However, all patients were acutely symptomatic, with the last symptomatic episode being < 1 week from presentation. Median NIHSS score at presentation was 4 (IQR: 1 to 10) in patients with ischemic stroke. There was insufficient data for NIHSS assessment in two patients.

Among the 24 patients, 12 had high-grade stenosis, 4 had moderate stenosis and 8 had no or mild stenosis on initial angiography per NASCET criteria.¹ One patient who presented with TIA and had no carotid stenosis on initial angiography had a history of ipsilateral CEA for severe stenosis one year prior. Decreased distal flow and/or luminal collapse of the distal ICA due to the ICT was noted in 14 of 24 patients.

Among the 7 patients with a hypercoagulable state, 6 had no or minimal stenosis and one had severe stenosis on initial angiography. Decreased distal flow and/or luminal collapse of the distal ICA was seen in 3 of these patients.

Initial Treatment

All patients were treated at presentation with anticoagulation. The anticoagulation regimen varied– unfractionated heparin (UFH) only (n=3), low molecular weight heparin (LMWH) only (n=2), warfarin only (n=1), UFH followed by warfarin (n=10), UFH with LMWH bridge followed by warfarin (n=4), LMWH followed by warfarin (n=3), and UFH followed by LMWH (n=1). None of the patients developed anticoagulation associated complications. Nineteen patients also received concurrent antiplatelet therapy of which 17 were treated with aspirin alone while 2 received aspirin and clopidogrel.

Among patients with a hypercoagulable state, 4 were anticoagulated with UFH and warfarin, 2 with UFH, LMWH bridge and warfarin, and 1 with LMWH followed by warfarin. Four patients also received antiplatelet agents (aspirin). Treatment details are summarized in Tables 3 and 4.

Angiographic Follow Up

Of the 8 patients with no or mild carotid stenosis on initial DSA, two had follow up vascular imaging. One patient underwent follow up magnetic resonance angiography 12 days after initiation of anticoagulation to evaluate new onset headache. It showed complete resolution of the ICT and absence of residual carotid stenosis. The other patient underwent follow up computed tomography angiography (CTA) 7 months after initiation of anticoagulation due to uncertainty regarding the degree of stenosis on initial DSA (49%). Complete resolution of the ICT and mild residual stenosis was noted. Of the 16 patients with moderate or high grade stenosis on initial DSA, 11 underwent repeat vascular imaging (all DSA). Complete

resolution of the ICT was observed in 10 of these 11 patients, while one had substantial but incomplete resolution of the ICT. Residual moderate to severe stenosis was observed in 10 of these 11 patients, while one patient had only minimal residual stenosis. Follow up data is outlined further in Tables 3 and 4.

Delayed Revascularization

Among the 8 patients with no or mild carotid stenosis on initial DSA, none were offered delayed revascularization. Among the 16 patients with moderate to high grade carotid stenosis on initial DSA, 8 underwent delayed CEA, 2 underwent delayed CAS, 2 refused delayed revascularization, 3 were not offered delayed revascularization (2 due to severe comorbidities; 1 due to downgrading of carotid stenosis to minimal on repeat DSA), and 1 was lost to follow up after initiation of anticoagulation. Mean interval from clinical presentation to delayed revascularization was 39.3 days (range 15 – 91 days).

Clinical Follow Up

Mean duration of anticoagulation was 30 days (range 7 – 53 days) in the 10 patients who underwent delayed revascularization and 103 days (range 7 – 318 days) in the 13 patients who underwent medical therapy alone (duration of anticoagulation was not available in one patient, who was lost to follow up). Mean duration of clinical follow up for the entire patient cohort was 70 weeks (range 5–368 weeks). No patient in this cohort suffered a recurrent ischemic stroke. One patient (4.2%), with high-grade stenosis on initial DSA, experienced TIAs while on anticoagulation. He underwent follow up DSA 28 days after initiation of anticoagulation, which revealed substantial but incomplete resolution of the ICT and 90% residual carotid stenosis. He underwent an uncomplicated CEA the next day where no evidence of ICT was noted, and he experienced no recurrent ischemic events in long-term follow up (74.5 weeks). Among patients who underwent delayed revascularization via CEA or CAS, none suffered peri-operative ischemic stroke or death. One patient had a post-CEA neck hematoma that required surgical evacuation. No procedural complications were noted in those undergoing CAS.

In long-term follow up, one patient died due to complications of metastatic breast cancer. This patient had no ischemic complications during the 90.2 weeks of available follow up. There was no other patient mortality. One patient was lost to follow up.

DISCUSSION

Symptomatic ICT is associated with high risk of recurrent ischemic events and poor patient outcome.⁴ Optimal management of this patient population remains controversial, mainly due to the rarity of reported cases. Here, we report excellent results with a strategy of initial anticoagulation followed by delayed revascularization (as indicated). None of our 24 patients (14 of whom were treated with anticoagulation alone; 10 of whom were treated with anticoagulation followed by delayed revascularization) suffered new ischemic or hemorrhagic stroke and none suffered ICT-related death in late clinical follow up (mean = 70 weeks) (Tables 1–4). When comparing our results to published reports with strategies of antiplatelet therapy alone (11% risk of stroke and death at 1 month)^{4,18} or early CEA (12–14% risk of stroke and death at 1 month),^{4,10,18} the strategy of initial anticoagulation ± antiplatelet therapy to permit ICT resolution followed by delayed revascularization (if needed) appears superior (Table 5).

Results from smaller case series in which ICT patients were treated with anticoagulation alone or in combination with delayed revascularization generally echo our experience (Table 5). Buchan et al.⁶ treated 6 ICT patients with anticoagulation alone and reported no recurrent

ischemic events and no deaths in late follow up. They treated 3 additional ICT patients with initial anticoagulation followed by delayed CEA and reported no recurrent ischemic events in the interim between initiation of anticoagulation and surgery. One of these 3 patients, however, did suffer a fatal post-CEA stroke. Combe et al.⁸ treated 5 ICT patients and Gonzalez et al.¹¹ treated 3 ICT patients with initial anticoagulation followed by delayed CEA and reported no recurrent ischemic events in the interim between initiation of anticoagulation and surgery and no incidence of post-CEA stroke or death.

Two alternative approaches to the management of patients with ICT and suspected underlying stenosis have been reported – early CEA and early CAS (Table 5). Regarding early CEA, results from NASCET (12% perioperative stroke/death rate for ICT patients vs. 6% perioperative stroke/death rate for non-ICT patients) and a large multicenter retrospective review (14% perioperative stroke rate for ICT patients vs. 5% perioperative stroke rate for non-ICT patients) strongly suggest that this treatment strategy for ICT patients likely carries unacceptably high risk. Smaller single institution case series generally corroborate this conclusion. Biller et al.⁵ treated 5 ICT patients with early CEA and noted a 20% risk of perioperative stroke/death. Walters et al.¹⁹ treated 11 ICT patients with early CEA and noted a 27% risk of perioperative stroke/death. Buchan et al.⁶ treated 14 ICT patients with early CEA and noted a 29% risk of perioperative stroke/death. In contradistinction, Caplan et al.⁷ treated 6 patients with early CEA and noted a 0% risk of perioperative stroke/death.

Regarding early CAS for ICT patients with underlying stenosis, initial results have been more favorable than that reported for early CEA. In the first reported case series, Tsumoto et al.¹⁷ treated 6 ICT patients with early CAS and noted a 17% risk of perioperative stroke/death. However, two subsequent series of 9 ICT patients¹³ and 6 acute stroke patients with ICT¹⁴ noted a 0% risk of stroke/death following early CAS. Taken together, these three small case series^{13,17} and other individual case reports^{12,17} suggest that early CAS for treatment of patients with symptomatic ICT may be a reasonable approach, though the number of reported cases remains small. However, it should be noted that the degree of stenosis in many of these patients maybe minimal once the thrombus has lysed. Our data suggests that anticoagulation for a week to 10 days followed by re-imaging may avoid a revascularization procedure in many patients, with minimal risk of stroke.

In most cases, ICT occurs in the setting of underlying atherosclerotic disease that predisposes to thromboembolism;⁶ however, it has also been reported in the absence of significant atherosclerotic disease. The latter usually occurs in the setting of a hypercoagulable state.^{3,7,9,16,20} In our series, 7 patients had a hypercoagulable condition, the most common of which was thrombocytosis. The formation of ICT in the setting of essential thrombocytosis or anemia with secondary thrombocytosis is rare, with only a few reported cases.^{3,7,9,16,20} Our results with a strategy of anticoagulation for this subset of ICT patients suggest that medical management may be preferred as compared to early CEA or early CAS.

Our study has several limitations. First, this is a retrospective case series with limitations inherent to the study design. Second, follow up angiography to confirm thrombus resolution was not obtained in all patients. Third, the methods and length of anticoagulation employed in our series were varied. Fourth, the concurrent use of antiplatelet therapy during the period of anticoagulation was not uniform. Fifth, the small sample size of our series could have underestimated the true incidence of stroke/death in patients who are treated with our strategy of anticoagulation alone, or anticoagulation followed by delayed revascularization. Given the rarity of this condition, however, such limitations would be difficult to avoid.

In conclusion, this is the largest case series to date demonstrating that patients with symptomatic ICT can be managed safely and effectively via a strategy of initial anticoagulation followed by delayed carotid revascularization (if indicated).

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Figure 1.

Intraluminal Carotid Thrombus (Case 3).

A 60 year old male presented with multiple TIAs. Digital subtraction angiography (DSA) revealed 80% stenosis of the right internal carotid artery and an intraluminal carotid thrombus (ICT) (black arrow) distal to the stenosis (Fig 1A). Anticoagulant therapy was administered for 29 days. He did not experience any ischemic events in the interim. Follow up DSA demonstrated complete resolution of the ICT and 80% residual carotid stenosis (Fig 1B). The patient subsequently underwent carotid endarterectomy. There were no new ischemic events over 87 weeks of follow up.

Patient Characteristics and Presentation (Patients with Carotid Stenosis)

Table 1

Patient	Age (years)	Gender	Presentation	Duration of symptoms	NIHSS	Admission Medications	Comorbidities	Hypercoagulable State?	Current smoker	Previous Stroke/TIA	Angiographic Findings			
											Ipsilateral Stenosis	Site of thrombus	Hemodynamic Effect (Ipsilateral)	Contralateral Stenosis
1	69	M	TIA	4 days	0	Clopidogrel	HTN, HL	No	No	TIA's, Stroke	80%	RICA	Decreased distal flow	68% stenosis
2	78	M	Stroke	1 day	3	None	HL, CAD, previous MI	No	No	Stroke	63%	RICA	No distal flow deficit	<20% stenosis
3	60	M	TIA	4.5 months	0	None	HTN, HL	No	Yes	Stroke	80%	RICA	No distal flow deficit	No stenosis
4	66	M	TIA	1 day	0	Warfarin	HTN, HL, CAD, AF	No	No	No	89%	RICA	Decreased distal flow	No stenosis
5	46	M	Stroke	1 day	4	Aspirin	HTN, HL, DM, CAD	No	No	No	49%	RICA	No distal flow deficit	No stenosis
6	59	F	Stroke	1 day	16	None	HL	No	No	No	70%	RICA	Decreased distal flow	No stenosis
7	47	M	Stroke	1 day	7	None	None	No	Yes	No	99%	LICA	Decreased distal flow	High grade (String sign)
8	57	M	Stroke	1 day		Aspirin	HTN, HL	Anemia+Thrombocytosis	Yes	No	90%	RICA	Decreased distal flow	No stenosis
9	59	M	Stroke	1 day	13	None	DM	No	Yes	No	80%	RICA	Decreased distal flow	No stenosis
10	52	M	TIA	1 day	0	Aspirin	HTN, HL, CAD	No	Yes	No	96%	LICA	Decreased distal flow	No stenosis
11	86	F	Stroke	1 day	2	None	HTN, CAD	No	No	No	78%	RICA	No distal flow deficit	No stenosis
12	74	M	Stroke	1 day	1	None	HL	No	No	No	50%	LICA	No distal flow deficit	No stenosis
13	39	M	Stroke	1 day		None	None	No	No	No	63%	RICA	Decreased distal flow	Minimal stenosis
14	57	M	Stroke	1 day	14	None	HTN	No	No	TIA	99%	RICA	Decreased distal flow	95-99%
15	81	F	Stroke	1 day	1	None	HTN, DM	No	No	No	99%	LCCA+LICA	Decreased distal flow	Minimal stenosis
16	46	M	Stroke	5 days	3	Aspirin	HL, DM	No	Yes	No	55%	LICA	Decreased distal flow	No stenosis
17	52	M	Stroke	1 day	1	None	HL	No	Yes	No	70%	LICA	Decreased distal flow	No stenosis

Table 2

Patient Characteristics and Presentation (Patients without Carotid Stenosis)

Patient	Age (years)	Gender	Presentation	Duration of symptoms	NIHSS	Admission Medications	Comorbidities	Hypercoagulable State?	Current smoker	Previous Stroke/TIA	Angiographic Findings			Contralateral Stenosis
											Ipsilateral Stenosis	Site of thrombus	Hemodynamic Effect (Ipsilateral)	
18	51	F	TIA	2 days	0	None	HTN	Anemia + Thrombocytosis	No	No	No stenosis	RICA	No distal flow deficit	No stenosis
19	25	M	Stroke	1 day	10	None	None	Thrombocytosis	No	No	No stenosis	LICA	No distal flow deficit	No stenosis
20	31	F	Stroke	2 days	1	None	HTN, DM	Anemia + Thrombocytosis	No	No	No stenosis	LCCA	Decreased distal flow	No stenosis
21	72	M	TIA	1 day	4	Aspirin	HTN, HL, CAD	No	No	Stroke, TIAs	No stenosis (previous CEA)	RICA	No distal flow deficit	35%
22	36	F	Stroke	1 day	1	None	None	Anemia + Thrombocytosis	Yes	Stroke	No stenosis	LICA	No distal flow deficit	No stenosis
23	49	F	Stroke	2 days	7	None	SLE	SLE with Thrombocytosis	No	No	No stenosis	Both ICAs	Decreased distal flow	No stenosis
24	43	F	Stroke	1 day	1	Aspirin	HTN	APS	No	No	No stenosis	LICA	No distal flow deficit	No stenosis

Table 3

Treatment and Follow Up (Patients with Carotid Stenosis)

Patient	Initial Treatment			Interim events			Follow up Imaging			Subsequent Treatment	Interval between Revascularization and Presentation (days)	Follow up Duration (weeks)	Final Outcome
	Anticoagulation	Duration (days)	Antiplatelets				Modality	ICT	Residual Stenosis				
1	LMWH	28	None, Clopidogrel discontinued	TIA			DSA	Near complete resolution	90%	CEA	29	74.5	No new ischemic events
2	Heparin	7	Aspirin	None			DSA	Resolved	62%	CAS recommended-patient declined	N/A	9.4	No new ischemic events
3	Heparin, Warfarin	23	Aspirin	None			DSA	Resolved	80%	CEA	29	86.8	No new ischemic events
4	Heparin, Warfarin	14	Aspirin, Clopidogrel	None			DSA	Resolved	95%	CAS	15	85	No new ischemic events
5	Heparin, Warfarin	223	Aspirin	None			CTA	Resolved	<50%	None	N/A	89.3	No new ischemic events
6	Heparin	12	Aspirin	None			DSA	Resolved	85%	CEA	19	121.4	No new ischemic events
7	LMWH	22	Aspirin	None			None	N/A	N/A	None	N/A	9.5	No new ischemic events
8	Heparin, LMWH, Warfarin	106	Aspirin	None			None	N/A	N/A	None	N/A	15.1	No new ischemic events
9	Heparin, Warfarin	42	Aspirin	None			None	N/A	N/A	None	N/A	6	No new ischemic events
10	Heparin, LMWH, Warfarin	40	Aspirin	None			DSA	Resolved	88%	CEA	41	7	No new ischemic events
11	Heparin	7	Aspirin, Clopidogrel	None			DSA	Resolved	80%	CAS	43	90.2	No new ischemic events (Died due to breast cancer related complications)
12	Heparin, Warfarin	42	Aspirin	None			None	N/A	N/A	CEA	43	30.8	No new ischemic events
13	Warfarin	203	Aspirin	None			None	N/A	N/A	None	N/A	368	No new ischemic events
14	Heparin, Warfarin	53	Aspirin	None			DSA	Resolved	60%	CEA	91	97.5	No new ischemic events
15	LMWH, Warfarin	318	Aspirin	None			DSA	Resolved	Minimal	None	N/A	45.4	No new ischemic events

Patient	Initial Treatment			Interim events	Follow up Imaging			Subsequent Treatment	Interval between Revascularization and Presentation (days)	Follow up Duration (weeks)	Final Outcome
	Anticoagulation	Duration (days)	Antiplatelets		Modality	ICT	Residual Stenosis				
16	Heparin, LMWH	42	Aspirin	None	DSA	Resolved	62%	CEA	43	21.3	No new ischemic events
17	Heparin, LMWH, Warfarin	39	None	None	DSA	Resolved	97%	CEA	40	6	No new ischemic events

Table 4

Treatment and Follow Up (Patients without Carotid Stenosis)

Patient	Initial Treatment				Follow up Imaging			Subsequent Treatment	Follow up Duration (weeks)	Final Outcome
	Anticoagulation	Duration (days)	Antiplatelets	Interim events	Modality	ICT	Residual Stenosis			
18	Heparin, Warfarin	215	None	None	None	N/A	N/A	None	30.7	No new ischemic events
19	Heparin, LMWH, Warfarin	35	Aspirin	None	None	N/A	N/A	None	5	No new ischemic events
20	Heparin, Warfarin	12	None	None	MRA	Resolved	None	None	56	No new ischemic events
21	LMWH, Warfarin		Aspirin	None	None	N/A	N/A	None	Lost	
22	LMWH, Warfarin	46	Aspirin	None	None	N/A	N/A	None	348	No new ischemic events
23	Heparin, Warfarin	61	Aspirin	None	None	N/A	N/A	None	8.7	No new ischemic events
24	Heparin, Warfarin	50	None	None	None	N/A	N/A	None	7.1	No new ischemic events

Summary of Literature

Table 5

Treatment Strategy	Study/Year	Type of Study	Number of ICT patients	Complications (Stroke or Death at 1 month)
Antiplatelet therapy	NASCET - Medical Arm ³	Multicenter Prospective RCT	28	11%
CEA *	NASCET - Surgical Arm ³	Multicenter Prospective RCT	25	12%
	Goldstein et al. ⁷ /1994	Multicenter Retrospective	28	14%
Early CEA †	Caplan et al. ⁵ /1984	Single Center Retrospective	6	0%
	Billar et al. ⁸ /1986	Single Center Retrospective	5	20%
	Walters et al. ⁶ /1987	Single Center Retrospective	11	27%
	Buchan et al. ¹ /1988	Single Center Retrospective	14	29%
Early CAS	Tsumoto et al. ¹⁶ /2006	Single Center Retrospective	6	17%
	Jahromi et al. ⁹ /2008	Single Center Retrospective	9	0%
	Kwon et al. ¹⁰ /2010	Single Center Retrospective	6	0%
Initial anticoagulation + Delayed Revascularization	Buchan et al. ¹ /1988	Single Center Retrospective	3	33%
	Combe et al. ¹¹ /1990	Single Center Retrospective	5	0%
	Gonzalez et al. ¹² /2004	Single Center Retrospective	3	0%
	<i>Present study</i>	<i>Single Center Retrospective</i>	<i>10</i>	<i>0%</i>
Anticoagulation without Delayed Revascularization	Buchan et al. ¹ /1988	Single Center Retrospective	6	0%
	<i>Present study</i>	<i>Single Center Retrospective</i>	<i>14</i>	<i>0%</i>

* CEA < 4 months after ischemic event;

† < 2 weeks after ischemic event