Safety of carotid endarterectomy following thrombolysis for acute ischemic stroke

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Background: The timing of carotid endarterectomy (CEA) following thrombolysis for acute ischemic stroke remains controversial. We have described our unit’s experience and performed a systematic review and pooled data synthesis of the safety of CEA in this group of patients.

Methods: Retrospective analysis of patients who had undergone CEA following thrombolysis between 2010 and 2012 was performed. A systematic review of the literature was also performed using PUBMED, EMBASE, and major conference proceedings. The primary outcome measure was 30-day postoperative intracerebral hemorrhage (ICH) and ischemic stroke. The secondary outcome measures were 30-day postoperative all-cause morbidity and mortality.

Results: Seven patients underwent CEA following thrombolysis with mean age ± standard deviation of 70.57 ± 7.57. Five were men and five had CEA under local anesthesia. CEA was performed in median of 7 days (range, 2-12) after onset of stroke. Thirty-day morbidity was 14% (1/7) with one patient who experienced ICH postoperatively. There was no mortality. Nine studies were identified for systemic review. Seventy patients were included in the pooled data synthesis. One patient (1%) experienced postoperative ICH. When our series was included, the 30-day postoperative ICH was 3% (2/77). The overall 30-day postoperative morbidity was 4% (3/77), and there was no mortality.

Conclusions: Despite slightly higher rates of postoperative hemorrhagic strokes than those in randomized trials, CEA appears safe following thrombolysis for acute ischemic stroke. However, more data on the timing of surgery is needed. (J Vasc Surg 2013;58:1671-7.)
body mass index, comorbidities, medications, and modified Rankin scale (mRS) on discharge.

Systematic thrombolysis with recombinant tPA was given up to 4.5 hours from the onset of stroke, in accordance to the national guidelines and randomized controlled trials. All patients have had CT of the brain prior to and 24 hours after thrombolysis to exclude ICH. ICH is defined as any apparent extravascular blood in the brain or within the cranium that was associated with clinical deterioration, as defined by an increase of four points or more in the score on the NIHSS or that led to death and that was identified as the predominant cause of the neurologic deterioration.

The degree of carotid stenosis was then determined by duplex ultrasound according to the North American Symptomatic Carotid Endarterectomy Trial (NASCET) criteria, and patients were referred to the vascular unit if the degree of carotid stenosis was significant (>50%). Decision for surgery was made by the consultant vascular surgeon on-call, and all patients were started on aspirin preoperatively (300 mg of loading dose, followed by 75 mg daily). The patients did not receive any anticoagulation prior to CEA. CEA was performed in the next available operating list following vascular referral. All patients were operated on the ipsilateral symptomatic internal carotid artery. Five patients were initially operated under local anesthesia, and two patients had general anesthetic with invasive blood pressure monitoring both intra- and postoperatively (for at least 2 hours) to maintain an acceptable range of blood pressure. All patients received between 3000-5000 units of heparin intraoperatively only depending on body weight. Patients were followed up at 6 weeks.

Systematic review

Search strategy. A literature review was undertaken for studies published between 1960 and August 2012 on the safety of CEA following thrombolysis for acute ischemic stroke. An electronic search on MEDLINE and EMBASE was performed using prespecified keywords: “carotid endarterectomy thrombolysis,” “carotid intervention thrombolysis,” and “carotid surgery thrombolysis.” The reference list of each article was also searched by hand. The electronic search was supplemented by a hand-search of published abstracts from meetings of the Vascular Society of Great Britain and Ireland, the Society for Vascular Surgery, the Association of Surgeons of Great Britain and Ireland, the European Society for Vascular Surgery, European Stroke Conference, World Stroke Congress, and International Stroke Conference.

Selection criteria. We included only articles published in English with data on patients who have had CEA following an acute ischemic stroke with significant carotid stenosis, after either intravenous or intra-arterial thrombolysis. Articles published in non-English language, or those on carotid thrombectomy or embolectomy and intraoperative thrombolysis were excluded (Fig). Selection of articles was performed independently by two of the authors (Y.Y., N.A.), and the discrepancies were resolved by consensus.

Outcome measures. The primary outcome was the incidence of 30-day postoperative ICH and ischemic stroke. The secondary outcome was 30-day postoperative all-cause morbidity and mortality.

Significant carotid stenosis was defined as ipsilateral >50% stenosis as determined by ultrasound, CT, magnetic resonance imaging (MRI), or cerebral angiography. Thrombolysis was defined as administration of thrombolytic agent within the time frame determined by local guidelines for the treatment of acute ischemic stroke, following exclusion of cerebral hemorrhage on brain imaging. Timing of CEA was defined as day(s) between onset of symptoms and CEA. Postoperative stroke was defined as new neurologic deficit lasting more than 24 hours postoperatively.

The data from eligible papers were abstracted in to a Microsoft Excel spread sheet. In cases where data were not available, the corresponding author was contacted by e-mail for clarification and request of additional data. However, only limited number of authors responded to enquiry.

RESULTS

Single-center review

A total of 214 patients received intravenous thrombolysis with alteplase between July 2010 and May 2012 for acute ischemic stroke. However, data was only available for 213 patients. The 30-day post-thrombolysis mortality was 12% (26/213). All patients received CT of the brain, of which 78% (167/213) had evidence of infarction. Nine percent of patients (19/213) subsequently received MRI of the brain (because of recurrent symptoms and the absence on acute infarction on CT), and all had evidence of infarction. On duplex ultrasound of the symptomatic carotid artery, 67% (142/213) had insignificant carotid stenosis, 6% (12/213) had occluded internal carotid artery (ICA), and 9% (19/213) had significant carotid stenosis. Among these patients with significant ipsilateral carotid stenosis, eight (4%, five male, mean age 70.6 ± 7) were referred to the vascular unit in Nottingham and seven (five male, mean age 70.57 ± 7.57) subsequently underwent CEA. One patient had a delayed CEA 71 days after the onset of index event because of acute myocardial infarction shortly after ischemic stroke. We have therefore excluded this patient from the analysis.

The rest of the 11 patients who had carotid stenosis did not receive CEA because of complications post-thrombolysis (one patient), refused surgery (one patient), and unfit for surgery (six patients, admitted with disabling stroke with unimproved neurologic status following systematic thrombolysis). None had further symptoms at median follow-up of 24.5 months (range, 10-31). One died 10 months after index event because of pneumonia, distal ICA stenosis (two patients), and referral to other vascular unit for intervention (one patient). Demographics, neurologic presentations, comorbidities, and risk factors are shown in Table I.

The median NIHSS score before and after thrombolysis was 15 (range, 10-22) and 11 (range, 2-18). CT of the
brain following thrombolysis showed that five patients had small subacute infarction, one had no evidence of acute infarction, and one had a small subacute infarction with small patches of petechial hemorrhage. CEA was performed on a median of 7 days (range, 2-12) from the onset of symptoms by a consultant vascular surgeon. Five patients were operated under local anesthesia, one under general anesthesia, and one was converted from local to general anesthesia because of agitation.

Of all patients who have subsequently received CEA, one (14%, patient number 5, Table II) suffered postoperative ICH on the same day following surgery. The patient became hypertensive toward the end of CEA. This was managed medically, and a subsequent CT head demonstrated an ICH at the point of previous infarct suggestive of a reperfusion injury. There was no 30-day postoperative recurrent ischemic stroke, morbidity, or mortality. The median mRS on discharge was 2 (range, 0-4).

**Systematic review**

A total of 14 articles were identified as potential source of information on the safety of CEA following thrombolysis for acute ischemic stroke. Four articles were excluded during the screening for abstract and language. Two articles were published in non-English language, one article performed intraoperative thrombolysis, and one article performed CEA and embolectomy in complete ICA occlusion. After assessing the remaining nine articles in full text, a further article was excluded for performing carotid embolectomy post-thrombolysis. A total of nine articles that consists of eight full text articles and one conference abstract were included in quantitative analysis. The author of the conference proceeding has been contacted. However, they did not publish their results. Seventy patients were identified in nine studies (eight retrospective and one prospective; Table III). A summary of systematic review is presented in Table IV.

**Preoperative management.** In addition to the initial diagnostic brain imaging, Lescche et al performed MRI of the brain if the time from onset of the initial symptoms was within 3 hours. Four studies published in non-English language, one article performed intraoperative thrombolysis, and one article performed CEA and embolectomy in complete ICA occlusion. After assessing the remaining nine articles in full text, a further article was excluded for performing carotid embolectomy post-thrombolysis. A total of nine articles that consists of eight full text articles and one conference abstract were included in quantitative analysis. The author of the conference proceeding has been contacted. However, they did not publish their results. Seventy patients were identified in nine studies (eight retrospective and one prospective; Table III). A summary of systematic review is presented in Table IV.

**Table I.** Baseline characteristics on admission to stroke unit for patients who have subsequently undergone carotid endarterectomy (CEA) following thrombolysis

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<th>HCL</th>
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AF, Atrial fibrillation; anti-BP, antihypertensive; BMI, body mass index; COPD, chronic obstructive pulmonary disease; DM, diabetes mellitus; Ex, ex-smoker; HCL, hypercholesterolemia; HTN, hypertension; IHD, ischemic heart disease; L, left; LAC, lacunar syndrome; N, no; PAC, partial anterior circulation syndrome; R, right; TAC, total anterior circulation syndrome; TIA/stroke, previous transient ischemic attack or stroke; Warf, warfarin; Y, yes.

*Type of stroke = stroke classification according to the Oxfordshire classification of stroke.
NIHSS score after thrombolysis. Bartoli et al. used the Score de l’Unite Neuro-Vasculaire after thrombolysis (median of 0, range, 0-1), which is a simplified version of the NIHSS score at the local center.

Six studies performed intravenous thrombolysis with rtPA, and two performed intra-arterial thrombolysis. Moreover, McPherson et al. performed intra-aortic thrombolysis with rtPA in patients with NIHSS >10 after the initial thrombolysis. Following the initial thrombolysis, Bartoli et al. reported that all patients had unfractonated heparin infusion 24 hours later until CEA, and McPherson et al. reported that three patients received intravenous heparin infusion.

**Patient selection criteria.** Although currently there is no general consensus on patient selection criteria for CEA following thrombolysis, few authors have reported their institutional protocol. Bartoli et al. have included patients with fully recanalized cerebral arteries, absence of hemorrhagic transformation or cerebral edema, stable neurologic status, and presence of ischemic infarct inferior to one-third of the middle cerebral artery (MCA) territory on brain imaging. Eckstein et al. included all patients without cerebral coma and ICH. Leseche et al. reported the contraindications for early CEA in patients with major disabling stroke (mRS >4), impaired consciousness, ischemic infarct of one-third or more of the MCA territory on brain imaging and hemorrhagic transformation or cerebral edema. Uno et al. included patients without cardiac arrhythmia or valvular heart disease, absence of new infarct on brain imaging, and presence of collateral circulation visualized on cerebral angiograms.

**CEA.** On the timing of CEA, results for individual patients were only available in five studies with a median of 5 days (range, 1-54 days). Crozier et al. reported a median of 8 days (range, 2-23), Ratheonborgh et al. reported a median of 11 days (interquartile range, 5-12) and Shalhoub et al. reported on the timing of CEA from thrombolysis. All three studies above did not publish the results for individual patient. Leseche et al. did not publish the timing of CEA in patients who received surgery following thrombolysis.

| Table II. Operative data of patients who have undergone carotid endarterectomy (CEA) following thrombolysis for acute ischemic stroke |
|------------------|------------------|------------------|------------------|------------------|------------------|------------------|
| **Patient** | **NIHSS pretrombolysis** | **NIHSS post-thrombolysis** | **Ipsilateral stenosis, %** | **Contralateral stenosis, %** | **Timing of CEA, day** | **Anesthesia Shunt Postop ICH mRS on discharge** |
| 1 | 16 | 3 | 80%-99% | <30 | 10 | LA N N 0 |
| 2 | 15 | 12 | 50%-59% | <30 | 11 | LA N N 3 |
| 3 | 15 | 11 | 60%-69% | <30 | 3 | LA N N 0 |
| 4 | 11 | 2 | 60%-69% | <30 | 7 | LA N N 2 |
| 5 | 10 | 10 | 70%-79% | 60-69 | 2 | LA N Y 4 |
| 6 | 18 | 18 | 60%-69% | <30 | 12 | Convert Y N 2 |
| 7 | 22 | 16 | 60%-69% | 80-95 | 7 | GA Y N 3 |

Convert: Converted from local to general anesthesia; GA, general anesthesia; ICH, intracerebral hemorrhage; LA, local anesthesia; mRS, modified Rankin scale; N, no; NIHSS, National Institutes of Health Stroke Scale; Y, yes.

| Table III. Data of eight series included in pooled data analysis and systematic review |
|------------------|------------------|------------------|------------------|------------------|------------------|
| **1st author, Study No. (M)** | **Mean age (range)** | **NIHSS on admission a** | **Degree of stenosis, %** | **Timing of CEA, days a** | **Postop morbidity** | **Postop ICH** | **mRS a** |
| Endo, 1998 8 R 1 (1) | 73 | >24 | NA | IA | 0 | 0 | 0 | 1 at 1 month |
| Eckstein, 1999 10 P 3 (2) | 51-75 | NA | >70 | IV | 14 (6-21) | 0 | 0 | 1 (0-3) at 6 months |
| Uno, 2001 11 R 4 (4) | 69 (64-77) | NA | NA | IA | 13.5 (1-54) | 0 | 0 | NA |
| McPherson, 2001 12 R 5 (4) | 59 (45-72) | 13 (6-24) | 99-100 | IV & IA | 1 (1-2) | 0 | 0 | NA |
| Bartoli, 2009 13 R 12 (10) | 60 (49-79) | 12 (5-21) | 50-99 | IV | 8 (1-16) | 0 | 1 | 1 (1-3) at 3 months |
| Crozier, 2010 14 R 10 (3) | 66 (50-89) a | NA | >65 | IV | 8 (2-23) | 3 | 0 | NA |
| Shalhoub, 2011 8 R 6 (4) | 69 (51-75) a | NA | NA | IV | 5.5 (4-9) a | 0 | 0 | NA |
| Leseche, 2012 15 R 7 (NA) | NA | 9 (5-15) | >60 | IV | NA | 0 | 0 | NA |
| Rathenbourgh, 2012 16 R 22 (7) | NA (52-74) | NA | 50-99 | IV | 11 (5-12) a | 0 | 0 | NA |

IA, Intra-arterial; ICH, intracerebral hemorrhage; IV, intravenous; M, male; mRS, modified Rankin Scale following discharge; NA, not available; NIHSS, National Institutes of Health Stroke Scale; P, prospective; R, retrospective.
aMedian (range).
bTiming of CEA from thrombolysis.
cMedian (interquartile range).
Following CEA, only two studies reported on postoperative care. Bartoli et al\textsuperscript{13} reported that caution was taken to prevent hypertension, and rehabilitation was commenced with gradual verticalization to prevent hemodynamic disturbance of the brain. Leseche et al\textsuperscript{15} maintained patients’ blood pressure $\leq 130/70$ mm Hg and commenced them with aspirin and/or clopidogrel on discharge.

**Thirty-day postoperative ICH and morbidity.** The 30-day postoperative morbidity was only reported by Crozier et al\textsuperscript{14} where three patients experienced bleeding from wound, wound hematoma, and postoperative confusion, respectively, which were all resolved prior to discharge. While assessing the methods and results section of other manuscripts, some did not report on the presence or absence of postoperative morbidity. We therefore assumed the lack of reporting meant that it did not occur. Overall, one patient (1%) in the pooled data analysis experienced postoperative ICH within 30 days. When we include our series in the pooled data analysis, the 30-day postoperative ICH was 3% (2/77). There was no 30-day postoperative recurrent ischemic stroke or mortality.

**Follow-up.** Endo et al\textsuperscript{9} followed-up patients at 3 to 6 months with MRI angiography, Bartoli et al\textsuperscript{13} at 3 months with duplex ultrasound, and Leseche et al\textsuperscript{15} at 1 and 3 months with duplex ultrasound as well as CT or MRI of the brain at 3 months. No patients had experienced recurrent stroke until the follow-up period.

**DISCUSSION**

The main concern on performing CEA after thrombolysis is whether the administration of thrombolytic agent, in combination of antiplatelet therapy and early CEA increase the risk of postoperative ICH. Although this is a small study, it is the first systematic review of the safety of CEA following thrombolysis for acute ischemic stroke in the literature. The findings of the present study revealed a postoperative ICH of 3%, slightly higher than the reported incidence of ICH in patients without thrombolysis (0.3%-1.2%).\textsuperscript{16-23} However, the limited number of patients and the nature of this study preclude any statistical analysis. In a review of ICH following CEA in patients without thrombolysis, the median duration from surgery to ICH was 4 days (range, intraoperative to 25 days).\textsuperscript{24} It was thought that following an ischemic infarction, there is a critical period in which an increase in cerebral perfusion pressure could lead to ICH adjacent to the infarct.\textsuperscript{25}

Several studies have investigated the risk factors of ICH after CEA in patients without thrombolysis. Common risk factors that were identified include a higher degree ($>90\%$) of ipsilateral carotid stenosis, limited hemispheric collateral flow, and hyperperfusion.\textsuperscript{21,23,24,26} Other risk factors were younger patients,\textsuperscript{26} hypertension,\textsuperscript{26} anticoagulation therapy,\textsuperscript{23} and perioperative ischemic event.\textsuperscript{23} In contrary, Russell et al\textsuperscript{24} reported that patients who suffer ICH are mostly normotensive, and it was difficult to know whether hypertension plays a role in the pathogenesis of ICH after CEA or if it is a normal response to a sudden rise in intracranial pressure. The cause of ICH is proposed to be cerebral hyperperfusion syndrome, which can be explained by the existence of a high grade carotid stenosis based on the normal pressure breakthrough theory.\textsuperscript{27} Chronic cerebral hemispheric hyperperfusion syndrome secondary to extracranial carotid stenosis causes compensatory intracranial vasodilation and therefore loss of autoregulation. Following CEA, normal
arterial flow at systolic pressure resumed in the ipsilateral hemisphere. The sudden increase of flow and pressure in the maximally dilated cerebral vasculature leads to hyperemia. These result in the clinical symptoms of cerebral hyperperfusion syndrome and may evolve into ICH when the cerebral vessels rupture. Therefore, several authors have advocated the prevention of postoperative hypertension to minimize the risk of ICH.21,23,24,26 Furthermore, perioperative ischemic events might also increase the risk of ICH when the fragmentation of emboli or relaxation of vascular spasm causes the embolus to move distally. This subsequently exposes the previously ischemic tissue to systolic blood pressure and, hence, hemorrhages from the injured capillaries.28

Comparing the two patients that suffered postoperative ICH in the present study, hypertension was a common risk factor. The patient in the Nottingham series, who had a normal preoperative pressure, experienced intraoperative and postoperative hypertension with a blood pressure of up to 200/100 mm Hg and 160/70 mm Hg, respectively. She had an ipsilateral carotid stenosis of 70%-79% and contralateral stenosis of 60%-69%. Our unit’s protocol was that all patients with hypertension post-CEA should be treated initially with labetalol, follow by intravenous beta-blocker infusion in refractory cases with close monitoring in high dependency or intensive care unit. Despite the blood pressure being managed with aggressive medical management, ICH with midline shift of 3 mL was confirmed on CT of the brain the same day after CEA. The other patient was reported by Bartoli et al13 to have a systolic blood pressure of 180 mm Hg postoperatively. She had an ipsilateral carotid stenosis of 90% without any stenosis on the contralateral side. Initial CT angiography showed that she had ipsilateral occlusion of the M1 segment of the MCA and intracranial internal carotid artery. However, the extent of ICH and the presence of midline shift were not reported. The European Cooperative Acute Stroke Study investigators suggested that only parenchymal hematomas with substantial space-occupying effect covering more than one-third of the infarcted tissue volume were associated with an increased risk of early deterioration, disability, and death.29

In addition to the aforementioned risk factors, patient selection is an important factor in determining surgical outcome. Besides the apparent exclusion of disabling stroke and ICH prior to surgery, some studies have suggested that patients should have minimal evidence of ischemic infarction on brain imaging.10,15 Three authors have reported the findings of brain imaging9–11 as minimal or small ischemic infarction. Nonetheless, there is no general consensus on the definition of minimal infarction. Furthermore, Eckstein et al9 suggested that simultaneous occlusion of the MCA to be an important factor in patient selection. In a study where 34 patients with acute ICA occlusion have undergone emergency CEA, nine patients were found to have thrombus in the MCA seen on angiography. These patients either died from severe infarction or hemorrhage, or performed poorly in postoperative period.30

The timing of CEA remains the key to successful reduction in the risk of recurrent ischemic stroke. There is robust evidence to suggest that patients will gain most benefit when CEA is performed within 14 days from the onset of symptoms.1 However, several authors7,8,10,11 have performed CEA beyond the 14-day time frame and two studies7,8 were published before the evidence for early CEA was published.1

There are several limitations to this study. It should be emphasized that we are focusing on a highly selected subgroup of patients who have received CEA following successful thrombolysis. Furthermore, the number of patients who received CEA after thrombolysis for acute ischemic stroke remains small, with a reported rate of 2%-6% in the literature.11,13,14 The largest series of patients having CEA after thrombolysis for acute ischemic stroke was reported by Rathenborg et al7 (n = 22). Secondly, data such as the time from onset of symptoms to thrombolysis, pre- and post-thrombolysis CT of the brain, pre- and post-thrombolysis NIHSS score, the degree of ipsilateral and contralateral stenosis, surgical technique, and mRS from series included were only partially available. Lastly, this is a systematic review of case series, which inevitably suffers from methodological quality, selection, and reporting bias, in which postoperative morbidities may have been under reported.

In conclusion, CEA following thrombolysis for acute ischemic stroke appears to be safe, with a relatively low risk of postoperative ICH. It could potentially be performed according to the current guideline on CEA in patients with minor stroke and transient ischemic attack.5 ICH after CEA following thrombolysis seems to be a trade-off for a lower risk of recurrent ischemic stroke, and it may never be able to be avoided entirely. A registry would be required to refine patient selection criteria and to identify complications as well as patients who are at high risk of postoperative ICH.

AUTHOR CONTRIBUTIONS
Conception and design: YY, JS, SM, NA
Analysis and interpretation: YY, KV
Data collection: YY, NA
Writing the article: YY, JS, SA, NS, KV, SM
Critical revision of the article: YY, JS, SA, NS, KV, SM, NA
Final approval of the article: YY, JS, SA, NS, KV, SM, NA
Statistical analysis: YY, KV
Obtained funding: Not applicable
Overall responsibility: NA

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Submitted Mar 13, 2013; accepted May 21, 2013.