Frequency and Clinical Course of Stroke and Transient Ischemic Attack Patients With Intracranial Nonocclusive Thrombus on Computed Tomographic Angiography

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Background and Purpose—We sought to determine the frequency and clinical course of patients with acute ischemic stroke or transient ischemic attack (TIA) who had intracranial nonocclusive thrombus (iNOT) on CT angiography (CTA).

Methods—We retrospectively (June 2002–March 2007) reviewed consecutive patients with acute ischemic stroke or TIA who had CTA performed acutely for diagnostic work-up. A neuroradiologist reviewed all cases with potential iNOT. Criteria to diagnose iNOT rather than occlusive thrombus or atherosclerotic stenosis were: (1) residual lumen present and eccentric; (2) nontapering thrombus; (3) smooth and well-defined thrombus margins; and (4) absence of vessel wall calcification. We defined functional independence at discharge as modified Rankin scale score ≤2.

Results—Of 865 patients, 23 (2.7%) exhibited iNOT on CTA (43% women, mean age 69±14 years, median National Institute of Health Stroke Scale score 3 [range, 0–23]; median onset-to-CTA time 3.5 hours [range, 0.9–75]). Four patients (17%) deteriorated clinically during the hospital course and had persistent new focal neurological deficits. All of them were functionally dependent at discharge. All 19 patients (83%) without persistent clinical deterioration (2 patients had recurrent TIAs) were functionally independent at discharge.

Conclusion—Intracranial nonocclusive thrombus on CTA is relatively uncommon. The majority of patients have a good clinical outcome. However, some patients deteriorate clinically and are functionally dependent at discharge.

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Key Words: acute ■ computed tomography angiography ■ stroke ■ thrombus ■ transient ischemic attack

In acute ischemic stroke, the presence of an intracranial arterial occlusion is associated with poor functional outcome and frequently prompts recanalization strategies.1–3 In patients presenting with minor or resolving symptoms, intracranial arterial occlusion and stenosis indicate high risk for stroke recurrence and deterioration after clinical improvement.4–6 Furthermore, a significant number of patients who are considered ineligible for thrombolysis because their symptoms are too mild or are rapidly improving will be functionally dependent or die because of clinical deterioration.6–8

CT angiography (CTA) is increasingly used to determine extracranial and intracranial vascular status in patients presenting with acute ischemic stroke or transient ischemic attack (TIA).9–12 Intracranial nonocclusive thrombi (iNOT) have been described on digital subtraction angiography or magnetic resonance angiography.13,14 These thrombi seem to be rare but may be more frequently diagnosed when performing early CTA in patients presenting with acute cerebrovascular ischemia. Particularly in patients with minor symptoms, iNOT may indicate increased risk for clinical deterioration. We present our experience with iNOT on CTA in patients with acute ischemic stroke or TIA.

Subjects and Methods

Patients

We retrospectively (June 2002–March 2007) analyzed consecutive patients with acute ischemic stroke or TIA who received CTA of the circle of Willis as part of their diagnostic work-up. Noncontrast CT (NCCT) is the default imaging modality in our stroke center. NCCT is followed by CTA in most, but not all, patients. Decision to proceed to CTA is at the discretion of the treating stroke neurologist based on baseline renal function and the clinical scenario.

For this study, we excluded patients with intracranial hemorrhage on baseline NCCT or if CTA was not performed for suspected cerebrovascular ischemia. We identified all patients with potential iNOT from CTA reports. CT angiograms of these patients were not...
reviewed by a neuroradiologist (M.G.). Neuroradiological criteria to verify iNOT and differentiate from occlusive thrombus or atherosclerotic stenosis were: (1) residual lumen present and eccentric; (2) non-tapering thrombus; (3) smooth and well-defined thrombus margins; and (4) absence of vessel wall calcification (Figure 1). To clearly characterize the clinical course of patients with iNOT, we excluded patients from analysis of iNOT if atherosclerotic stenosis or occlusive thrombus were thought likely or if iNOT and additional occlusive thrombus were present. Patients without verified iNOT were categorized as patients with an intracranial occlusion and patients without an intracranial occlusion on CTA according to the neuroradiologists’ report.

Clinical baseline variables including National Institute of Health Stroke Scale (NIHSS) score are recorded prospectively in the patient record. In cases in which these scores were unavailable, they were derived retrospectively. We defined neurological deficits at baseline as nondisabling if associated with an NIHSS score ≤5 and disabling if associated with an NIHSS score >5. Clinical deterioration during the hospital course was defined as any increase from baseline NIHSS score to the discharge NIHSS score or to the 24-hour NIHSS score using the last score carried forward method. We assessed functional outcome at discharge using the modified Rankin Scale score and defined functional independence as an modified Rankin Scale score ≤2. The local institutional ethics committee approved this study.

Imaging
We obtained CTA with a helical scan technique on a multislice CT scanner (GE Medical Systems or Siemens). Acquisitions were obtained after a single bolus intravenous (IV) contrast injection of 90 to 120 mL nonionic contrast media into an antecubital vein at 3 to 5 mL/sec. Imaging was auto-triggered by the appearance of contrast material in the ascending aorta. Minimum coverage was from foramen magnum to centrum semiovale with 0.6- to 1.25-mm slice thickness. Multiplanar volume-reformatted images were immediately created by the CT technologist with 2.5- to 4.0-mm slice thickness in axial, sagittal, and coronal planes. This image reformatting was typically completed within minutes and available for review.

Image Analysis
A neuroradiologist (M.G.) reviewed source images and reformatted images of patients with potential iNOT (as stated in the CTA reports) at a digital workstation with a large high-resolution monitor. In patients with verified iNOT, we classified the degree of the resulting stenosis at the site of maximal luminal narrowing as ≥50% or <50% by Warfarin-Aspirin Symptomatic Intracranial Disease criteria.15 When severe preocclusive stenosis made it too difficult to perform an accurate measurement, we classified stenoses as near occlusive.

For repeated CTA, follow-up digital subtraction angiography, or magnetic resonance angiography, we classified iNOT status as unchanged or enlarged versus diminished or resolved as stated in the neuroradiology report. For transcranial Doppler sonography, we compared baseline with follow-up mean flow velocities and classified iNOT as diminished or resolved if mean flow velocities decreased by ≥30 cm/sec.16

Statistical Analysis
Data are reported using standard descriptive statistics. We used the Fisher exact test for comparison of proportions, Student t test to compare means, Kruskal-Wallis test to compare ordinal variables across groups, and 1-way ANOVA to compare means across groups. We considered P<0.05 as statistically significant. All tests were 2-sided.

Results
Patients
Of 865 consecutive patients who received CTA for acute ischemic stroke or TIA, 30 patients had potential iNOT as stated in the CTA report. Of these, no residual lumen was present in 3 patients, no definite intraluminal thrombus was noted in 3 patients, and iNOT (basilar artery) and additional occlusive thrombus (posterior cerebral artery) were present in 1 patient. We therefore verified isolated iNOT in 23 of 865 patients (2.7%). Six hundred and three patients (69.7%) had no intracranial occlusion and 239 patients (27.6%) had an intracranial occlusion on CTA. Patient characteristics according to vascular status are summarized in Table 1. In contrast to patients with an intracranial occlusion, the majority of patients without an intracranial occlusion or with iNOT had nondisabling baseline deficits (P<0.001).

Characteristics of Patients With iNOT
Details of patients with iNOT are summarized in Table 2. Nonocclusive thrombi were all located in anterior circulation. Three cases involved the internal carotid artery distally. Twenty cases involved the middle cerebral artery (MCA) equally distributed in the M1 segment (10 cases) and M2 segment (10 cases). We treated 2 patients (patients 7 and 8; Table 2) with IV alteplase (tPA) 110 minutes and 250 minutes from symptom onset, respectively. Thrombolysis was withheld because of minor symptoms, clinical improvement, late presentation with well-defined early ischemic changes on NCCT, or medical contraindications in all further patients with iNOT who were treated with various single, dual, or triple antithrombotic regimens consisting of aspirin, clopidogrel, or IV heparin, as indicated by the treating stroke neurologist (Table 2).

Overall Clinical Course and Functional Outcome
Overall at discharge, 483 of 603 patients (80%) without an intracranial occlusion, 80 of 239 patients (33%) with an intracranial occlusion, and 19 of 23 patients (83%) with iNOT were functionally independent (Fisher exact test, P<0.001). Of 828 patients with available discharge NIHSS score or 24-hour NIHSS score, 41 of 578 patients (7%) without an intracranial occlusion, 39 of 227 patients (17%) with an intracranial occlusion, and 4 of 23 patients (17%) with iNOT deteriorated clinically during the hospital course and had persistent neurological deficits (Fisher exact test, P<0.001). Sixty-eight of 84 patients (81%) who deteriorated clinically were functionally dependent at discharge or were deceased.

Clinical Course and Functional Outcome of Patients With iNOT
Patients With a Favorable Outcome
Of 19 patients with iNOT who had an independent functional outcome, 2 patients deteriorated clinically during the hospital...
course but returned to their baseline deficits without specific therapy. These were a 78-year-old woman who had iNOT in a left M2 segment (patient 14) and a 72-year-old man (patient 10) who had iNOT in the right M1 segment. The first patient was initially treated with single antithrombotic therapy (aspirin). She had 2 recurrent TIAs with expressive aphasia 20 hours and 23 hours from symptom onset. After the second event she was additionally treated with clopidogrel and experienced no further clinical deterioration. The second patient had progressive left hemiparesis 4 hours from symptom onset. He improved spontaneously and did not further deteriorate during the hospital course.

Both patients with iNOT who were treated with IV tPA improved clinically and were functionally independent at discharge (patients 7 and 8; Table 2). Three patients (patients 1, 2, and 16) who presented with disabling focal neurological deficits improved clinically without thrombolysis and were functionally independent at discharge. One patient (patient 5) had a benign clinical course despite iNOT progression to complete occlusion.

Patients With an Unfavorable Outcome
Among 4 patients with iNOT who deteriorated clinically and had persistent neurological deficits, median time from first CTA to onset of deterioration was 36 hours (range, 0 hours to 3 days). Three of these patients (patients 20, 21, and 22) presented with nondisabling baseline deficits.

Case 1
A 69-year-old woman (patient 20) had iNOT in the terminal left internal carotid artery (Figure 2A). She was treated with triple antithrombotic therapy; however, she deteriorated clinically 3 days after admission with global aphasia and fluctuating right hemiparesis. Repeat CTA demonstrated iNOT progression to near occlusion (Figure 2B). On NCCT, she also exhibited small left-convexity subarachnoid hemorrhage remote to the iNOT site. Perfusion MRI indicated severe time-to-peak delay in the left MCA territory (Figure 2C). Conventional angiography confirmed nonocclusive thrombus (Figure 2D). Mechanical revascularization was considered but not attempted because of preserved antegrade blood flow and assumed high periprocedural risk. She exhibited large left MCA infarction on follow-up NCCT (Figure 2E).

Case 2
An 85-year-old man (patient 21) presented with transient expressive aphasia. He was treated with IV heparin for presumed cardioembolic etiology (ECG showed atrial flutter); however, he deteriorated clinically with recurrent expressive aphasia and mild right hemiparesis 2 days later. CTA performed 3 days after admission revealed iNOT in the left M1 segment (Figure 3A). Nonocclusive thrombus was initially missed but identified retrospectively when confirmed by magnetic resonance angiography after further deterioration. Aspirin was then added for secondary prophylaxis. MRI demonstrated multiple left hemispheric infarcts in a deep watershed distribution (Figure 3B).

Case 3
This 70-year-old woman (patient 22) has been presented in a case report. In brief, she had nonocclusive calcific embolus in the left M1 segment after coronary angiography (Figure 3C). She was treated with aspirin, clopidogrel, and IV heparin, but deteriorated clinically the next day with global aphasia and right hemiplegia. Digital subtraction angiography verified nonocclusive thrombus (Figure 3D) in the left MCA bifurcation. Endovascular embolectomy was attempted but not feasible. Diffusion-weighted MRI revealed left MCA infarction (Figure 3E).

Case 4
A 51-year-old man (patient 23) had iNOT in a left M2 segment 1 day after anterior wall myocardial infarction. The patient was already receiving triple antithrombotic therapy and had intra-aortic balloon pump for left ventricular failure when the stroke occurred, and so IV thrombolysis was withheld. He further deteriorated clinically and exhibited large left MCA infarction on follow-up NCCT.

Vascular Outcome in Patients With iNOT
Seventeen patients with iNOT had repeated vascular studies after a median time of 2.8 days (range, 0.5–9) since first CTA. Nonocclusive thrombus was diminished or resolved in 10 patients. None of these patients (0%) deteriorated clinically during the hospital course. In the remaining 7 patients with unchanged or enlarged iNOT, 4 patients (57%) deteriorated clinically (Fisher exact test, \( P = 0.09 \)); 3 of them had persistent and 1 had nonpersistent new focal neurological deficits.

Table 1. Patient Characteristics According to Intracranial Vascular Status

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Intracranial Occlusion</th>
<th>Intracranial Occlusion</th>
<th>iNOT</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>N, n</td>
<td>603</td>
<td>239</td>
<td>23</td>
<td>...</td>
</tr>
<tr>
<td>Age, yr, mean ± SD</td>
<td>64 ± 16</td>
<td>68 ± 15</td>
<td>69 ± 14</td>
<td>0.006</td>
</tr>
<tr>
<td>Male, n (%)</td>
<td>364 (60)</td>
<td>138 (58)</td>
<td>13 (57)</td>
<td>0.517</td>
</tr>
<tr>
<td>Onset-to-CTA time (hr), median</td>
<td>5.1 (0.5–127.7)</td>
<td>3.5 (0.2–66.5)</td>
<td>3.5 (0.9–75)</td>
<td>0.001</td>
</tr>
<tr>
<td>Baseline NIHSS score, median</td>
<td>3 (0–35)</td>
<td>12 (0–35)</td>
<td>3 (0–23)</td>
<td>0.001</td>
</tr>
<tr>
<td>Nondisabling baseline deficit, n (%)</td>
<td>437 (72)</td>
<td>45 (19)</td>
<td>16 (70.0)</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Any thrombolysis, n (%)</td>
<td>58 (10)</td>
<td>112 (47)</td>
<td>2 (9)</td>
<td>&lt; 0.001</td>
</tr>
</tbody>
</table>

Nondisabling baseline deficit defined as baseline NIHSS score = 5.
Table 2. Baseline Characteristics, Clinical Course, and Outcome of 23 Patients With iNOT on CTA

<table>
<thead>
<tr>
<th>No.</th>
<th>Age (yr.)</th>
<th>Sex</th>
<th>Site, Stenosis</th>
<th>Comment</th>
<th>Treatment</th>
<th>NIHSS Baseline, 24 hr</th>
<th>Clinical Deterioration</th>
<th>mRS</th>
<th>Vascular Outcome: Modality, Time Since First CTA, iNOT Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>52</td>
<td>F</td>
<td>L ICA, ≥50%</td>
<td></td>
<td>Coumadin for s/p MVR, INR 3.2</td>
<td>Hep</td>
<td>23, 8, 2</td>
<td>No</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>75</td>
<td>M</td>
<td>L M2, ≥50%</td>
<td></td>
<td>Coumadin for s/p DVT, INR 1.1</td>
<td>Hep</td>
<td>6, 2, 1</td>
<td>No</td>
<td>1</td>
</tr>
<tr>
<td>3</td>
<td>44</td>
<td>F</td>
<td>R M1, near occl</td>
<td></td>
<td>...</td>
<td>Hep</td>
<td>4, 1, 0</td>
<td>No</td>
<td>1</td>
</tr>
<tr>
<td>4</td>
<td>56</td>
<td>M</td>
<td>R M1, ≥50%</td>
<td></td>
<td>...</td>
<td>ASA</td>
<td>1, 0, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>5</td>
<td>70</td>
<td>M</td>
<td>L M2, near occl</td>
<td></td>
<td>...</td>
<td>ASA</td>
<td>3, 2, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>6</td>
<td>79</td>
<td>F</td>
<td>R M2, ≥50%</td>
<td></td>
<td>...</td>
<td>ASA</td>
<td>3, 2, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>7</td>
<td>81</td>
<td>M</td>
<td>L M2, near occl</td>
<td>IV tPA</td>
<td>ASA (at 1 day)</td>
<td>9, 8, 0</td>
<td>No</td>
<td>0</td>
<td>...</td>
</tr>
<tr>
<td>8</td>
<td>74</td>
<td>F</td>
<td>L M1, near occl</td>
<td>IV tPA</td>
<td>ASA (at 1 day)</td>
<td>18, 2, 2</td>
<td>No</td>
<td>1</td>
<td>MRA, 3 days, resolved</td>
</tr>
<tr>
<td>9</td>
<td>58</td>
<td>M</td>
<td>R M2, near occl</td>
<td>Coumadin for s/p MVR, INR 2.4</td>
<td></td>
<td>ASA, Hep</td>
<td>2, 0, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>10</td>
<td>72</td>
<td>M</td>
<td>R M1, near occl</td>
<td></td>
<td>...</td>
<td>ASA, Hep</td>
<td>4, 3, 0</td>
<td>Yes (non-persistent)</td>
<td>1</td>
</tr>
<tr>
<td>11</td>
<td>62</td>
<td>M</td>
<td>R M2, near occl</td>
<td></td>
<td>...</td>
<td>ASA, Clop</td>
<td>1, 1, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>12</td>
<td>68</td>
<td>M</td>
<td>L M1, near occl</td>
<td></td>
<td>...</td>
<td>ASA, Clop</td>
<td>0, 0, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>13</td>
<td>68</td>
<td>M</td>
<td>R M1, ≥50%</td>
<td></td>
<td>...</td>
<td>ASA, Clop</td>
<td>3, 1, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>14</td>
<td>78</td>
<td>F</td>
<td>L M2, ≥50%</td>
<td>2 TIAs with ASA; no further TIAs with ASA, Clop</td>
<td></td>
<td>ASA, (+ Clop at 1 day)</td>
<td>1, 0, 0</td>
<td>Yes (non-persistent)</td>
<td>0</td>
</tr>
<tr>
<td>15</td>
<td>70</td>
<td>F</td>
<td>L M2, near occl</td>
<td></td>
<td>...</td>
<td>ASA, Clop</td>
<td>4, 1, 1</td>
<td>No</td>
<td>1</td>
</tr>
<tr>
<td>16</td>
<td>30</td>
<td>F</td>
<td>L M1, near occl</td>
<td></td>
<td>...</td>
<td>ASA, Clop</td>
<td>13, 7, 3</td>
<td>No</td>
<td>2</td>
</tr>
<tr>
<td>17</td>
<td>58</td>
<td>M</td>
<td>R M1, &lt;50%</td>
<td></td>
<td>...</td>
<td>ASA, Clop, Hep</td>
<td>0, 0, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>18</td>
<td>44</td>
<td>M</td>
<td>L M2, ≥50%</td>
<td></td>
<td>...</td>
<td>ASA, Clop, Hep</td>
<td>3, 0, 0</td>
<td>No</td>
<td>0</td>
</tr>
<tr>
<td>19</td>
<td>77</td>
<td>F</td>
<td>L ICA, ≥50%</td>
<td>TEE: thrombus aortic valve; small left frontal SAH (NCCT 3 days) as complication of therapy</td>
<td></td>
<td>ASA, Clop, Hep</td>
<td>1, 1, 11</td>
<td>Yes (persistent)</td>
<td>4</td>
</tr>
<tr>
<td>20</td>
<td>69</td>
<td>F</td>
<td>L ICA, ≥50%</td>
<td>iNOT initially missed</td>
<td></td>
<td>Hep, (+ ASA at 8 days)</td>
<td>0, 0, 10</td>
<td>Yes (persistent)</td>
<td>4</td>
</tr>
<tr>
<td>21</td>
<td>85</td>
<td>M</td>
<td>L M1, ≥50%</td>
<td></td>
<td>iNOT initially missed</td>
<td>Hep, (+ ASA at 8 days)</td>
<td>0, 0, 10</td>
<td>Yes (persistent)</td>
<td>4</td>
</tr>
<tr>
<td>22</td>
<td>70</td>
<td>F</td>
<td>L M1, near occl</td>
<td></td>
<td>Calcified iNOT after coronary angiogram</td>
<td>ASA, Clop, Hep</td>
<td>2, 8, 16</td>
<td>Yes (persistent)</td>
<td>4</td>
</tr>
<tr>
<td>23</td>
<td>51</td>
<td>M</td>
<td>L M2, near occl</td>
<td>MI with left ventricular failure 24 hr earlier; treated with ASA, Clop, Hep, IABP</td>
<td></td>
<td>ASA, Hep</td>
<td>13, 16, 20</td>
<td>Yes (persistent)</td>
<td>5</td>
</tr>
</tbody>
</table>

ASA indicates aspirin; Clop, clopidogrel; DVT, deep venous thrombosis; F, female; Hep, IV heparin; INR, international normalized ratio; IABP, intraaortic balloon pump; L, left; M, male; MI, myocardial infarction; MVR, mitral valve replacement; occl, occlusion; R, right; s/p, status post; SAH, subarachnoid hemorrhage; TEE, transesophageal echocardiography.
Repeated vascular studies were performed after onset of clinical deterioration in each case.

We observed a nonsignificant trend toward higher percentages diminished or resolved iNOT in patients with lower baseline stenosis degree and treatment with dual or triple compared to single antithrombotic therapy (Table 3). Anticoagulation with IV Heparin did not facilitate iNOT dissolution.

**Discussion**

Intracranial nonocclusive thrombus was not commonly identified in a large cohort of consecutive patients who received CTA for acute cerebral ischemia. The overall clinical course and functional outcome of patients with iNOT was good in our study. However, a subgroup deteriorated clinically during the hospital course with subsequent unfavorable functional outcome.

The incidence of iNOT in our study (2.7%) is low as reported for intraluminal thrombi in the extracranial internal carotid artery diagnosed with conventional angiography (0.4%–1.5%). Intracranial nonocclusive thrombi have been reported in a case report or as an additional finding in patients with extracranial carotid thrombus. It needs to be determined if iNOT may be more frequently detected with urgent vascular imaging of selected patients who present with resolving deficits. Furthermore, newer-generation CT scanners may increase the detection of iNOT.

In contrast to patients with an intracranial occlusion in our study and in previous reports, the majority of patients with iNOT presented with nondisabling deficits. In patients with early spontaneous improvement before arrival in the emergency room, iNOT may represent remnants of previous occlusive thrombus with spontaneous recanalization. We also assume such a course in 3 patients who presented with disabling focal neurological deficits but who improved spontaneously before CTA was performed. Alternatively, iNOT may have been nonocclusive from the beginning and associated with minor symptoms attributable to preserved antegrade blood flow.

Similar to patients without an intracranial arterial occlusion, the majority of patients with iNOT had a benign clinical course and independent functional outcome. However, 4 patients (17%) deteriorated clinically and had persistent new focal neurological deficits. Three of them were minimally affected at baseline. Moreover, 2 patients fluctuated without persistent deficits. The proportion of iNOT patients with clinical deterioration was similar to the proportion of patients with intracranial occlusion who deteriorated clinically in our study. iNOT may therefore represent a rare but important cause for clinical deterioration in patients with TIA or minor stroke. Our study supports the importance of urgent vascular imaging in these patients.

Why do patients with iNOT deteriorate? Infarct distribution in the internal border zone (in patients 21 and 22) and findings on perfusion MRI (in patient 20) suggest deterioration attributable to hemodynamic compromise. Internal bor-
der zone infarcts resemble the infarct pattern associated with atherosclerotic MCA disease. Measures to improve blood flow may be crucial. In line with our study, hemodynamic compromise has been suggested as the main cause for deterioration in patients who deteriorate after spontaneous improvement. We cannot comment on the frequency of deterioration in patients who deteriorate after spontaneous compromise has been suggested as the main cause for missed. Furthermore, suppression of microembolic signals may have occurred in the patient in whom recurrent TIAs ceased after he was treated with aspirin and clopidogrel. More aggressive therapies (like mechanical embolectomy or surgical thrombus removal) to restore cerebral blood flow may be justified in patients who have persistent or enlarged iNOT and deteriorate clinically despite combination antithrombotic therapy. As one would expect, clinical deterioration was associated with unchanged or enlarged iNOT in repeated vascular studies, whereas diminished or resolved iNOT was associated with a benign clinical course. As repeated studies were performed after onset of clinical deterioration, iNOT status did not predict clinical deterioration in our study. In line with a published case report, combination antithrombotic therapies tended to facilitate iNOT dissolution and may be the best treatment approach in patients with iNOT not eligible for thrombolysis. However, 1 patient (patient 20) whom we treated with triple antithrombotic therapy had subarachnoid hemorrhage on follow-up NCCT. In contrast to reports on extracranial intracarotid thrombi, anticoagulation with IV heparin was not associated with iNOT dissolution in our study. Small numbers and broad variation of treatment regimens limit conclusions.

Our study has limitations. Clinical data were collected retrospectively. We established criteria to diagnose iNOT and cases were reviewed by a neuroradiologist. However, we cannot exclude that intracranial atherosclerotic stenosis with appositional thrombus or intracranial dissection may have been mistaken for iNOT in some cases, particularly if iNOT had not dissolved in repeated vascular studies. MRI with T2* sequences might help to further characterize such lesions. Furthermore, repeated vascular studies were performed with different modalities than initial CTA in most patients, thus limiting the significance of our findings on iNOT course. Patients were treated with a variety of antithrombotic therapies, and it is impossible to know if the treatments had any effects on the prognosis in these patients.

In conclusion, iNOT appears to be rare in patients with acute ischemic stroke or TIA. The majority of patients have a benign clinical course. However, some patients deteriorate clinically because of persistent or enlarged thrombus. The best way to manage these patients requires further study.

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Disclosures

None.

References


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