Clinical Risk Factors and Plaque Characteristics Associated with New Development of Contralateral Stenosis in Patients Undergoing Carotid Endarterectomy

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The contralateral carotid artery after carotid endarterectomy (CEA): a mid-term follow-up study

New contralateral stenosis after CEA is common

Gives more new symptoms

Associated with clinical and plaque risk factors

Need for follow-up of patients at risk for new contralateral stenosis
Background

- After CEA ipsilateral restenosis and contralateral stenosis is common

- New stenosis may produce new cerebral symptoms and need for re-intervention

- Risk factors allow for patient specific follow-up
  - Clinical risk factors
  - Plaque characteristics
Aim of this study

- Incidence of new contralateral stenosis after CEA
- Clinical risk factors
- Plaque characteristics
- Outcome of patients with new contralateral stenosis
Methods: Athero-Express Biobank

**Inclusion**

- **CEA**
- Plaque collected + histological examination

**3 months**
- Duplex + Clinical follow-up

**1 year**
- Duplex + Clinical follow-up

**... yearly**
- Duplex + Clinical follow-up

Considered as stenosis if:

- > 50% stenosis
- > 210 cm/s PSV value
Results: inclusion and exclusion of patients

760 CEA's included in study

Excluded for study analysis (n = 228)
- Contralateral stenosis at baseline (n = 138)
- Data missing (n = 90)

Development of contralateral stenosis (n = 108)

No development of contralateral stenosis (n = 424)

High grade stenosis (n = 57)
Occlusion (n = 51)

1 of 5 patients developed new contralateral stenosis
Results:
Clinical risk factors associated are history of coronary artery disease (CAD) & asymptomatic presentation

<table>
<thead>
<tr>
<th>Patient characteristics</th>
<th>Contralateral stenosis</th>
<th>No stenosis</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Female gender</td>
<td>27/108 (25)</td>
<td>142/424 (34)</td>
<td>0.091</td>
</tr>
<tr>
<td>Mean age</td>
<td>69 (9)</td>
<td>68 (10)</td>
<td>0.444</td>
</tr>
<tr>
<td>Current smoker</td>
<td>41/108 (38)</td>
<td>132/418 (32)</td>
<td>0.208</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>25/108 (23)</td>
<td>88/424 (21)</td>
<td>0.587</td>
</tr>
<tr>
<td>Hypertension</td>
<td>79/106 (74)</td>
<td>307/420 (73)</td>
<td>0.765</td>
</tr>
<tr>
<td>Mean body mass index</td>
<td>26.5 (4)</td>
<td>26.4 (4)</td>
<td>0.285</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>70/96 (73)</td>
<td>278/395 (70)</td>
<td>0.624</td>
</tr>
<tr>
<td><strong>History of CAD</strong></td>
<td>42/108 (39)</td>
<td>119/422 (28)</td>
<td><strong>0.031</strong>*</td>
</tr>
<tr>
<td>History of PI</td>
<td>15/108 (14)</td>
<td>80/421 (19)</td>
<td>0.217</td>
</tr>
<tr>
<td>Acetylsalicylic acid</td>
<td>88/108 (82)</td>
<td>350/421 (83)</td>
<td>0.685</td>
</tr>
<tr>
<td>Antiplatelet therapy</td>
<td>91/108 (84)</td>
<td>373/421 (87)</td>
<td>0.220</td>
</tr>
<tr>
<td>Statin use</td>
<td>97/109 (90)</td>
<td>347/422 (82)</td>
<td>0.056</td>
</tr>
<tr>
<td>Mean total cholesterol</td>
<td>4.2 (1.1)</td>
<td>4.6 (1.2)</td>
<td>0.339</td>
</tr>
<tr>
<td>High density lipoprotein</td>
<td>1.1 (0.36)</td>
<td>1.2 (0.51)</td>
<td>0.289</td>
</tr>
<tr>
<td>Low density lipoprotein</td>
<td>2.4 (0.89)</td>
<td>2.7 (0.99)</td>
<td>0.389</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>1.6 (0.82)</td>
<td>1.6 (0.99)</td>
<td>0.378</td>
</tr>
<tr>
<td>Mean GFR, CG</td>
<td>72 (2.9)</td>
<td>73 (1.3)</td>
<td>0.114</td>
</tr>
<tr>
<td><strong>Asymptomatic</strong></td>
<td>24/108 (22)</td>
<td>40/420 (10)</td>
<td><strong>0.000</strong>*</td>
</tr>
<tr>
<td>Amourosis fugax</td>
<td>16/108 (15)</td>
<td>64/420 (15)</td>
<td>0.133</td>
</tr>
<tr>
<td>TIA</td>
<td>45/108 (42)</td>
<td>209/420 (50)</td>
<td>0.368</td>
</tr>
<tr>
<td>Stroke</td>
<td>23/108 (21)</td>
<td>107/420 (26)</td>
<td>0.913</td>
</tr>
<tr>
<td>Type of closure</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No patch, primary closure</td>
<td>5/107 (5)</td>
<td>28/418 (7)</td>
<td>0.441</td>
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<tr>
<td>Venous</td>
<td>69/107 (65)</td>
<td>273/418 (65)</td>
<td>0.873</td>
</tr>
<tr>
<td>Bovine</td>
<td>22/107 (21)</td>
<td>85/418 (20)</td>
<td>0.959</td>
</tr>
<tr>
<td>Dacron</td>
<td>11/107 (10)</td>
<td>32/418 (8)</td>
<td>0.377</td>
</tr>
</tbody>
</table>
Results:
Plaque characteristics associated are small lipid core, high collagen and high smooth muscle cells.

Stable plaque characteristics
Results: clinical outcome

1. Patients with new contralateral stenosis showed more often new cerebral symptoms
   • 14.3 % vs 6.6% in patients with no contralateral stenosis ($P=0.049$)

2. Contralateral stenosis free survival indicated earlier development of contralateral stenosis in asymptomatic patients
   • Hazard ratio 0.27 after 3 years in asymptomatic patients
   • Hazard ratio 0.26 after 6 years in symptomatic patients
Conclusions:

1 in 5 patients develops new contralateral stenosis after CEA

50% of those are occlusions

Asymptomatic patients show more often and earlier new development of contralateral stenosis after CEA

Stable plaque characteristics are associated with new contralateral stenosis after CEA

New contralateral stenosis after CEA gives more new cerebral symptoms
Conclusions:

Asymptomatic patients show more often and earlier new development of contralateral stenosis after CEA.

New contralateral stenosis after CEA gives more new cerebral symptoms.

Stable plaque characteristics are associated with new contralateral stenosis after CEA.

1 in 5 patients develops new contralateral stenosis after CEA. 50% of those are occlusions.

Take home message:

- Contralateral stenosis after CEA is common.
- Patients at risk: more intensive follow-up.
- Future: development of individual treatment algorithms.