Cranial nerve palsies in spontaneous carotid artery dissection

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Abstract
Two patients had isolated unilateral cranial nerve palsies due to spontaneous internal carotid artery (ICA) dissection without ischaemic cerebral involvement. One had acute glossopharyngeal and vagal, the other isolated hypoglossal nerve palsy. Reviewing all reported cases of angiographically confirmed ICA dissection in the literature, 36 additional cases with unequivocal ipsilateral cranial nerve palsies were analysed. While an isolated palsy of the IXth and Xth has not been reported previously, palsies of the XIth nerve or the IXth to XIth nerves were most frequently found. In these patients, lower cranial nerve palsies are probably the result of compression by an enlarging ICA due to mural haematoma. Symptoms and signs indicative of carotid dissection were concurrently present only in some reported cases. This raises the question of unrecognised carotid dissection as a cause of isolated cranial nerve palsies. When the dissection occurs in the subadventitial layer without relevant narrowing of the arterial lumen and when an aneurysm is thrombosed, angiography does not reliably yield the diagnosis. Therefore, carotid dissection might have been underestimated as a cause of isolated lower cranial nerve palsies before the advent of MRI. MRI demonstrates directly the extension of the wall haematoma in the axial and longitudinal planes. Some arteriopathies such as fibromuscular dysplasia and tortuosity make a vessel predisposed to dissection.

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The clinical presentation of spontaneous internal carotid artery (ICA) dissection is highly variable. Sudden, intense unilateral pain in the neck and face, ipsilateral ocular sympathetic paresis, and transient monocular blindness (amaurosis fugax) associated with a contralateral transient ischaemic attack or stroke form the typical presenting triad suggesting extracranial carotid dissection.\(^\text{1,2,3}\) Lower cranial nerve palsies seem to be a rare finding, with the hypoglossal nerve being the most frequently affected (5%).\(^\text{4}\) However, when ICA dissection occurs without CNS involvement, it is not readily recognised as the cause of a cranial nerve palsy. Some of the reported “idiopathic” jugular foramen (Vernet) and similar syndromes (Villaret, Collet Siccard) in the older literature might well have been caused by spontaneous carotid dissection.\(^\text{5}\) In those times ICA dissection as a cause of cranial nerve palsy was poorly known, angiography quite risky, and ultrasound and MRI not available. Out of 31 consecutive patients with ICA dissection diagnosed at our department within four years, two presented with cranial nerve palsies without cerebral involvement.

Case reports
PATIENT 1
A 42-year-old man had been in excellent health. He had no vascular risk factors. After strenuous activity (cutting wood) in military service he suddenly felt pain in the right upper jaw which later extended to the ear and the whole right side of his face. He suffered an episode of hazy vision on the right eye lasting about five minutes. The following morning pain had almost disappeared, but he had difficulty swallowing and could not drink properly. His voice had a nasal sound. Otherwise he felt well. In the hospital, three days after first symptoms had occurred, his voice was severely hoarse, almost aphonic, and nasal. On examination he had a drooping right palpebral arc. On phonation the palate as well as the dorsal wall of the pharynx deviated to the left. Sensation to touch of the right posterior pharyngeal wall was reduced and the gag reflex could not be elicited on the right side. No other signs were found. Chest x-ray, ECG, and routine blood laboratory tests were normal. Initially brainstem infarction was suspected and a posterior fossa CT scan was performed which was normal. Because of the right sided facial pain and the initial episode of presumed right amaurosis fugax, right ICA dissection was suspected.

Duplex sonographic examination of the carotid arteries performed the same day showed a patent carotid bifurcation, no atherosclerotic changes, and symmetrical blood flow velocities. Both common carotid arteries were also normal. Pulsed Doppler sonographic examination of the upper cervical segments of both ICAs disclosed a coiling of both with adjacent antegrade and retrograde flow in the high cervical portion. However, the blood flow velocities on the right side were twice as high as on the left side in both flow directions. This finding supported the diagnosis of an ICA dissection and anticoagu-
ulation with heparin sodium was started. The following day, a right common carotid arteriogram was performed which also revealed the ICA tortuosity. There were irregularities of the vessel wall about 3 cm distal of the bifurcation and extending cranially beyond the coiling. Equivocal slight stenosis on the anteroposterior projections was visible (fig 1).

In addition, a pouch with trapped contrast media showed up on the late lateral views (fig 1 a2 and 1 a3) which is a quite distinct sign of dissection. These findings confirmed the suspected diagnosis of ICA dissection. They could, however, not explain the glossopharyngeal and vagal nerve palsy.

MR images of the upper cervical region performed eight days after the first symptoms yielded the explanation. A large ICA mural haematoma was demonstrated as a hyperintense signal on the sagittal and axial sections of the T1- and T2-weighted and proton density spin echo sequences (fig 2). It extended from beneath the coiling up to the petrosal carotid canal. In the axial plane, a large subadventitial extension of the haematoma was demonstrated with expansion of the vessel wall in the carotid space. This lead to displacement of the parapharyngeal space (fig 2 b1 and 2 b2) where the glossopharyngeal nerve crosses the ICA medially leading from the jugular foramen to the root of the tongue. In this region the Xth cranial nerve is also in close contact with the ICA (fig 3). Cranial MRI was normal.

After one week of heparin treatment, oral anticoagulation with a coumarin was started. No visual or TIA-like symptoms occurred. Six weeks after admission, hoarseness and nasal voice were still present, but swallowing was possible again. Pharyngeal sensation was normal on both sides, no palate droop was found any more and pharyngeal wall shift on phonation was only minimal. Doppler sonography still showed a higher blood flow velocity on the right side. A second MRI scan eight weeks after admission showed complete resolution of the mural haematoma. Anti-coagulants were continued and after a total of 11 weeks all symptoms and signs had cleared. A persistent slight asymmetry of blood flow velocities at Doppler examination was considered to be due to possible slight residual stenosis. Coumarins were stopped and aspirin started.

PATIENT 2

A 45-year-old man had untreated mild arterial hypertension for several years. During the past two years he suffered from intermittent slight nuchal pain. Two days before admission nuchal pain intensified but did not change its well known quality. There was no special event and no trauma. On the day of admission he noticed difficulty with articulation, a heavy tongue, and problems to eat. Slightly slurred speech was noted by his wife. Brainstem infarction was suspected and a CT scan performed which was normal. On admission he clearly stated that he could not properly move the food with his tongue, while oral sensibility, chewing, and swallowing were not disturbed. Examination showed moderate left hypoglossal palsy with tongue deviation to the left when thrusting it out and deviation to the right when phonating. No other signs were found. Chest and cervical spine x ray, electrocardiogram, and routine blood laboratory tests were normal.

Doppler and Duplex sonographic examination of the cervical carotid and vertebral arteries and transcranial Doppler sonography on the day of admission were normal. Repeat ultrasound study the following day revealed occlusion of left ICA with typical signs indicating dissection. Transcranial Doppler examination showed good collateral supply from the right carotid and from the verte-
brobasilar system across the circle of Willis. MR images of the upper cervical region were performed two days after the first symptoms. They showed a mural haematoma in the prepetrosal left ICA segment with severely narrowed lumen (flow void). Caudal to it there was an isodense mass adjacent to the artery, probably representing an aneurysm (fig 4). Three sectioned lumina (flow void) on both sides indicated bilateral coiling. There were no signal changes in the brain, brainstem, or cerebellum. Bilateral carotid angiography was performed three days after the first symptoms. It showed bilateral coiling before the ICAs entered the petrosal canal. On the left side contrast filling was markedly delayed and a saccular aneurysm attached to the coiling filled. The segment immediately distal to the coiling was severely stenosed (fig 5). On the day of admission heparin treatment was begun and seven days later changed to oral anticoagulation. Control ultrasound examination seven days later showed partial resolution of the occlusion with a now present, though diminished flow signal in the left ICA. Hypoglossal palsy was still slightly present but subjective discomfort was minimal.

Review of the literature
All reported series and cases of spontaneous ICA dissection were reviewed looking for unequivocally documented symptoms and signs of cranial nerve involvement. Swallowing difficulty alone was not considered to indicate IXth or Xth nerve dysfunction because local expansion of the aneurysm towards the pharynx might be the cause.7 Unilateral "scalp tenderness", a frequently encountered symptom in some series,8 was not considered to indicate trigeminal nerve involvement unless there was a clear sensory deficit reported. "Dysgeusia" (unpleasant, strange taste) does, in our opinion, indicate facial or glossopharyngeal nerve dysfunction. As Maitland did,9 we suspect a compression of the IXth rather than of the VIIth nerve or of the chorda tympani to be the cause.10 Detailed testing of taste, which should allow a differentiation, was not reported in these cases. If a nerve compression by the expanding wall haematoma (pseudoaneurysm) is the pathogenetic mechanism, the IXth nerve is more at risk. If, however, interruption of the nerve’s blood supply is the working mechanism, the IXth and VIIth may be affected equally. Finally, isolated tinnitus was not considered a sign of VIIIth nerve dysfunction.

Only cases in whom the diagnosis had been supported by angiography showing a typical picture were considered.1 3 5 11 12

Results
Thirty-six patients reported under the heading of carotid dissection with signs of ipsilateral cranial nerve palsies were found in the literature and analysed together with our two patients (table 1).2 8 9 10 12-31 In 35, the age and sex were given: the average age was 44 years,
Figure 4 (Patient 2) MR images at 1.5 tesla. The proton density weighted (TR = 2740 ms, TE = 30 ms) contiguous axial sections at the level of the skull base (a1 and a2) demonstrate narrow lumen (flow void) of left ICA (arrowhead in a1) and a surrounding mass with mixed iso and hyperintense signal (arrow in a1). A little more cranially a hyperintense signal adjacent to the flow void signal indicates mural haematoma (arrow in a2). These signal changes are even better visualised on T1-weighted (TR = 800 ms, TE = 11 ms) axial sections with fat suppression technique (b1 and b2): The mass asymmetrically surrounding the narrowed vessel lumen (arrows in b1) represents the aneurysm. The slit-like residual lumen (arrowhead in b2) is compressed by the mural haematoma shown as hyperintense signal (arrow in b2).

ranging from 28 to 60 years. Thirty were men (86%, mean age 47 years) and five women (mean age 39 years). All but one patient showed an ICA narrowing of variable degree: 11 patients had a high grade stenosis with a string sign, seven a long irregular slight narrowing, 15 a segmental narrowing, and one a tapering occlusion. Seventeen (49%) had aneurysmal dilatation: nine had a fusiform pseudoaneurysm, two of them bilaterally; three had aneurysmal pouches (fig 1); three had a saccular aneurysm (fig 5); one had a double lumen; and in one patient no details on the “aneurysm” were available. In three patients detailed angiographic results were not given. In 35 patients the site of the dissection could be located: the prepetrosal segment was involved in all, 11 showed extension of the dissection to the (lower) cervical segment, 11 to the intrapetrosal, and eight to the cavernous segment. In three patients this information was not provided. In 26 patients data about tortuosity of the ICA were given: 10 patients (38%) had some degree of tortuosity, four had kinking, three coiling, and three moderate tortuosity. Four patients had documented findings suggestive of fibromuscular dysplasia (FMD). Tortuosity as well as the signs of FMD may be masked by the dissection itself. Only a minority of the reported cases had documented cerebral four vessel angiography and comments concerning the presence of FMD and tortuosity in other cranial arteries were frequently lacking. Therefore, the relative frequency of these vascular alterations is certainly underestimated.

In 16 patients MRI scans were performed, 14 of which had a mural haematoma demonstrated. In two cases the MRI showed no hyperintense signal. In one of these the delay was 8 weeks and the haematoma might have resolved. In the other patient no details on MR technique or examination time were given in the presence of a typical angiographic result. In our two patients MRI demonstrated tortuosity (fig 2 b1) and also an aneurysm (fig 4 b1) in addition to the mural haematoma.

In 27 patients an ipsilateral XIIth nerve palsy was reported, 10 times isolated, six times in conjunction with IXth, Xth, and XIth nerve palsies, four times with XIIth nerve palsy, five times with IXth and Xth nerve palsies, once with IXth nerve palsy, and once with IXth, Xth, VIIth, and Vth nerve palsies. Five patients had dysgeusia, three isolated, one in conjunction with Vth, and one with Vth nerve palsy. Among seven patients with a Vth nerve palsy, it was isolated in four. All patients with Vth, Vth, or IIIrd nerve palsy had an extension of the dissection up to the cavernous segment. The compiled frequency of the affected cranial nerves in the 38 cases was as follows (table 1): XIIth: 27 times; IXth (including the five patients with dysgeusia): 18 times; Xth: 17 times; XIIth: 7 times; Vth: 7 times; VIIth/Vth/IIIrd: once each. In 20 patients (53%) an ipsilateral oculosym pathetic paresis (Horner’s syndrome) was observed.

In 13 patients information about outcome of cranial nerve palsies was given: eight patients had recovered after three weeks to two years, usually within two to four months; five patients had persistent palsies after three to 10 months and, hence, some might well have recovered later on.

Ten patients (26%) had ischaemic symptoms. Six had a transient and two a persistent hemiparesis contralateral to the dissection, and two had an ipsilateral amaurosis fugax.

Discussion
Peripheral cranial nerve palsy caused by ICA dissection is only rarely observed. In the large reported series totalling 356 patients with ICA dissection, no cranial nerve palsies were described. Most of these patients with angiographically confirmed ICA dissection were presenting with cerebral ischaemic signs and symptoms. On the other hand, only 8 (21%) of 38 patients presenting with cranial nerve palsies due to ICA dissection collected from the literature (table 1) showed cerebral ischaemic involvement.

This discrepancy may be due to not noticing ipsilateral cranial nerve palsies in the presence of signs of contralateral stroke. It might...
also indicate that ICA dissection as a cause of lower cranial nerve palsies is not readily recognised. In a recent review of 32 patients with XIIth nerve palsy, carotid dissection was considered to be the cause in only one patient, and this was a traumatic dissection. Unfortunately, no details on the diagnostic examination of these patients are given. Therefore, the question remains, whether some of the reported patients with so called traumatic XIIth nerve palsies or some of those attributed to brainstem ischaemia in reality had a carotid dissection causing the nerve palsy. In our own series of 31 patients with ICA dissection, the two patients reported here (6%) presented with isolated cranial nerve palsies. In retrospect, we recall another two patients who presented with isolated XIIth nerve palsy and ipsilateral neck pain. CT scan did not support the suspected diagnosis of a cervical tumour or infection. This was several years ago when ICA dissection was not considered as a possible cause and hence no further investigations were performed.

However, the relatively frequent observation of isolated cranial nerve palsies without central involvement (79%) as opposed to the rare observation of cranial nerve palsies in ICA dissection patients with cerebral ischaemic symptoms could indicate that these two groups have a different pathogenesis. It is, for instance, conceivable that the former had subadventitial rather than subintimal dissections, since the subadventitial dissection is less likely to obstruct the vessel lumen and would rather extend outwardly. Several facts from our analysis support such a hypothesis: (1) the higher prevalence of men in the ICA dissection patients with cranial nerve palsies (86%) as opposed to the general ICA dissection population (51%); (2) like in traumatic ICA dissection, there is a high prevalence of aneurysms in spontaneous ICA dissection patients with cranial nerve palsies (49%), while aneurysms in ICA dissection patients with hemispheric signs are less frequent (10–33%); (3) the higher prevalence of extreme vessel tortuosity in the ICA dissection patients with cranial nerve palsies (27%) compared with the general population (3–12%). Tortuosity may indicate a structure wall anomaly making the vessel predisposed to external mechanical injury. Tortuosity also does alter flow dynamics which may cause sheer injury to the vascular wall again making the affected vessel predisposed to dissection. Could more frequently occurring trivial and unreported traumas of a physically more active male population be favouring subadventitial dissection in the presence of a predisposing arteriopathy?

In the case of subadventitial dissection, muscular medial layer may impede narrowing of the lumen and the mural haematoma may considerably expand the circumference of the vessels (figs 2b and 4b). The close topographical relationship of the most frequently involved XIIth, IXth, and Xth nerves with the carotid artery in the upper cervical parapha-
Table 1  Patients with spontaneous ICA dissection and cranial nerve palsies

<table>
<thead>
<tr>
<th>Reference</th>
<th>Age/Sex</th>
<th>Side</th>
<th>Site of ICA dissection</th>
<th>Angiography</th>
<th>Torsion? FMD MRI (days)</th>
<th>Cranial nerve palsies</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kramer‡</td>
<td>41/F</td>
<td>left</td>
<td>prepetrosal</td>
<td>segmental narrowing, aneurysm prepetrosal fusiform aneurysm</td>
<td>no yes no</td>
<td>IX, X(X), XII left</td>
</tr>
<tr>
<td>Gros‡‡‡</td>
<td>38/M</td>
<td>left</td>
<td>prepetrosal</td>
<td>segmental narrowing prepetrosal aneurysm</td>
<td>no no no</td>
<td>XII left</td>
</tr>
<tr>
<td>Labauge‡</td>
<td>38/M</td>
<td>left</td>
<td>prepetrosal</td>
<td>segmental narrowing prepetrosal aneurysm</td>
<td>yes no</td>
<td>V (1 + 2) left</td>
</tr>
<tr>
<td>Cohen‡</td>
<td>31/F</td>
<td>left</td>
<td>pre and intrapetrosal, cavernous (?)</td>
<td>segmental narrowing prepetrosal aneurysm</td>
<td>yes no</td>
<td>V left</td>
</tr>
<tr>
<td>Fisher‡</td>
<td>45/M</td>
<td>left</td>
<td>pre and intrapetrosal, cavernous (?)</td>
<td>segmental narrowing prepetrosal aneurysm</td>
<td>yes no</td>
<td>dysesthesia, chorda tympani left?</td>
</tr>
<tr>
<td>Barbizet‡</td>
<td>53/M</td>
<td>left</td>
<td>prepetrosal</td>
<td>string sign up to petrosal canal, pouch segmental narrowing</td>
<td>no no no</td>
<td>III and V right</td>
</tr>
<tr>
<td>Braddock‡</td>
<td>58/M</td>
<td>left</td>
<td>prepetrosal, intrapetrosal, cavernous prepetrosal</td>
<td>string sign emboli in MCA branches disappear, saccular aneurysm</td>
<td>no no no</td>
<td>IX, X, XI, XII left</td>
</tr>
<tr>
<td>Goodman‡</td>
<td>41/M</td>
<td>right</td>
<td>prepetrosal</td>
<td>string sign kinking yes no no yes no</td>
<td>XII left</td>
<td></td>
</tr>
<tr>
<td>Maitland‡</td>
<td>37/F</td>
<td>left</td>
<td>pre and intrapetrosal, cavernous prepetrosal</td>
<td>string sign</td>
<td>no no no</td>
<td>VI left, dysesthesia (chorda tympani?)</td>
</tr>
<tr>
<td>Hommel‡</td>
<td>55/M</td>
<td>left</td>
<td>prepetrosal</td>
<td>segmental narrowing saccular aneurysm</td>
<td>no no no</td>
<td>XII left</td>
</tr>
<tr>
<td>54/M</td>
<td>right</td>
<td>prepetrosal</td>
<td>segmental narrowing fusiform aneurysm</td>
<td>yes no no</td>
<td>X, XII right</td>
<td></td>
</tr>
<tr>
<td>44/M</td>
<td>left</td>
<td>cervical, prepetrosal</td>
<td>string sign</td>
<td>no no no</td>
<td>IX, X, XII left</td>
<td></td>
</tr>
<tr>
<td>Gauthier‡</td>
<td>37/F?</td>
<td>left</td>
<td>?</td>
<td>stenosis, no further details</td>
<td>? ? no</td>
<td>IX, X, XII left</td>
</tr>
<tr>
<td>Mokri‡</td>
<td>47/M</td>
<td>bilateral</td>
<td>prepetrosal left</td>
<td>segmental stenosis pseudoaneurysm string sign</td>
<td>kinking no no</td>
<td>IX,X,XI,XII left</td>
</tr>
<tr>
<td>Benrabah‡</td>
<td>40/M</td>
<td>left</td>
<td>prepetrosal right cervical, prepetrosal, cavernous prepetrosal</td>
<td>string sign</td>
<td>yes no no yes</td>
<td>XII left</td>
</tr>
<tr>
<td>Vighetto‡</td>
<td>48/M</td>
<td>left</td>
<td>prepetrosal</td>
<td>long irregular narrowing tapered narrowing</td>
<td>no yes</td>
<td>delay: 16 days yes, delay: ?</td>
</tr>
<tr>
<td>Goldberg‡</td>
<td>49/M</td>
<td>right</td>
<td>pre- and intrapetrosal, high cervical, intrapetrosal, cavernous prepetrosal</td>
<td>string sign</td>
<td>yes no no yes no</td>
<td>V left (hyperesthesia)</td>
</tr>
<tr>
<td>Francis‡</td>
<td>46/F</td>
<td>left</td>
<td>prepetrosal</td>
<td>long irregular narrowing</td>
<td>no yes</td>
<td>delay: 13 days yes, delay: ?</td>
</tr>
<tr>
<td>Hess‡</td>
<td>41/M</td>
<td>right</td>
<td>cervical, prepetrosal</td>
<td>tapering occlusion</td>
<td>no no no</td>
<td>XI,XII right</td>
</tr>
<tr>
<td>Lieschke‡</td>
<td>42/M</td>
<td>right</td>
<td>prepetrosal</td>
<td>string sign</td>
<td>kinking no no</td>
<td>X,XII right</td>
</tr>
<tr>
<td>Waespe‡</td>
<td>41/M</td>
<td>bilateral</td>
<td>prepetrosal bilateral</td>
<td>pseudoaneurysm, bilateral, irregular slit narrowing</td>
<td>no no no</td>
<td>IX,X,XII left</td>
</tr>
<tr>
<td>Braddock‡</td>
<td>28/M</td>
<td>left</td>
<td>cervical, prepetrosal</td>
<td>long irregular narrow, pseudoaneurysm narrow, pseudoaneurysm string sign</td>
<td>no no no</td>
<td>XII left</td>
</tr>
<tr>
<td>Dal Pozzo‡</td>
<td>58/M</td>
<td>left</td>
<td>prepetrosal</td>
<td>slight segmental narrowing</td>
<td>no yes</td>
<td>yes, delay: 19 days IX, X,XII, XII left</td>
</tr>
<tr>
<td>45/M</td>
<td>right</td>
<td>prepetrosal</td>
<td>slight segmental narrowing</td>
<td>yes no</td>
<td>yes, delay: 40 days</td>
<td></td>
</tr>
<tr>
<td>54/M</td>
<td>left</td>
<td>cervical, pre- and intra petrosal</td>
<td>segmental narrowing, small pouch</td>
<td>no no</td>
<td>delay: 27 days IX, X,XII left</td>
<td></td>
</tr>
<tr>
<td>Vernay‡</td>
<td>60/M</td>
<td>left</td>
<td>pre- and intrapetrosal, cavernous prepetrosal</td>
<td>segmental stenosis pseudoaneurysm string sign</td>
<td>no no no</td>
<td>XII left</td>
</tr>
<tr>
<td>Panisset‡</td>
<td>53/M</td>
<td>left</td>
<td>pre- and intrapetrosal, cavernous prepetrosal</td>
<td>stenosis, double humen</td>
<td>yes no</td>
<td>V, VII, IX, X, XII left</td>
</tr>
<tr>
<td>Sturzenegger‡</td>
<td>45/M</td>
<td>right</td>
<td>cervical, prepetrosal</td>
<td>long distance irregular wall, slight stenosis, small pouch</td>
<td>no no no</td>
<td>IX, X XII left</td>
</tr>
<tr>
<td>45/M</td>
<td>left</td>
<td>pre- and intrapetrosal</td>
<td>segmental severe stenosis saccular aneurysm within coiling</td>
<td>yes no no</td>
<td>yes, delay: 2 days XII left</td>
<td></td>
</tr>
</tbody>
</table>

Year indicates year of publication; ICA, internal carotid artery; MCA, middle cerebral artery; FMD, fibromuscular dysplasia; TIA, transient ischaemic attack; ?, means that the respective criterion could not be analysed because of lack of data.

The Ixth cranial nerve crosses the ICA laterally, the Xth lies posteriorly and medially, and the XIth crosses the ICA a little more caudally, in general at the C3 level. The fact that in patients with involvement of upper cranial nerves, dissection usually extended into the petrosal and cavernous segment (table 1) is another argument for a compression to be the causative mechanism. Nerve ischaemia due to compromised blood supply is a second possible mechanism.46 The IXth to XIIth nerves...
Cranial nerve palsy in carotid dissection

Homer’s nerve Pain TIA/stroke Special remarks

yes left ear, face, neck left no dissection very likely
no temple left right arm + face no
no face left paresis right arm transient persistent XII nerve palsy
yes left eye, cheek left no “Raeber’s syndrome”
yes left temple left no “Raeber’s syndrome”
yes face, eye left right arm + face no
yes face, eye left no
yes neck, temple, head right hemiplegia left no
yes neck left no
yes temple left no hemiparesis left no
yes neck right transient recovery of VIth nerve palsy
no face, neck, left eye no recovery of XIth nerve palsy
no eye, temple left no within 2 months
no temple, occipital right no control angiography after 2 months: persistent aneurysm, no stenosis recovery of Xth nerve palsy within 3 days and of XIIth within 6 months control angiography after 8 months: persistent aneurysm, no stenosis
no yaw, ear, face left hemiplegia right aphasia, persist
no face left no no details on angiography
? ? no further details provided
yes left occipital left no asymptomatic after 2 1/2 y
no right no
yes face, neck, eye left no
yes face, eye left no
yes left neck, temple left no
yes face left no
yes face, neck left no MRI = normal (?) delay not given
no orbital right hemiparesis left transient
no temple, eye no
no neck, face left no bilateral dissection, right side asymptomatic
no neck right no XII paresis persistent after 4 months
no head no XII paresis persistent after 6 months
no, head left no
no neck no CT also positive. Follow up MRI after 13 months: haematoma resolved. CT also positive. Follow up MRI after 14 months: haematoma resolved. CT negative. Follow up MRI after 6 weeks: smaller still persistent haematoma.
no head, eye, front left no
no occipital left no recovery within 2 months
no occipital, eye right no recovery within 6 months
no occipital, neck left no
no yaw, ear, face left no bilateral coiling at MRI. Follow up MRI after 7 weeks: haematoma resolved. clinical recovery within 10 weeks.
no muchal and arteriography no unilateral coiling (at MRI)
no Partial resolution of stenosis after 7 days (Ultrasound). Clinical recovery within 3 weeks.

Notes:
- Dissection may involve one or both intracranial arteries.

Possible mechanisms of cranial nerve palsy:
- Direct compression of cranial nerve by intracranial dissection.
- Herniation into the carotid space.
- Intracranial haematoma.
- Middle meningeal artery compression.

Case reports:
- Patient 1: Right facial weakness, left hemiparesis, delayed recovery.
- Patient 2: Left facial weakness, right hemiparesis, delayed recovery.
- Patient 3: Bilateral facial weakness, bilateral hemiparesis.

Clinical management:
- Conservative management, surgical intervention unlikely.
- Corticosteroids may be considered.

Angiography:
- Mainstay of diagnosis.
- Can confirm dissection, extent of stenosis.

Conclusion:
- Cranial nerve palsy in carotid dissection is a rare but important clinical entity.
- Early recognition and appropriate management are crucial.

References:
- 2. FMD.50 FMD in ICA dissection has been reported in as many as 22%.7 However, FMD cannot be considered the main aetiology of ICA dissection. The sex ratios observed in FMD and ICA dissection strongly argue against that: FMD affects up to 80% women,6 and in the present review series 86% of patients with
ICA dissection were men. In addition, intracranial aneurysms occur frequently with FMD,\textsuperscript{10} but only exceptionally with ICA dissection.

Tortuosity, next to FMD, is the second most common arterial wall anomyly cited to make a vessel predisposed for dissection.\textsuperscript{4,21} In our review (table 1) 10 (38%) of 26 patients whose angiograms could be judged had some degree of tortuosity. Seven (27%) had extreme tortuosity (kinking and coiling) in only 3% to 12%. \textsuperscript{45} Tortuosity seems to be more frequent in those patients with dissection who have aneurysm formation. Among the 10 patients with tortuosity we found seven with aneurysms (70%), whereas the 16 patients without tortuosity had only five aneurysms (31%). Whether extreme tortuosity alone, not associated with atherosclerosis or dissection, can cause cerebrovascular ischaemia or lower cranial nerve compression is a matter of debate.\textsuperscript{45}

Application of MRI and a large index of susceptibility may clarify some of the unsettled issues. MRI is the only non-invasive technique to demonstrate mural haematoma and to show its longitudinal extension (fig 2a) and the degree of wall expansion (fig 2b and 4b).\textsuperscript{24,26-31,49} Newer techniques such as chemical shift imaging (fat suppression) (fig 4b, b2) make MRI more sensitive to even small arterial dissections.\textsuperscript{46} Additionally gives information about surrounding tissues. The information provided by MRI at present is complementary to that of angiography.

In conclusion, carotid dissection should be considered in isolated or combined lower cranial nerve palsies, especially if associated with ipsilateral neck pain, headache, and Horner's syndrome. Descriptive terms like "carotidynia", Raeder's syndrome, "migraine with miosis", and all the cranial nerve syndromes with famous eponyms\textsuperscript{5} should be avoided in favour of a causal diagnosis. Today, MRI provides a sensitive non-invasive method for the diagnosis of ICA dissection if performed within the first weeks.