Clinical Identification of TIAs Due to Carotid Stenosis


SUMMARY A retrospective case note survey of 139 cases of carotid territory TIAs was carried out. Angiographic evidence of carotid stenosis was more frequently encountered when the patient's attacks consisted of symptoms suggestive of ischemia of small cortical territories with involvement restricted to the arm or leg or to dysphasia. Attacks of hemiparesis affecting face, arm and leg, or arm and leg were less often associated with carotid stenosis. If patients described any attacks of a restricted nature the chance of finding carotid stenosis was 47%, if not 16%. It is argued that these findings are a reflection of the varied pathogenesis of TIAs, and the relevance of this heterogeneity to the interpretation of clinical trials is briefly mentioned.

PATIENTS who describe symptoms suggestive of transient ischemia in the territory of the internal carotid artery are often referred for angiography in the search for evidence of carotid stenosis or occlusion. Certain clinical features are believed to be associated with an increased chance of positive angiographic studies. Thus the presence of a bruit, the occurrence of retinal symptoms and the brevity of attacks have all been claimed to be useful clinical pointers.

In the present study we investigated the possibility that the nature of the hemispheric symptoms might also be helpful.

Materials and Methods

The case records of 139 consecutive patients with transient cerebral ischemic attacks affecting the carotid territory referred to one of us (JM) were reviewed together with their angiographic findings. Note was taken of the symptoms described and whether more than one kind of attack occurred. For analysis patients were separated into those with a complaint of weakness or sensory disturbance in both the arm and the leg ('hemi' attacks), and those whose symptoms were restricted to either the arm or the leg, or to isolated dysphasia ('restricted' attacks).

The angiograms were assessed by one of us (MH) unaware of the clinical findings, and were classified as being normal, showing minor irregularity at the carotid bifurcation, stenosis at the bifurcation with encroachment on the lumen of at least 20%, occlusion in the neck, or intracranial abnormality. When angiograms showed abnormalities both in the neck and intracranially they were classified according to the site of the more severe lesion.

Results

In 69 patients the symptoms were confined to an arm or leg or to dysphasia ('restricted' attacks). Twenty-six had symptoms in an arm only, 7 in a leg only. Five had isolated dysphasia; 16 described simultaneous problems in an upper limb together with dysphasia. Fifteen had involvement of the ipsilateral arm and face or dysphasia.

The other 70 patients gave a history of involvement of both the arm and the leg. In 30, the face was also affected or speech was also involved. Twenty of these 70 patients, at other times, had attacks of a more restricted nature with symptoms in an arm or leg only.

Of the angiograms 50 showed carotid stenosis which was severe in 20 (60%), moderate in 9 (31–60%) and mild (20–30%) in 16. In 5 cases 'significant' stenosis was recorded by the radiologist, but the films were not available for review so the severity could not be assessed further. Five cases had carotid occlusion. Twenty-five angiograms were normal and 43 showed only minor irregularity of doubtful relevance to the symptoms.

As table 1 shows, the angiographic findings differed in those with 'restricted' attacks when compared with those with hemiparesis or hemisensory change (Chi square 10.4 p < 0.02 with DF3 as the two smallest groups were considered together). Forty-five percent of the former had carotid stenosis, 27% of the latter (Chi square 4.75 p < 0.05). The severity of the stenosis was no greater in those with restricted attacks (table 2; Chi square 0.99 DF3 N.S.). There was no difference between the restricted and more complex attacks in terms of the duration of attacks. There was no difference in angiographic findings between those whose attacks affected motor behaviour rather than sensation or in those whose symptoms affected both motor and sensory phenomena.

Some of the patients describing symptoms in the arm and the leg said that at other times they also had attacks of a restricted nature. These individuals with occasional restricted attacks were more likely to have a carotid stenosis than those with no such episodes (11/20 cf 8/50 Chi square 10.9 p < 0.01).

Overall the description of an attack of limited topographical extent i.e. less than a hemiparesis or hemisensory loss, whether or not 'hemi' attacks also occurred, carried a higher chance of the angiographic finding of carotid stenosis (42/89 cf 8/50 Chi square 13.5 p < 0.001).
TABLE 1  Relationship Between Angiographic Findings and Nature of TIAs

<table>
<thead>
<tr>
<th>Angiogram</th>
<th>TIA</th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Restricted</td>
<td>Hemiphenomena</td>
<td>Total</td>
<td></td>
</tr>
<tr>
<td>Normal</td>
<td>15</td>
<td>14</td>
<td>29</td>
<td></td>
</tr>
<tr>
<td>Atheroma</td>
<td>20</td>
<td>23</td>
<td>43</td>
<td></td>
</tr>
<tr>
<td>Stenosis</td>
<td>31 (45%)</td>
<td>19 (27%)</td>
<td>50</td>
<td></td>
</tr>
<tr>
<td>Occlusion</td>
<td>0</td>
<td>5</td>
<td>5</td>
<td></td>
</tr>
<tr>
<td>Intracranial</td>
<td>3</td>
<td>9</td>
<td>12</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>69</td>
<td>70</td>
<td>139</td>
<td></td>
</tr>
</tbody>
</table>

Chi square for table 10.4, \(p < 0.02\) DF3.
Chi square for stenosis 4.75, \(p < 0.05\).

Discussion

These studies reveal that the detailed nature of the symptoms described by patients with transient cerebral ischemic attacks do differ in those whose angiograms confirm the presence of stenosis of the internal carotid artery.

Attacks consisting of isolated dysphasia, or involvement of a single limb prove more likely to be associated with carotid stenosis than do those in which a hemiparesis occurs affecting arm and leg.

This difference is presumably a reflection of differences in pathogenesis. Small emboli originating in the diseased carotid artery can migrate into peripheral branches of the middle cerebral artery producing ischemia of only a small cortical territory. When the symptoms affect the arm and leg one might suppose that a larger embolus has occluded the trunk of the middle cerebral artery or the carotid artery has become occluded. There is evidence from a study of retinal ischemic attacks that larger occluding emboli more often originate in the heart, and this would fit in with the relative rarity of a carotid embolic source when the cerebral symptoms suggest a larger embolus.

Alternatively it is possible that the brief episodes of hemiparesis are a reflection of lacunar disease, and so less often directly related to atheroma of cervical arteries.

These findings may help anticipate the yield of angiography in TIA victims. They also highlight the heterogeneity of TIAs which is of increasing importance in the analysis of trials of medical or surgical intervention, which tend to be based on a unitary concept of pathogenesis, such as embolism from the carotid bifurcation.

Acknowledgment

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References

Clinical identification of TIAs due to carotid stenosis.
M J Harrison, R Iansek and J Marshall

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