

SHORT REPORT

Clinical features of transient monocular blindness and the likelihood of atherosclerotic lesions of the internal carotid artery

R C J M Donders for the Dutch TMB Study Group (see appendix)

Abstract

To assess which features of transient monocular blindness (TMB) are associated with atherosclerotic changes in the ipsilateral internal carotid artery (ICA), 337 patients with sudden, transient monocular loss of vision were prospectively studied. History characteristics of the attack were compared with the presence of atherosclerotic lesions of the ipsilateral ICA. All patients were directly interviewed by a single investigator. Of all patients, 159 had a normal ICA on the relevant side, 33 had a stenosis between 0%–69%, 100 had a stenosis of 70%–99%, and 45 had an ICA occlusion.

An altitudinal onset or disappearance of symptoms was associated with atherosclerotic lesions of the ipsilateral ICA. A severe (70%–99%) stenosis was also associated with a duration between 1 and 10 minutes, and with a speed of onset in seconds. An ICA occlusion was associated with attacks being provoked by bright light, an altitudinal onset, and the occurrence of more than 10 attacks. Patients who could not remember details about the mode of onset, disappearance, or duration of the attack were likely to have a normal ICA. Our findings may facilitate the clinical decision whether or not to perform ancillary investigations in these patients.

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Transient monocular blindness (TMB) is usually attributed to transient ischaemia of the retina in one eye, or part of it. These episodes are considered transient ischaemic attacks (TIAs) in the territory of the internal carotid artery (ICA).¹ Other causes of transient loss of vision include migraine, optic neuropathy, and intrinsic eye diseases.²

Patients with TMB associated with atheromatous disease have a risk of ipsilateral stroke of about 3% a year.³ This risk is two to three times lower than in patients with cerebral TIAs.⁴ Despite the lower risk, they should receive anti-thrombotic treatment and are candidates for

carotid endarterectomy in cases of a stenosis of the ipsilateral ICA of more than 70%.³

Although early identification of patients with TMB is important, it is often difficult to distinguish these patients from those with a more benign cause. The range of symptoms of TMB is wide and patients often find it difficult to describe what happened to their eyesight some time ago.^{6,7} Yet the management can only be based on the history.

To assess which symptoms of TMB are associated with large vessel atherosclerosis, we prospectively related different characteristics of the history with the presence or absence of atherosclerotic lesions in the ipsilateral ICA.

Methods

We included 337 consecutive patients who recently experienced a sudden, transient loss of vision in one eye that lasted no longer than 24 hours and was not caused by overt ophthalmological disease. Patients were initially seen by neurologists or ophthalmologists in 18 participating centres (see appendix). After written consent, all patients were questioned by direct interview about the details of their transient loss of vision by one of us (RCJMD). This detailed history was based on a standardised questionnaire. Patients were interviewed after a median period of 6 weeks after their last attack. We classified characteristics of the history according to the following characteristics: the extent of the visual field involved, visual symptoms experienced, mode and speed of onset and of disappearance of symptoms, duration and number of attacks, the performance of a cover test during attacks, and provocation by bright light only (retinal claudication⁸). If the patient did not spontaneously mention the nature of a predefined characteristic, the possibilities in that category were read out by the investigator. The patient could answer “yes”, “no”, or “I don’t know”. If the patient had no memory of certain details of the attack, those characteristics were classified as unknown. If an uncategorised symptom was mentioned by the patient, this was recorded in the patient’s own words and classified afterwards in one of the predefined categories by two of us (RCJMD, LJK), who were at that time blinded to the results of ancillary investigations. Disagreement was resolved by means of

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Table 1 Characteristics of the history of 337 patients with transient loss of vision of one eye with sudden onset that are significantly associated with the absence or presence of any atherosclerotic lesion of the ipsilateral ICA. Other characteristics were not associated with the absence or presence of any atherosclerotic lesion of the ipsilateral ICA

Characteristic	Any atherosclerotic lesion in the ipsilateral ICA				95% CI
	Presence (n=337) n (%)	Yes (n=178) %	No (n=159) %	OR*	
Altitudinal onset	57 (17)	25	8	4.1	2.0–8.7
Altitudinal disappearance	54 (16)	22	10	2.7	1.4–5.4
Unknown mode of onset	41 (12)	7	18	0.4	0.2–0.8
Unknown mode of disappearance	65 (19)	15	24	0.6	0.3–1.0
Unknown duration	8 (2)	1	4	0.1	0–1.0

ICA=internal carotid artery.

*Odds ratio (OR) for presence versus absence of any atherosclerotic lesion in the ipsilateral ICA.

discussion. To exclude intrinsic eye disease all patients were required to undergo ophthalmological examination. Duplex ultrasonography of the carotid arteries was performed in all patients to determine the presence of atherosclerotic lesions in the ICA.

We related history characteristics with the presence or absence of any atherosclerotic lesion in the ipsilateral ICA. In a secondary analysis the characteristics of the history were related with three degrees of stenosis: 1% and 69%, between 70% and 99%, and occlusion. Relations are expressed by means of odds ratios (ORs) with their corresponding 95% confidence intervals (95% CIs). History characteristics that were significantly associated with one of the categories of atherosclerotic lesions in the ipsilateral ICA were entered in a multivariate logistic regression model to assess the independence of the association. A *p* value <0.05 was considered statistically significant.

Results

Thirty three (10%) patients had an ipsilateral ICA stenosis between 1%–69%, 100 (30%) had an ipsilateral ICA stenosis of 70–99%, and 45 (13%) patients had an ipsilateral ICA occlusion.

Table 1 shows the history characteristics that were significantly associated with the presence or absence of any atherosclerotic lesions in the ipsilateral ICA. An onset or disappearance of symptoms from above or below (altitudinal) was significantly associated with the presence of an atherosclerotic lesion of the ipsilateral ICA. Attacks of which patients could not remember the mode of onset or disappearance, or the duration of symptoms, were associated with normal carotid arteries. Positive visual symptoms had no predictive value, but a history of migraine was significantly associated with a normal ICA (OR 0.3; 95% CI 0.2–0.7).

Table 2 summarises the results for patients with 1%–69% stenosis, 70%–99% stenosis, and for patients with an occlusion of the ipsilateral ICA. Attacks with an altitudinal onset of symptoms, and with an altitudinal disappearance of symptoms were strongly associated with both 1%–69% ICA stenosis, 70%–99% ICA stenosis, and ICA occlusion. An onset in seconds, and a duration of symptoms between 1 and 10 minutes were significantly associated with an ipsilateral ICA stenosis of 70%–99% only. Patients with more than 10 attacks of TMB or with retinal claudication were more likely to have an ipsilateral ICA occlusion.

On multivariate analysis, an altitudinal onset of symptoms remained in the model as a strong predictor of any atherosclerotic lesion of the ipsilateral ICA. Patients with attacks of TMB with an unknown onset were likely to have a normal ipsilateral ICA (OR 2.2, 95% CI 1.1–4.4), whereas patients with a history of retinal claudication were more prone to have an ipsilateral ICA occlusion (OR 4.9, 95% CI 1.2–9.4). With a duration of symptoms between 1 and 10 minutes patients were relatively likely to have a 70%–99% ICA stenosis (OR 1.7, 95% CI 1.0–2.8).

Discussion

To the best of our knowledge, this is the largest prospective series of patients with TMB studied to date. The “classic” pattern of brief attacks with sudden onset and an altitudinal onset or disappearance of symptoms was associated with severe atherosclerotic lesions in the ICA. Patients unable to remember details about the onset, duration, or disappearance of symptoms are likely to have a normal ipsilateral ICA.

Table 2 History characteristics of 178 patients with transient loss of vision in one eye related with the presence of 1%–69% stenosis, 70%–99% stenosis, or occlusion of the ipsilateral ICA

Characteristic	n	Ipsilateral ICA					
		1%–69% stenosis only (n=33)		70%–99% stenosis only (n=100)		Occlusion only (n=45)	
		OR*	95% CI	OR*	95% CI	OR*	95% CI
Altitudinal onset	57	7.0	2.5–19.5	3.7	1.6–8.3	3.5	1.3–9.6
Altitudinal disappearance	54	4.8	1.8–12.9	2.3	1.0–5.0	2.4	0.9–6.4
Speed of onset in seconds	94	1.2	0.5–3.1	1.8	1.0–3.2	1.3	0.6–3.0
Duration of 1–10 minutes	171	0.6	0.2–1.3	1.7	1.0–2.9	1.1	0.6–2.3
More than 10 attacks	91	0.6	0.2–1.8	1.7	0.9–3.1	2.1	1.0–4.5
Retinal claudication	13	1.2	0–12.8	1.2	0.2–7.2	4.8	1.0–25.3
Unknown mode of onset	41	0.3	0–1.3	0.5	0.2–1.1	0.2	0–1.0

ICA=internal carotid artery.

*Odds ratio(OR) for the presence of 1%–69% stenosis, ≥70% stenosis, or occlusion versus normal ICA.

Earlier studies addressed the association between TMB and the presence of atherosclerotic lesions of the (ipsilateral) carotid arteries,^{2,9-11} but only few studies related the fine details of TMB to atherosclerotic lesions of the ipsilateral ICA.^{6,12} Prediction of the presence of a high grade ICA stenosis from the type of TMB was unsuccessful in one study.¹² Another study showed that TMB with a gradual onset was associated with normal carotid arteries.¹⁵ In only a single study patterns of loss of vision in one eye were prospectively compared with the presence of (ipsilateral) carotid atherosclerosis in 100 patients.⁶ An altitudinal pattern of transient loss of vision in one eye was associated with both a carotid stenosis and a source of emboli in the heart, whereas attacks with a diffuse onset of symptoms or attacks in which the visual field concentrically constricted were more likely to be caused by a non-embolic cause.⁶ Wray classified TMB into four subtypes according to the presumed underlying pathophysiological disorder, but did not formally test this hypothesis.⁷ She considered embolism the most probable cause in patients with sudden attacks of partial or complete involvement of the visual field, which last minutes and recover completely. Attacks that come and go gradually and last longer than a few minutes were considered typical for retinal hypoperfusion, but might also be caused by retinal angiospasm if impaired retinal perfusion is absent. Patients in whom no cause could be established were categorised in a separate group.⁷

The strength of our study is that all patients were included according to strict criteria, with the exclusion of patients with intrinsic eye disease, and that all patients were systematically questioned by one and the same physician by means of a standardised questionnaire. A limitation of our study is that it was hospital based and therefore cannot be considered representative for patients with TMB who are seen in primary care; although it was a multicentre study, most patients were referred to university hospitals with facilities to perform carotid endarterectomy (see appendix).

Most neurologists and ophthalmologists consider positive visual phenomena such as scintillations and flashes of light as benign.¹⁴ However, we did not find that such positive symptoms predicted a normal ipsilateral ICA; therefore this assumption may be wrong.

In our study a rather large proportion of patients with TMB had a normal ipsilateral ICA. To explain the attacks in these patients, it might be speculated that they were caused by vasospasm.¹⁵ Such spasm may be related to migraine; the significant association of previous migraine attacks with the absence of atherosclerotic lesions in the ICA favours this argument. Alternatively, the source of emboli could have been in the heart.

In conclusion, patients with attacks of TMB that come and go from above or below, that have a speed of onset in seconds, and that last 1 to 10 minutes, are relatively prone to have a high grade stenosis of the ipsilateral ICA. Attacks of TMB provoked by bright light are

associated with an ipsilateral ICA occlusion. Patients with TMB who could not remember details of the attack more commonly have a normal ipsilateral ICA. These results may help to identify patients with sudden, transient loss of vision in one eye who have a high or a low likelihood of severe atherosclerosis of the ipsilateral ICA.

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Appendix: The Dutch TMB Study Group

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Figures in parentheses represent the number of patients from that centre

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