Short report

Unilateral occipital infarction: evaluation of the risks of developing bilateral loss of vision

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SUMMARY Fifty-eight patients with a unilateral infarction in the superficial area supplied by a posterior cerebral artery were followed (mean: 39.6 months). Thirteen (22.4%) developed cortical blindness associated with a delayed contralateral occipital infarction. Advanced age, general vascular risk, a history of strokes, Sylvian border-zone extension of the initial infarct, and an absence of improvement of initial visual field defects were strongly associated with spread to the other side. The lack of visual field improvement most accurately predicted a high risk of cortical blindness. A careful follow-up and controlled medical therapy is particularly indicated in these patients.

Cortical blindness is defined as the partial or complete loss of vision from bilateral occipital infarction often associated with disorientation, amnesia, visual fabrications, and denial of blindness.¹⁻³ We examined the question why infarcts in the areas of the posterior cerebral arteries occur bilaterally in some patients and remain unilateral in others.

Patients and methods

We studied 58 cases of infarction in the superficial area of a posterior cerebral artery (31 left, 27 right, 39 male, 19 female, average age 59 years). Two patients showed alexia without agraphia. There was deeper thalamo-mesencephalic involvement in 19 cases. Follow-up (12-72 months, mean = 39.6) was by revisiting, or by telephone interviews. Forty-eight patients were started on specific medical therapy. The following parameters were studied: thrombocytosis ($>350,000/\text{mm}^3$), increased haematocrit (male $>52\%$, female $>47\%$), increased haemoglobin (male $>177 \text{ mg/dl}$, female $>157 \text{ mg/dl}$), known hypertension ($>160/90 \text{ mmHg}$), cardiac ischaemia (on ECG or clinical grounds), diabetes, increased cholesterol ($>6.5 \text{ mmol/l}$), smoking ($>8 \text{ cigarettes/day}$), family history of cardiac or cerebrovascular disease, previous occurrence of brain infarct, former vertebrobasilar insufficiency, borderzone extension of infarction into the Sylvian area (evidenced by CT scan and clinical features), association of thalamo-mesencephalic involvement, nature of initial visual

disturbance (hemi/quadrantopia, alexia), medical therapy, improvement of initial visual field defect.

Global disability at the end of the follow-up was evaluated according to the Ad Hoc Committee for Cerebrovascular Diseases (class I-IV).⁴ The Exact Probability Fisher Test was used for statistical analysis.

Results

(a) Clinical follow-up

Nine patients died one day to 4 years after initial infarction. Delayed Sylvian stroke occurred in three cases. Visual field disturbances quickly improved or disappeared within the first 3 months in 32 patients.

Forty-five patients (29 (64.4%) male: 16 (35.6%) female) remained with a unilateral occipital lobe infarction (77.6%, mean age = 55.8 years (19-78), but eight of the patients were under 35). Mean follow-up duration was 42.1 months. Four deaths occurred from cardio-pulmonary causes.

Thirteen patients (10 (76.9%) male: three (23.7%) female) later suffered a contralateral occipital infarction (22.4% mean age = 69.2 years (63-78), with the exception of a woman of 35). Mean follow-up duration was 31.1 months. Five deaths occurred from cardio-pulmonary causes. The contralateral occipital stroke occurred within 2 days to 12 months (mean: 4.2 months). Six patients became totally blind, whereas seven showed a partial preservation of vision. Spatial disorientation, visual fabrications, mnesic disturbances and denial of blindness were present in seven patients (two with total blindness, five with partial blindness).

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(b) *Analysis of risk factors and clinical parameters:*

In none of the patients was there thrombocytosis, increased haematocrit or haemoglobin. Factors that were analysed are summarised in the table. No significant difference was found between the unilateral and bilateral groups in terms of sex, side of lesion, nature of the initial visual symptoms, hyperlipaemia, vertebrobasilar insufficiency, or involvement of the

deep area supplied by the posterior cerebral artery. On the other hand older age, absence of improvement of the initial visual field defect, presence of an extension of the infarct towards the Sylvian area, former stroke, hypertension, cardiac disease, smoking, diabetes, and a family history of vascular disease were significantly more frequent in the bilateral group. The presence of two or more "risk factors" (hypertension, cardiac disease, diabetes, hyperlipaemia, or smoking) was strongly associated with a bilateral occipital stroke. No significant difference was found between the patients with total or partial blindness. It was the absence of visual field improvement that most accurately predicted the probability of subsequent bilateralisation (3.1% of cases with visual field improvement).

Two of the eight patients aged under 35 years did not improve their visual field defect, but none developed a contralateral stroke. In these patients it is probable that younger age (<60) was a more important prognostic factor than visual recovery.

(c) *Functional evolution:*

Bilateralisation of occipital stroke was associated with diminished functional ability at the end of the follow-up. 86% (12/14) of the severely impaired (class IV) had bilateral infarction, whereas among the patients without or with mild impairment (classes I and II) none showed bilateralisation. 65% (13/20) of classes III and IV taken together belonged to the bilateral group. All of the patients with bilateral infarction but only 15.5% (7/45) of those with unilateral infarction belonged to classes III and IV. Of those in the latter group 33.3% (15/45) had a class I and 51.1% (23/45) had a class II disability.

Discussion

Our study shows the association of older age, family history of vascular disease, hypertension, cardiac disease, smoking, diabetes, border-zone extension of infarct towards the Sylvian area, and absence of visual field improvement, with the occurrence of a contralateral occipital stroke in patients with a unilateral occipital infarction. Many of these factors increase the occurrence of cerebrovascular disease generally,⁵ so it is not surprising to find their association with bilateral posterior cerebral artery strokes. However, it should be pointed out that there were only three delayed Sylvian strokes, vs 13 contralateral occipital infarctions, suggesting that the risk factors might not have the same consequences in occipital stroke patients.

We found a clear-cut association between the occurrence of bilateral occipital infarction and a lack of improvement of the initial visual field defect, and

Table Probability of a contralateral occipital infarction developing in 58 cases of unilateral occipital infarction

	Probability of contralateral occipital infarction: 13/58 cases (22.4%)	p
sex: male	25.6% (10/39)	= 0.3
female	15.8% (3/19)	
age < 60	4.8% (1/21)	< 0.02
(yr) ≥ 60	32.4% (12/37)	
< 65	6.5% (2/31)	= 0.02
≥ 65	40.7% (11/27)	
< 70	9.1% (4/44)	= 0.01
≥ 70	64.3% (9/14)	
side: right	22.2% (6/27)	= 0.6
left	22.6% (7/31)	
initial visual disturbance:		
hemianopia	22.6% (12/53)	= 0.7
quadrantopia	25% (1/4)	> 0.6
no symptom	0% (0/1)	> 0.7
alexia without agraphia	0% (0/1)	> 0.6
improvement of initial visual disturbance	3.1% (1/32)	< 0.001
thalamo-mesencephalic involvement	36.8% (7/18)	= 0.07
Sylvian border-zone involvement	62.5% (10/16)	≪ 0.001
symptoms of vertebrobasilar insufficiency	40% (2/5)	= 0.3
family history of vascular disease	40% (8/20)	< 0.03
former stroke	54.5% (6/11)	= 0.01
Risk factors (RF)*:		
hypertension	47.4% (9/19)	= 0.003
cardiac disease	41.7% (10/24)	= 0.004
smoking	39.1% (9/23)	< 0.02
diabetes	41.7% (5/12)	< 0.001
hyperlipaemia	30.1% (4/13)	> 0.05
≤ 1 RF*	3.3% (1/30)	= 0.0003
≥ 2 RF	42.9% (12/28)	
≤ 2 RF	13% (6/46)	= 0.003
≥ 3 RF	42.9% (12/28)	
≤ 3 RF	19.6% (9/46)	= 0.02
≥ 4 RF	33.3% (4/12)	

*Family history is not included as it could not be obtained in all cases.

borderzone extension of the original infarct towards the Sylvian area. These parameters are not general vascular risk factors, but they do considerably favour the development of cortical blindness after a unilateral posterior cerebral artery stroke. The absence of visual field improvement has the most predictive value. Such a risk decreases considerably after twelve months, as in no case was there the development of bilateral vision loss after this period.

Of those patients whose occipital infarctions remained unilateral, 84.4% belonged to the non or mildly disabled functional classes, whereas 92.3% of the patients who developed cortical blindness belonged to the severely disabled class. Thus, bilateralisation means a poor functional prognosis.

We found a relatively high percentage of delayed contralateral occipital infarction (22.4%). This frequency has not been reported in the literature. Only one case rapidly developed bilateral blindness (within two days). Supporting this are the descriptions in different studies of premonitory visual field defects.⁶⁻⁸

The institution of medical therapy directed against the risk factors did not seem to affect the probability of a contralateral occipital stroke. None the less, the high-risk patients should be carefully followed. The prevention of cortical blindness may be possible with

an early detection of the symptoms of bilateralisation and the institution of an adequate therapy.

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