longer be dissociated from public health programmes for preventing and managing infection with HIV-I and HIV-II.

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Prognosis and prognostic factors of retinal infarction: a prospective cohort study

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Abstract

***Objective*—To determine the prognosis and adverse prognostic factors in patients with retinal infarction due to presumed atheromatous thromboembolism or cardiogenic embolism.**

***Design*—Prospective cohort study.**

***Setting*—University hospital departments of clinical neurology.**

***Patients*—99 patients with retinal infarction, without prior stroke, referred to a single neurologist between 1976 and 1986 and evaluated and followed up prospectively until death or the end of 1986 (mean follow up 4·2 years).**

***Interventions*—Cerebral angiography (55 patients), aspirin treatment (37), oral anticoagulant treatment (eight), carotid endarterectomy (13), cardiac surgery (six), and peripheral vascular surgery (two).**

***Main outcome measures*—Death, stroke, coronary events, contralateral retinal infarction; survival analysis confined to 98 patients with retinal infarction due to presumed atheromatous thromboembolism or cardiogenic embolism (one patient with giant cell arteritis excluded), and Cox's proportional hazards regression analysis, including age as a prognostic factor.**

***Results*—During follow up 29 patients died (21 of vascular causes and eight of non-vascular or unknown causes), 10 had a first ever stroke, 19 had a coronary event, and only one developed contralateral retinal infarction. A coronary event accounted for more than half (59%) of the deaths whereas stroke was the cause of only one death (3%). Over the first five years after retinal infarction the actuarial average absolute risk of death was 8% per year; of stroke 2·5% per year (7·4% in the first year); of coronary events 5·3% per year, exceeding that of stroke; and of stroke, myocardial infarction, or vascular death 7·4% per year. Prognostic factors associated with an increased risk of death were increasing age, peripheral vascular disease, cardiomegaly, and carotid bruit. Adverse prognostic factors for serious vascular events were increasing age and carotid bruit for stroke, and increasing age, cardiomegaly, and carotid bruit both for coronary events and for stroke, myocardial infarction, or vascular death.**

***Conclusions*—Patients who present with retinal infarction due to presumed atherothromboembolism or cardiogenic embolism are at considerable risk of a coronary event. The risk of stroke, although high, is not so great. Not all strokes occurring after retinal infarction relate directly to disease of the ipsilateral carotid system, although this is probably the most common cause. Few patients experience contralateral retinal infarction. Non-arteritic retinal infarction should be diagnosed or confirmed by an ophthalmologist, and the long term care of patients with the condition should involve a physician who has an active interest in managing vascular disease.**

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Introduction

Retinal infarction may complicate a wide variety of diseases of the blood, heart, and arteries, and many patients with retinal infarction have a similar vascular disease and risk factor profiles to those with cerebral infarction.[1-15] Because the effects of retinal infarction may be observed directly with an ophthalmoscope the condition may be regarded as an easily visible surrogate for cerebral infarction. Despite this, far less is known about the cause(s) and prognosis of retinal infarction. We have no sound data which allow us to advise patients about the risk of blindness in the other eye, stroke, or other serious vascular events such as myocardial infarction and sudden death of presumed cardiac cause. No prospective studies, and only a handful of retrospective studies, are available.[3 4 13 16-23]

We report a prospective study of 98 patients who presented with retinal infarction without previous stroke, in whom complete follow up was achieved. Our aim was to determine the prognosis of retinal infarction for serious vascular events and to identify any prog-

nostic factors which might help to predict which patients are at high, or low, risk.

Patients and methods

Between 1977 and 1986 we assembled an inception cohort of 99 consecutive patients presenting with retinal infarction to one of us (CPW). Patients were referred at an early and uniform point (inception) in the course of their disease, soon after the onset of their symptoms and before any stroke had occurred. The referral source was the Oxford Eye Hospital in 91% of cases, the patient's general practitioner in 5%, and another hospital consultant in 4%. Standard diagnostic criteria and outcome criteria were applied. One patient with temporal arteritis was excluded from analysis. The day of neurological consultation with CPW was taken as day 0 of follow up. Complete follow up was achieved either by regular clinical review at four to 12 month intervals until the patient died or until the end of 1986, when CPW left Oxford, or, in a few cases, by writing to the patients or their general practitioners, or both. All patients experiencing a vascular event of any consequence were evaluated by CPW, and all available records, including postmortem reports, were examined. A second observer (GJH) reviewed the records at the completion of the study.

Cerebral angiography was performed if a patient was a potential candidate for carotid endarterectomy on clinical grounds and had agreed to consider accepting surgery if the angiogram showed a potentially operable lesion of the internal carotid artery. The decision to proceed to cerebral angiography was not influenced by the presence or absence of a carotid bruit. Non-invasive carotid ultrasonographic studies were not available to screen and select patients for angiography. Diameter stenosis of the internal carotid artery origin on each side was calculated.[24]

STATISTICAL ANALYSIS

Analysis of survival of the population with retinal infarction was confined to the 98 patients (99%) in whom the cause of infarction was considered to be atheromatous arterial thromboembolism or cardiogenic embolism. Data were analysed in two steps. Firstly, the Kaplan-Meier product limit technique was used to calculate survival curves.[25] Actuarial estimation of the probability of an event, such as stroke, among patients with retinal infarction is a method of assessing the occurrence of only that event. Those patients who died of a cause other than stroke were removed from the denominator for the observed interval. This denominator then represented only those patients at continuing risk of a stroke. A 95% confidence interval was calculated for the value of the survival proportion at particular times during the follow up.[26] The average annual rate of each outcome event (z%) was calculated using the 5 year event rate (y) (derived from the Kaplan-Meier data) as follows: $z=100(1-(1-y/100)^{1/5})\%$. A linear approximation of the average annual rate (the arithmetic average annual risk) may be obtained by simply dividing y by 5, but this is only fairly accurate if y is very small.

The influence of potential prognostic factors (all those listed in table I, aspirin treatment, anticoagulation and carotid endarterectomy) on the rates of outcome events (such as death or stroke) was determined by analysing the survival data with a computerised biomedical data program (BMDP) statistical package (P2L), based on the stepwise Cox's proportional hazards regression model.[27] Proportional hazards analysis provides estimates of regression parameters (the β coefficient) for each independent (prognostic) variable with an associated standard error and p value. If the ratio of the β coefficient to the standard error exceeds 1·96 then the p value is less than 0·05. With the exception of age, univariate factors which were significant at the 0·05 level were included in the model, and a forward stepwise procedure was used, beginning with the most significant variable and then the other univariate factors, in order of significance. Variables were excluded if the p value exceeded 0·05. Age was forced into the model for reasons of medical commonsense as it was not always selected in the stepwise process.

The hazard ratio is the ratio of the rate of an outcome event (such as death or stroke) occurring at some point in time (conditional on survival until that time) in the follow up of a group of patients with a given (prognostic) factor to the rate of the outcome event occurring in a group of patients without that factor. This was derived by calculating the exponential of the β coefficient: suppose h (t;**z**) is the hazard rate for a subject with prognostic factor **z**; the proportional hazards model is given by: $h(t;\mathbf{z})=h_0(t)\exp(\mathbf{B}'\mathbf{z})$, where **B** is a vector of unknown regression coefficients and $h_0(t)$ is an unknown hazard function for an individual with prognostic vector $\mathbf{z}=\mathbf{0}$. The 95% confidence interval of the hazard ratio was calculated by: exponential (β coefficient $\pm$ 1·96 $\times$ standard error).

DEFINITIONS

Retinal infarction—An acute painless and persistent (beyond 24 hours) monocular loss of visual acuity or visual field with ophthalmoscopic findings of pallor of all or a section of the retina. Additional findings often included an afferent pupillary defect, embolic material in the retinal arteries or arterioles, or a cherry-red spot over the fovea in cases of central retinal artery occlusion.

Central retinal artery occlusion—Acute loss of vision with discontinuity in the circulation of the retina or segmentation of the blood column (box-car segmentation) in the veins or, later, attenuation of the retinal arteries, arterioles, and veins; pallor of the retina (especially in the thickest region around the macula, due to cloudy swelling of the ganglion cells); and a central cherry-red spot (due to accentuation of the normal fovea, which is devoid of ganglion cells, by the opalescent halo). If a portion of the retina was supplied by a cilioretinal artery fed from the choroidal circulation, perception of light or of hand movement may have been preserved in a small central segment of the visual field.

Branch retinal artery occlusion—Ophthalmological evidence of embolic occlusion of a branch of the central retinal artery together with oedema and infarction of the distal retina and visual field abnormalities corresponding to the distribution of retinal oedema and infarction. Visual loss was variable in site and severity.

Atheroma—A clinical diagnosis whereby structural change (such as hardening and thickening) in the arterial wall, causing luminal stenosis and thromboembolism, was the presumed cause of the target organ ischaemic syndrome. This was based on the absence of any other identifiable causes such as inflammatory arterial disease, cardiogenic embolism, hypercoagulability, and trauma.

Cardiogenic embolism—A clinical diagnosis favoured by the occurrence of retinal infarction in a relatively young patient (less than about 50) with no other vascular diseases or risk factors or if a probable cardiac source of embolism was shown by clinical examination, electrocardiography, chest radiography, or echocardiography.

Major stroke—An acute disturbance of focal neurological function with symptoms lasting more than one week and thought to be of vascular origin.

Minor stroke—An acute disturbance of focal neurological function with symptoms lasting more than 24

hours and less than one week and thought to be of vascular origin.

Results

BASELINE CHARACTERISTICS

Like most hospital series of patients with retinal infarction, the patients were elderly and characterised by a high prevalence of vascular disease and risk factors (table I). A probable cardiac source of embolism was present in 24 patients, of whom 18 had valve disease confirmed both clinically and echocardiographically and six had had a myocardial infarction within the previous year. The valve lesions were aortic stenosis (seven patients), aortic sclerosis (seven), and mitral calcification (four). Seven patients had other cardiac abnormalities, such as mitral valve prolapse, which were not considered to be of probable aetiological significance. Atheroma of the ipsilateral carotid system (either proximally or distally) was the presumed cause of retinal infarction in 74 patients and coexisted with a probable cardiac source of embolism in seven other patients (table I). The internal carotid artery on the symptomatic side was studied angiographically in 55 patients, and in 25 the contralateral internal carotid artery was also examined because a potentially operable lesion was shown on the symptomatic side (fig 1).

At the time of the retinal infarction 21 patients were being treated with drugs for hypertension, two were taking aspirin, one dipyridamole, and two oral anticoagulants. The median time interval between the infarction and evaluation by the neurologist (CPW) was 28 days (interquartile range 14 to 49 days). At that time 30 patients had noticed some improvement in vision in the symptomatic eye, but complete recovery was uncommon.

TABLE I—*Baseline characteristics of 98 patients with retinal infarction. Figures are numbers of patients except when otherwise stated*

	Value
Pathogenesis:	
Probable atheroma	74
Probable cardiogenic embolism	17
Probable atheroma and cardiogenic embolism	7
Visible emboli	43
Platelet	3
Calcific	6
Cholesterol	26
Other (fibrin, tumour, etc)	8
Mean (SD) age (years)	64 (14)
Men	55
Current smokers within previous 12 months	39
With treated hypertension or diastolic blood pressure ≥100 mm Hg, or both	56
Mean (SD) systolic blood pressure (mm Hg)	166 (31)
Mean (SD) diastolic blood pressure (mm Hg)	91 (13)
Peripheral vascular disease*	13
Ischaemic heart disease†	18
Valve disease	25
Atrial fibrillation‡	4
Ventricular hypertrophy§ or strain‖ on electrocardiography	18
Cardiomegaly¶	16
Diabetes mellitus**	5
Arcus senilis	56
Carotid bruit[24]	31
Carotid bruit over symptomatic carotid artery	26
Mean (SD) blood glucose (mmol/l)††	5·3 (1·8)
Mean (SD) plasma cholesterol (mmol/l)††	7·3 (1·8)
Hypercholesterolaemia‡‡	53
Median % symptomatic internal carotid artery stenosis (n=55 arteries)	24
Median % asymptomatic internal carotid artery stenosis (n=25 arteries)	33

*Intermittent claudication, ischaemic rest pain, or previous peripheral vascular surgery.
†History of angina, myocardial infarction, or coronary artery bypass surgery.
‡Confirmed by electocardiography before or at presentation.
§Voltage sum of the tallest R wave and the deepest S wave ≥40 mm in the precordial leads, provided QRS duration <0·10 s.
‖ST segment depression and T wave inversion in leads I, II, AVL, V4-6.
¶Cardiothoracic ratio ≥0·5 on chest radiograph.
**Fasting plasma glucose ≥8·0 mmol/l, random plasma glucose ≥11·0 mmol/l, or treated diabetes.
††Fasting specimen 92%, non-fasting specimen 8%.
‡‡Plasma cholesterol ≥7·0 mmol/l or treated hypercholesterolaemia.

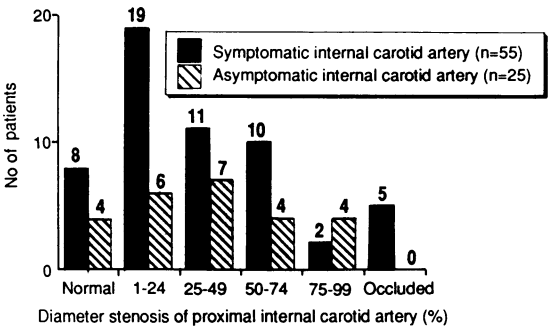

FIG 1—*Degree of diameter stenosis of the origin of the internal carotid artery on carotid angiography on side ipsilateral (symptomatic) and contralateral (asymptomatic) to retinal infarction in 55 patients with retinal infarction*

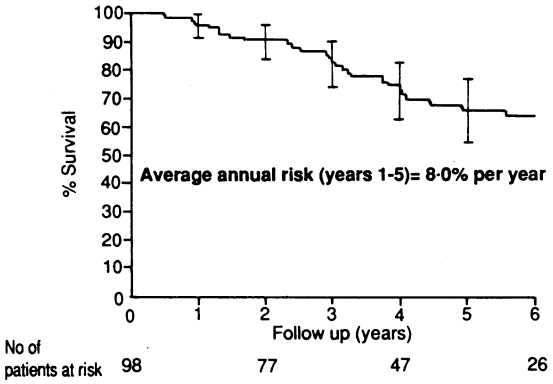

FIG 2—*Kaplan-Meier survival curve for first six years after retinal infarction. Bars are 95% confidence intervals*

TABLE II—*Major outcome events during follow up of 98 patients with retinal infarction*

Outcome event	No (%) of patients	95% Confidence interval (%)
Retinal infarction (contralateral)	1	0 to 6
Death	29	21 to 39
Vascular*	21	13 to 29
Fatal stroke	1	
Coronary	17	
Other†	3	
Non-vascular‡	7	3 to 14
Not known	1	0 to 6
Stroke	10	4 to 16
Coronary event§	19	12 to 27
Stroke or myocardial infarction or vascular death	30	21 to 40

*Death due to stroke, myocardial infarction, sudden presumed cardiac death, cardiomyopathy, cardiac failure, rheumatic heart disease, or ruptured aortic aneurysm.
†Death due to valve disease (two patients) and ruptured aortic aneurysm (one).
‡Death due to some disorder not related to vascular disease—for example, cancer, suicide, pneumonia, etc.
§Definite myocardial infarction, sudden death due to presumed or known ischaemic heart disease, or death due to cardiac failure as a consequence of ischaemic heart disease.

FOLLOW UP

Complete follow up was obtained until either the patient's death or until the end of 1986. Mean follow up was 4·2 years (range one to 10 years). At some stage during follow up 37 patients were taking aspirin and eight were treated with oral anticoagulants; 13 patients had carotid endarterectomy, one had coronary artery bypass surgery, five had cardiac valve surgery, and two had peripheral vascular surgery.

During follow up transient ischaemic attacks of the brain occurred in 10 patients and of the eye (amaurosis fugax) in two (definitions have been given previously[10 11 24]); hypertension requiring treatment developed in nine patients, angina in five, atrial fibrillation in six, diabetes mellitus in one, intermittent claudication in three, and cardiac failure in 12.

Table II shows the numbers of each of the major outcome events that occurred during follow up; table

III the average annual risk and actuarial risk at one year and five years of each major event; figures 2 to 5 the Kaplan-Meier survival curves for each major event; and table IV the results of the proportional hazards analysis.

Stroke—Twelve strokes (three minor, eight major non-fatal, and one fatal) occurred in 10 patients. Clinically there was no doubt about the diagnosis of stroke (previous definitions[28 29]); three strokes were definite infarcts, four were probable infarcts, and in five cases the pathological diagnosis was uncertain because the patient had neither computed tomography nor a postmortem examination (previous definitions[30]). There were no definite intracerebral haemorrhages. Stroke occurred in the same vascular territory as the presenting retinal infarction in eight of the 10 patients and only one was of lacunar type.[31] Two patients had a stroke after angiography and one other a stroke within 24 hours after carotid endarterectomy. These strokes were included in the total of 12 and in the survival analysis.

Coronary events—Nineteen patients had one or more coronary events. Non-fatal myocardial infarction occurred in six patients (four of whom had a later fatal myocardial infarction), fatal myocardial infarction in eight (one patient died of a myocardial infarction within 24 hours of peripheral vascular surgery; these results were included in the survival analysis), sudden death in seven, and fatal cardiac failure in two. Cardiovascular death unrelated to ischaemic heart disease occurred in two patients and one patient died of a ruptured aortic aneurysm.

Discussion

STUDY SAMPLE

Most patients were referred from the Oxford Eye Hospital, whose ophthalmologists were aware of the interest of one of us (CPW) in vascular disease. Consequently, patients with retinal infarction due, for example, to coagulopathies and arteritides, who were managed by the ophthalmologists, were not represented in this cohort, nor probably were older patients, who are less likely to be referred to hospital. The prevalence of other vascular diseases and risk factors was similar to that in patients with transient or permanent ischaemia of the brain.[9-11]

TABLE III—*Summary of risks of the different outcome measures for 98 patients with retinal infarction*

Outcome	Risk (%) 1 Year	Risk (%) 5 Years	95% Confidence interval	Average annual risk (years 1-5)	95% Confidence interval
Death	4·3	34·1	23·1 to 45·1	8·0	5·1 to 11·3
Stroke	7·4	11·7	3·9 to 19·5	2·5	0·7 to 4·2
Coronary event	5·4	24·0	13·8 to 34·2	5·3	2·9 to 8·0
Disabling stroke or vascular death	6·4	27·2	16·4 to 38·0	6·2	3·5 to 9·1
Stroke, myocardial infarction, or vascular death	13·8	32·0	21·3 to 42·7	7·4	4·7 to 10·5
Stroke, myocardial infarction, or death	13·8	39·9	31·5 to 48·3	9·7	7·3 to 12·4

TABLE IV—*Proportional hazards analysis in 98 patients with retinal infarction*

Outcome event and prognostic factors	β Coefficient	Coefficient/SE	Hazard ratio	95% Confidence interval of hazard ratio
Death:				
Peripheral vascular disease	1·65	3·46	5·2	2·1 to 13·3
Cardiomegaly	1·74	3·42	5·7	2·1 to 15·5
Carotid bruit	1·47	3·08	4·3	1·7 to 11·0
Age (10 year interval)	0·08	0·29	1·1	0·9 to 1·6
Stroke:				
Carotid bruit	1·63	2·32	5·1	1·3 to 20
Age (10 year interval)	0·65	1·38	1·9	0·8 to 4·8
Coronary events:				
Cardiomegaly	1·27	2·41	3·6	1·3 to 10
Carotid bruit	1·02	2·05	2·8	1·1 to 7·3
Age (10 year interval)	0·37	1·10	1·5	0·8 to 2·8
Stroke, myocardial infarction, or vascular death:				
Carotid bruit	1·17	2·93	3·2	1·5 to 7·1
Cardiomegaly	1·02	2·44	2·8	1·2 to 6·3
Age (10 year interval)	0·41	1·56	1·5	0·9 to 2·5

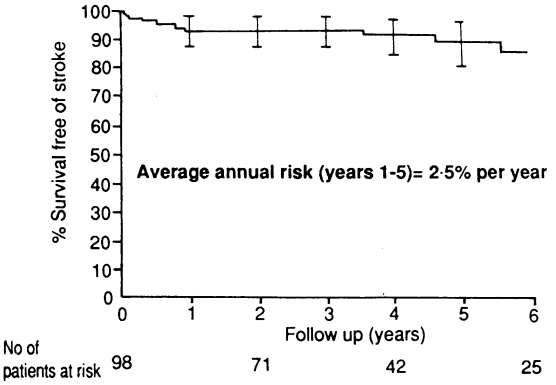

FIG 3—*Kaplan-Meier survival curve of survival free of stroke (excluding patients dying of other causes) during first six years after retinal infarction. Bars are 95% confidence intervals*

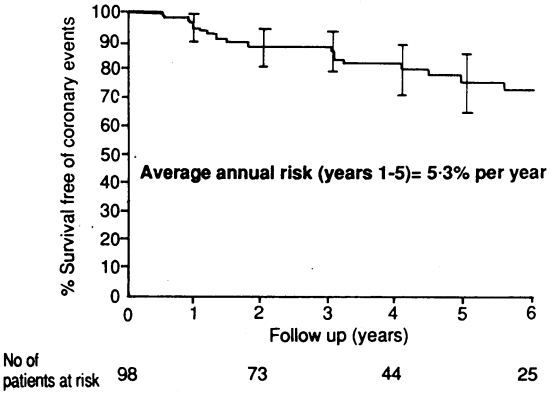

FIG 4—*Kaplan-Meier survival curve of survival free of coronary events (excluding patients dying of other causes) during first six years after retinal infarction. Bars are 95% confidence intervals*

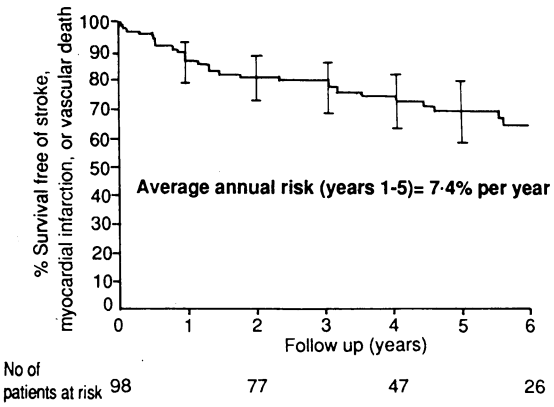

FIG 5—*Kaplan-Meier survival curve of survival free of a stroke, myocardial infarction, or vascular death (excluding patients dying of other causes) during first six years after retinal infarction. Bars are 95% confidence intervals*

DIAGNOSIS

The diagnosis of retinal infarction was unlikely to be susceptible to much variation within or among observers because the initial diagnosis was by ophthalmologists and was confirmed by a single neurologist experienced in assessing patients with neurovascular disease (CPW) and the records were reviewed by a second observer (GJH) at the completion of the study.

AETIOLOGY

Retinal infarction is caused by complete occlusion of the central retinal artery or its tributaries for about an hour or more.[8] Occlusion may be due to thrombosis,

embolism, or possibly vasospasm. Thrombotic occlusion has four principle causes: (*a*) atheroma, (*b*) hypertensive small vessel disease, (*c*) retinal endarteritis, and (*d*) hydrostatic occlusion due to either high intraocular pressure or low retinal blood pressure.[8 32] Embolic occlusion is believed to be the most common cause of retinal infarction,[32] but differentiating between thrombosis and embolism is difficult unless visible emboli have escaped into the retinal circulation and lodged there. Potential sources of embolism include the venous system (through a right to left cardiac shunt), the heart, and the ipsilateral internal carotid artery. In our series 31 patients (32%) had evidence of a potential cardiac embolic source, but in only 24 was the heart a likely source of embolism to the eye; it was the sole cause in 17 patients and coexistent with carotid atheroma in another seven. Atheromatous disease of varying severity was present at the origin of the internal carotid artery on the symptomatic side in 47 of the 55 patients (85%) who underwent carotid angiography; the median diameter stenosis was 24% (fig 1, table I). Similar findings were reported in other studies.[2 5 7] The high prevalence of proximal internal carotid disease on the asymptomatic side (fig 1, table I) probably reflects some selection bias as the asymptomatic carotid system was usually studied angiographically only if a potentially operable lesion had been shown on the symptomatic side. Although these results were probably influenced by sampling error, selection bias, and referral bias, an association between extracranial internal carotid artery disease and retinal infarction seems to be more than coincidental, in the same way as the association of internal carotid artery disease with transient ischaemic attack and cerebral infarction.

It has been suggested that the aetiology of retinal infarction may be suspected from ophthalmoscopic evidence of either branch retinal artery occlusion or central retinal artery occlusion (branch occlusion being more often embolic from cardiac or carotid disease[6 33]), but other studies did not support this hypothesis.[7] We did not subclassify our patients according to the presence of central or branch retinal artery occlusion. We were interested, however, to find that among the 10 patients with a stroke that occurred during follow up after retinal infarction eight strokes were in the same vascular territory as that of the retinal infarct and only one of these was of lacunar type (and therefore due to presumed small vessel disease). This observation suggests that atheroma of the origin of the internal carotid artery on the symptomatic side may have been important in the pathogenesis of both the retinal infarction and subsequent cerebral infarction in up to seven patients, but the numbers were too small for definite conclusions to be reached.

PROGNOSIS

There are many causes of retinal infarction and the prognosis of patients with retinal infarction due to different disorders must differ. To overcome this problem of aetiological (and therefore prognostic) heterogeneity the survival analysis of our cohort of patients was confined to a more homogeneous group pathophysiologically—that is, patients with retinal infarction without a haematological or inflammatory vascular disorder presumed to be due to atherothromboembolism or cardiogenic embolism.

The prognosis for vision, at least in the contralateral eye, was very good. Patients were at much higher risk of other serious vascular events such as stroke, myocardial infarction, or vascular death (about 7·4% per year). Most vascular events were cardiac, and the major cause of death was a coronary event (59%). Two thirds of the strokes were non-disabling.

It is difficult, if not impossible, to make valid comparisons between this study and previous studies because of the heterogeneity in terms of patient selection, methods of ascertaining cases, adequacy of follow up, definition of outcome events, and methods of analysis. Almost all previous studies[3 16-19] included patients with transient retinal ischaemia or asymptomatic retinal emboli, or both, together with patients with retinal infarction; all have been retrospective and hampered by incomplete follow up,[3 4 16-19] and only two have used actuarial methods of analysis.[16 17] In these two studies, which comprised 86 patients[16] and 66 patients[17] respectively, the arithmetic average annual mortality was about 4·6% per year[16] and 3% per year,[17] and the arithmetic average annual risk of stroke was about 3% per year.[7]

PROGNOSTIC FACTORS

The vascular outcome event that occurred most commonly (and was therefore least susceptible to sampling error) was stroke, myocardial infarction, or vascular death. The significant adverse prognostic factors were increasing age, carotid bruit, and cardiomegaly (table IV). For example, a patient with retinal infarction, carotid bruit (hazard ratio 3·2), and cardiomegaly (2·8) was 13·4 times (3·2×2·8×1·5) more likely to have a stroke, myocardial infarction, or vascular death at any point than a patient with retinal infarction who had none of these and was 10 years younger. Carotid bruit was also a significant predictor of a stroke, as has been suggested in some previous studies.[17 18] Treatment with aspirin, anticoagulation, or carotid endarterectomy did not have a significant effect on the risk of subsequent stroke but the numbers were small. The risk of a coronary event was nearly four times greater (95% confidence interval 1·3 to 10) for patients with cardiomegaly than for patients without. Contrary to the suggestions of some authors,[16 19 34] we were unable to attribute any prognostic significance to the presence or absence of visible retinal emboli and do not think that this distinction is important; in any event retinal emboli may be asymptomatic (particularly cholesterol emboli) or symptomatic, and their detection opthalmoscopically depends on when the patient is examined, by whom, and whether the pupil is dilated. We have studied prospectively two cohorts of patients with transient monocular blindness (amaurosis fugax) due to atheromatous thromboembolism or cardiogenic embolism and, like other researchers,[34 35] found that their prognosis was better than patients with retinal infarction.[36 37] The reason for this is not entirely clear at present; it may be due to the greater age and degree of atheroma in the retinal infarction group. It seems, therefore, more sensible to classify patients according to their symptoms (that is, transient versus sustained monocular blindness) rather than according to the presence or absence of visible retinal emboli.

In conclusion, patients with non-arteritic retinal infarction have a high prevalence of vascular diseases and risk factors, a significant prevalence (about 25%) of valve disease, a higher subsequent risk of death than reported in patients with transient ischaemic attacks of the brain or eye or in community controls, and a greater risk of having an ischaemic coronary event than an ischaemic cerebral event (stroke). The prognosis for vision in the affected eye seems to be quite poor,[8] but that for vision in the unaffected eye is probably extremely good. The implications of these findings are that measures of secondary prevention should be aimed at reducing the progression and complications of generalised arterial disease, particularly coronary artery disease. Control of vascular risk factors and the use of long term antiplatelet treatment are likely to have the greatest impact whereas other more focused and invasive treatments such as carotid endarterectomy may be worth while only for a selected subgroup of patients.

The results and conclusions from this study, however, were derived from a fairly small number of patients and outcome events in one patient population. The estimates are imprecise owing to the potential for sampling error and cannot necessarily be generalised to other patient populations until they have been tested and confirmed in other studies.

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Outcome of patients with chest pain discharged from an accident and emergency department

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Chest pain is common in patients seen in the accident and emergency department. Though there is considerable information on the outcome of those patients who are admitted, little is known about those who are discharged.

Patients, methods, and results

We studied all patients with chest pain attending the accident and emergency department of the Central Middlesex Hospital over two months. Fourteen days after attendance all those patients who had been discharged home were sent a questionnaire on further treatment and the persistence of symptoms. Reminders were sent if no reply was received, and general practitioners or hospital consultants contacted if further consultation had been necessary. The casualty officers were unaware that the study was being done, and their diagnosis in the casualty notes was used as the discharge diagnosis. Unless the patient had sought further medical treatment no attempt was made to confirm or refute this diagnosis. The notes were then assessed for errors by one of us (MB) as an impartial observer.

Overall, 179 of 314 patients with chest pain were discharged during the study, as shown in the figure. Adequate information was obtained at two weeks on 174 of these patients. Electrocardiography was performed on 120 patients, 60% (68/113) of the patients under 50 years of age having this compared with 85% (52/61) of those over 50. Potential errors in diagnosis at discharge were judged to have been made in four patients. One patient presented again with a myocardial infarction, one was deemed to have been underinvestigated, and two patients were sent home with unstable angina which needed further treatment. Twenty six patients continued to have chest pain at two weeks but without any change in symptoms to suggest more serious disease.

Comment

Thrombolytic treatment is effective in reducing mortality from myocardial infarction if started early.[1] Twenty four of the 43 patients referred by their general practitioners were sent specifically or implicitly for electrocardiography and if thrombolytic treatment is to be started early this may result in unnecessary delay.