

Short report

The clinical course and prognosis of carotid artery occlusion

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SUMMARY The clinical course of 100 consecutive patients with angiographically proven carotid occlusion was reviewed. Ninety-three patients had been hospitalised for early stroke appropriate to the occlusion, and seven for transient ischaemic attacks. 68 patients were followed up from 1.5 to 5.9 years. The observed five year survival rate on an actuarial basis was 62.3%, compared to the expected rate of 90% in a matched normal population. The observed rate of recurrent stroke was 4.8% at 1 year, 12.2% at 3 years, 17.1% at 5 years. Among survivors, six patients (11.3%) developed seizures.

Conflicting reports concerning the prognosis of carotid artery occlusion have been published. Long-term observation has indicated a risk of further strokes varying between 7%¹ and 25%.² An unanswered question is whether a new stroke is more likely to occur ipsilateral to the occluded carotid; this is very important since an increasing number of patients with symptomatic carotid artery occlusion are being referred for extracranial to intracranial bypass grafts. Recently, Furlan *et al*³ reported that in patients with minimal or no neurological deficits after carotid occlusion, the risk of an ipsilateral stroke was 2% per year. In this report we review the clinical course of 100 patients with consecutive carotid occlusion and evaluate the long-term prognosis of survivors.

Material and methods

From 1975 to 1979 there were 100 patients with carotid occlusion, verified angiographically. Ages ranged from 15 to 72 years (average 51.2 years). The age and sex distribution of the patients are shown in table 1. Ten patients with carotid occlusion were under 35 years of age. These occlusions probably were not due to atherosclerosis. None of the seven women under 44 years of age was taking birth control pills. Forty-four patients had an occlusion of the right internal carotid artery, 57 of the left internal carotid, two of the common carotid (including three with bilateral

carotid occlusion); 17 patients had carotid stenosis contralateral to the occlusion.

Ninety-three patients had been hospitalised for early stroke ipsilateral to carotid occlusion; seven patients for ipsilateral transient ischaemic attack. During the acute phase of the stroke, three patients developed status epilepticus. Before the diagnosis of carotid artery occlusion was established, 30 patients had experienced transient ischaemic attacks which had occurred in 22 patients within the prior 6 months. Of the 100 patients, 13 died within the first month, 11 underwent surgery (end-arterectomy, by-pass operation) and were excluded from study. Eight could not be traced. The follow-up data were obtained in 68 patients by personal examination or by telephone interviews with the local physician (for those who had died during the follow-up period). Information included the general and neurological condition, development of convulsions, occurrence of new stroke and cause of death. The observation period (from the time that the angiography was done) ranged from 1.5 to 5.9 years. The net probability of a stroke and of death was determined by actuarial analysis⁴ starting with the date of the angiography. The survival rate observed was compared to the expected survival rate of a normal population of the same age and sex distribution.

Table Distribution by age and sex of patients with carotid artery occlusion

Age (yr)	Males	Females	Total
<35	8	2	10
35-44	6	5	11
45-54	27	6	33
55-64	33	1	34
65-74	11	1	12
All ages	85	15	100

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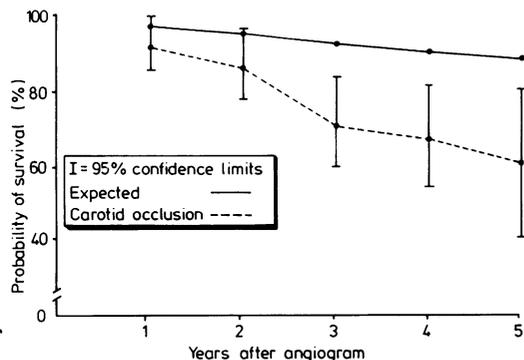


Fig 1 The observed survival after carotid artery occlusion in 68 patients. Expected survival is for a 1970–1972 Italian population of the same age and sex distribution.

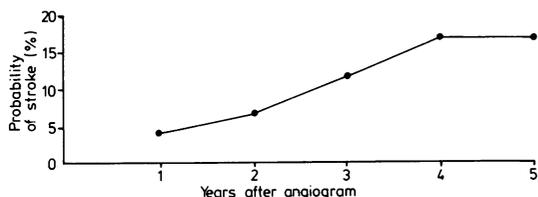


Fig 2 The observed stroke rate after carotid artery occlusion in 68 patients.

Results

None of the patients studied received anticoagulant or antiaggregant treatment after diagnosis of carotid occlusion. During the observation period 15 patients died. A stroke was the cause of death in four (26.7%), cardiac disease in eight (53.3%), with miscellaneous causes in three (20%).

The observed 5-year survival rate on an actuarial basis was 62.3% compared to the expected rate of 90.1% in a normal population (fig 1). Seven patients developed a new stroke during follow-up. On an actuarial basis, the observed stroke rate was 4.7% at 1 year, 12.2% at 3 years, 17.1% at 5 years (fig 2). Three patients suffered non-fatal strokes appropriate to the occlusion; in the four who had fatal strokes the site was not known.

There was no difference in the rate of subsequent strokes among patients in the 45–54 age group and in the 55–64 age group. None of the patients of less than 35 years of age had a new stroke. None of the five patients with transient ischaemic attacks at time of angiography suffered subsequent strokes. None of the three patients with bilateral occlusion had a second stroke. Four patients had ipsilateral transient ischaemic attacks during follow-up. Of the 68 patients studied, 40 had one or more of the following risk factors: hypertension (30.9% of cases), cardiac

disease (26.5%), diabetes mellitus (8.8%). There was no significant difference in the rate of subsequent strokes among patients with or without risk factors. Among survivors, six patients (11.3%) developed recurrent seizures, which were of a partial motor type in two cases and of a secondarily generalised partial type in four cases. Seizure disorders occurred from three to six months after brain infarction.

Discussion

This study indicated that the risk of further strokes after carotid occlusion was about 12% in the first three years and about 17% in the first five years. The presence of risk factors for cerebro-vascular diseases did not create a higher risk of further strokes. Among the survivors, 11.3% developed epileptic seizures, readily controlled with anticonvulsant medication.

The observed survival rate was lower than the probability of survival in a matched normal population. This unfavourable course was related mostly to the risk of dying of cardiac disease, as was reported by Grillo and Patterson,⁵ by Mc Dowell *et al*⁶ and Furlan *et al*.³

The natural course of carotid occlusion includes new strokes, sometimes on the side of the occlusion. Four studies furnished information on the site of new stroke. Grillo and Patterson⁵ found that all further strokes occurred contralateral to carotid occlusion. The Joint Study of Extracranial Arterial Occlusion² reported 35 ipsilateral infarctions in follow-up of 359 patients with unilateral carotid occlusion. The location was not known in 34 other fatal strokes. Furlan *et al*³ found that 11 strokes out of 17 occurred ipsilaterally to carotid occlusion in follow-up of 138 patients. Norrving and Nilsson⁷ found that two strokes out of five were appropriate to the involved artery in follow-up of 59 patients.

The different patterns of collateral circulation would seem to be a deciding factor in the subsequent evolution of carotid occlusion. Barnett⁸ has reviewed angiographically any cases which developed ischaemic events after occlusion. Embolism through collateral circulation resulting from the occluded carotid was the most frequent mechanism of new strokes, while the presence of haemodynamic factors and embolism from the tail of the thrombus were less frequent causes.

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