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## Transient ischaemic attacks: a retrospective study of 43 cases

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#### Summary

Forty-three Caucasians with transient ischaemic attacks (TIAs), all age- and sex-match controlled, were retrospectively studied. There was no difference in sex distribution. The peak age for TIAs was 55-64. The haematocrit and cholesterol levels of men were significantly elevated (0.01 > P > 0.001 for haematocrit, and 0.01 > P > 0.001 for cholesterol). Six patients were controlled hypertensives but in general there was no significant difference in the blood pressures of patients and controls. The risk factors for TIAs and strokes are discussed.

#### Résumé

Quarante-trois Caucasiens avec TIAs ont été étudié en retrospective. Les facteurs d'âge et de sexe étaient contrôlés. Il n'y avait pas de difference de distribution en ce qui concerne le sexe. L'âge maximum pour le TIAs était entre 55 et 64. Les niveaux d'hématocrite et de cholésterol des hommes étaient élevés de manière significative (0.01 > P > 0.001 pour l'hématocrite et 0.01 > P > 0.001 pour le cholésterol). Six malades étaient des hypertendus controllés mais en général il n'y avait pas une différence significative dans la pression sanguine des malades et des contrôles. Les facteurs de risque pour les TIAs et les 'strokes' ont été discuté.

#### Introduction

Several studies have highlighted the risk factors for transient ischaemic attacks (TIAs) and

\*Correspondence: Dr S. A. Bwala, Department of Medicine, College of Medical Sciences, University of Maiduguri, PMB 1069, Maiduguri, Nigeria. strokes [1]. Among these, hypertension, extracranial arterial atherothrombo-embolism and elevated haematocrit have received a lot of attention [2–4]. That strokes are preceded by TIAs is well established although the proportion of those who develop strokes varies from 4 to 39% [5,6]. The evidence for prevention has therefore been compelling. In this study we have looked at possible epidemiological patterns and have tried to identify any particular age and sex at risk in TIA patients referred to the neurology department.

#### Patients and methods

A review of the hospital records of 43 TIA patients and 43 age- and sex-matched neurological controls was done on a one-to-one basis. The neurological controls had various disorders as shown in Table 1 and were mostly out-

Table 1. Neurological disorders in control groups

Diagnosis	Male	Female	Total
Multiple sclerosis	1	5	6
Peripheral neuropathy	5	1	6
Parkinson's disease	4	2	6
Epilepsy	2	0	2
Trigeminal neuralgia	2	0	2
Dementia	1	2	3
Headaches/migraine	2	0	2
Cervical spondylosis/ lumbar disc disease	2	3	5
Brain tumor (primary/secondary)	1	2	3
Miscellaneous	2	6	8
Total	22	21	43

patients. The diagnosis of TIA was by currently accepted definition [7]. Casual blood pressure was recorded with a mercury sphygmomanometer at time of consultation. Blood for haematocrit was taken at the same time. Fasting lipids were estimated a few days later by the enzymatic colorimetric method. Most patients had computerized axial tomographic (CT) scans, unenhanced and enhanced. All patients and controls had blood sugars measured routinely.

#### Results

The age and sex distribution of TIA patients and controls is shown in Table 2. There were 22 male and 21 female TIA patients. The peak age for TIAs is 55-64 for both sexes (Fig. 1). In all, there were 26 carotid TIAs and 17 vertebrobasilar TIAs. Fasting cholesterol was significantly elevated in male TIA patients as measured by Student's t-test (t = 3.38, 0.01 > P >0.001; Fig. 2), with a mean value of 7.27 mmol/l. The mean cholesterol levels of female TIA patients was not statistically different from the controls. Haemocrit was significantly elevated in men only (t = 3.37, 0.01 > P > 0.001) with a mean value of 46.60. Six patients were known hypertensives (three males and three females) on treatment. However, there was no difference between the blood pressures of patients and controls. Seven patients had carotid bruits, but none were recorded in the controls. Twenty-seven of the 43 patients had CT-scans: of these three had cerebral infarcts without significant neurological deficit, two of the three had carotid bruit, and the third was a known hypertensive. Except one patient, who was

Table 2. Age and sex distribution of transient ischaemic attack patients and control

Age (years)	Patients		Controls	
	Male	Female	Male	Female
35–44	3	2	3	2
45-54	3	3	3	3
55-64	10	9	10	9
65-74	5	6	5	6
75-84	1	1	1	1
Total	22	21	22	21

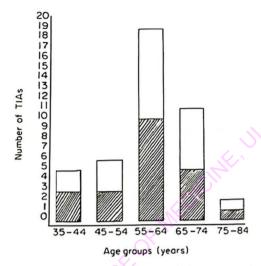


Fig. 1. Histogram of transient ischaemic attack patients showing age and sex distribution. (□) Females and (ℤ) males.

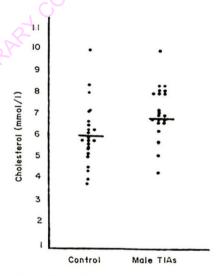


Fig. 2. Fasting cholesterol levels in male TIAs compared to control males (0.01 > P > 0.001). Lines indicate mean levels.

known to be an insulin-dependent diabetic, the rest of the patients and controls had normal routine blood sugar.

#### Discussion

There was no difference in the sex distribution of TIA patients in this study as found by some

workers [8]. However, other reviews have shown a preponderance of either sex [9,10]. Transient ischaemic attacks were predominant in the 55-64 age group. Sixty per cent of the patients had carotid TIAs indicating that preventive measures are urgently required in this group since carotid TIAs are more likely to give rise to strokes, especially in the first year of onset [11,12]. Seven patients had carotid bruits, but could not be studied further with ophthalmodynamometry and digital subtraction angiography. This is important because one-third to one-half of all significant lesions may not cause a bruit [13]. The significance of atherothromboembolism in TIAs and strokes is well known [14], with atheromatous plaques interacting with platelets in various ways such as aggregation [15]. This has led to the study of the effect of antiplatelet agents in preventing TIAs [16,17].

Another preventive measure is the reduction of the atherosclerotic process, possibly by a dietary reduction of saturated fatty acids. This may be helpful as indicated by the significantly elevated fasting cholesterol levels in men. Although there was no statistically significant difference between the mean cholesterol levels in female TIA patients and controls, all male and female TIA patients have been advised to take low saturated fatty acids, because there is the possible added benefit of a reduction in ischaemic heart disease as the two seem to be linked in pathogenesis [18]. The commonest cause of death in TIA and stroke patients in fact appears to be ischaemic heart disease [19].

Only men had significantly elevated haematocrits. Reduction of a high normal haematocrit is said to improve cerebral blood flow [4], as in the presence of a stenotic vessel wall platelet

also said to elevate the haematocrit level [21] and advice against this may be beneficial. In this study patients with haematocrits of 48 and over had regular venesection.

Evidence from several studies has shown that hypertension is an important risk factor in stroke [1]. Six of the TIA patients were known hypertensives on treatment, but there were no known hypertensives in the control group. However, the blood pressures of the TIA patients were not statistically different from the controls.

In conclusion, men have a tendency to have

elevated cholesterol levels and high normal haematocrits. Early investigation and treatment is paramount in this group in view of the known tendency to develop stroke.

#### References

- Acheson J, Hutchinson EC. Observation on the natural history of transient cerebral ischaemia. Lancet 1964;ii:871-4.
- Canadian Co-Operative Study Group. A randomised trial of aspirin and sulphinpyrazone in threatened stoke. N Engl J Med 1978;299:53-9.
- David NJ, Heyman A. Factors influencing the prognosis of cerebral thrombosis and infarction due to atherosclerosis. J Chronic Dis 1960;11: 394-404.
- Fisher CM. The use of anticoagulants in cerebral thrombosis. Neurology 1958;8:311-32.
- Friedman GD, Wilson WS, Mosier JM, et al. Transient ischaemic attacks in a community. J Am Med Assoc 1969;210:1428–34.
- Harrison MJ-G, Pollock S, Kendall BE, Marshall J. Effect of haematocrit on carotid stenosis and cerebral infarction. Lancet 1981;ii: 114-15.
- Harrison MJ-G, Pollock S, Thomas D, Marshall J. Heamatocrit, hypertension and smoking in patients with transient ischaemic attacks and in age and sex matched controls. J Neurol Neurosurg Psychiatry 1982;45:550-1.
- Imparato AM, Riles TS, Gorstein F. The carotid bifurcation plaque: pathologic findings associated with cerebral ischaemia. Stroke 1979; 10:238–45.
- Kannell WB. Current status of epidemiology of brain infarction associated with occlusive arterial disease. Stroke 1971;2:295–318.
- Karp HR, Heyman A, Heyden S, et al. Transient cerebral ischaemia. Prevalence and prognosis in a bi-racial community. J Am Med Assoc 1973;225:125–8.
- Marti-Uilalta JA, Lopez-Pousas S, Grau JM, Barraquer L. Transient ischaemic attacks. Retrospective study of 150 cases of ischaemic infarct in the territory of the middle cerebral artery. Stroke 1979;10:259-62.
- Mitchell JRA, Schwartz CJ. Relationship between arterial disease in different sites. A study of the aorta and coronary, carotid and iliac arteries. Br Med J 1962;1:1293–1301.
- Moncada S, Vane JR. Arachidonic acid metabolites and the interactions between platelets and blood vessel walls. N Engl J Med 1979;300: 1142-7.

- Harrison MJ-G. Actiopathogenesis of transient ischaemic attacks. In: Tunbridge WMG, ed. Advanced Medicine. Edinburgh: Pitman Medical. 1981: 272-3.
- Thomas DJ, Boulay GH, Marshall J, Pearson TC, Ross-Russel Symon L, Wetherley-Mein G, Zilkha E. Effect of haematocrit on cerebral blood-flow in man. Lancet 1977;ii:941-3.
- UK-TIA Study Group. Design and protocol of the UK-TIA aspirin study. In: Toguoin G, Grattini S, eds. Drug treatment and Prevention in Cerebrovascular Disorders. Amsterdam: Elsevier. 1979.
- Warlow C. Transient ischaemic attacks. In: Mathews WB, Glaser GH, eds. Recent Ad-

- vances in Clinical Neurology. Edinburgh: Churchill Livingstone, 1982:191.
- Warlow C. Cerebrovascular disease. Med Intern 1983;31:1445.
- World Health Organization 1978. Cerebrovascular disorders: a clinical and research classification. World Health Organization, Offset Publication No. 43, Geneva.
- Ziegler DK, Hassanein RS. Prognosis in patients with transient ischaemic attacks. Stroke 1973;4:666–73.
- Isager H, Hagerup L. Relationship between cigarette smoking and high packed cell volume and haemoglobin levels. Scand J Haematol 1971;8:241-4.

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