Research from the South

Strokes among black people in Harare, Zimbabwe: results of computed tomography and associated risk factors

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Abstract

Computed tomography was performed and risk factors evaluated in 100 consecutive adult patients presenting to the two teaching hospitals in Harare with a clinical diagnosis of stroke. The mean age of the patients was 52; only 28 were 65 or older. Non-stroke lesions were found in seven patients and were predicted by a recent history of convulsions ($p<0.0001$). Five lesions (four subdural haematomas and one cerebral cysticercosis) were remediable. Hypertension was present in 27 (93%) of the 29 patients with cerebral haemorrhage and in 49 (53%) of the 93 patients with stroke lesions. In 22 (45%) of these patients the hypertension had not been diagnosed, and another 22 had defaulted from treatment. All 13 patients who died before computed tomography had hypertension, and over half showed evidence of haemorrhagic stroke. There was a cardiac source for all 12 cases of cerebral embolism. In eight of the 100 patients cerebral infarction was attributed to neurosyphilis. None of the patients had clinical evidence of atherosclerosis. Smoking and oral contraceptives did not seem important risk factors for stroke.

Detection and control of hypertension remain the most important measures needed to reduce the incidence of and mortality from stroke in Zimbabwe.

Introduction

Strokes are a common cause of morbidity and mortality among black people in Zimbabwe. Although the exact incidence of stroke is not known, cerebrovascular disease accounted for 4·5% of all registered deaths of black Zimbabweans aged over 5 in 1982.[1] Strong cultural attitudes against postmortem examinations, and the lack of qualified pathologists, have contributed to the paucity of information about the pathological types of strokes seen. Knowledge of the underlying pathology is important because it correlates with outcome[2,3] and also allows a better understanding of the aetiology and pathogenesis of strokes locally. Clinical differentiation of haemorrhage from infarction is often inaccurate.[4,5]

The introduction of computed tomography has allowed accurate and non-invasive diagnosis of the type of stroke.[6] We studied the pattern of strokes in black hospital patients in Harare and determined the proportion of non-stroke lesions presenting as strokes. Although computed tomography is expensive, we thought that its use was justified in research as no information of this sort exists for southern and central Africa.

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Patients and methods

We studied consecutive adult patients admitted to the general medical wards of the two teaching hospitals in Harare with a clinical diagnosis of stroke. To allow for repairs to the computed tomographic scanner we used two separate study periods (27 November 1984 to 1 March 1985 and 3 April to 2 July 1985). The study ended when 100 scans had been performed. All patients were seen by one of two investigators (JM and IK) within 48 hours after admission. Whenever possible the initial history was confirmed. A full neurological and cardiovascular examination was performed; carotid arteries were auscultated in all cases, and peripheral pulses were carefully examined.

Hypertension was defined as a diastolic pressure greater than 95 mm Hg sustained for at least 72 hours after admission or, in cases of early death, corroborated by clinical or electrocardiographic evidence of left ventricular hypertrophy or strain, or both, or by a verifiable history of antihypertensive treatment. Evidence of atherosclerosis was taken to be the presence of carotid bruits, previous myocardial infarction, angina, claudication, or absent peripheral pulses. Stroke was defined as acute loss of focal and at times global cerebral function, the symptoms lasting more than 24 hours or leading to death, and the only apparent cause being vascular.[7] Patients with an initial diagnosis of stroke in whom the onset was atypical of a vascular episode were excluded from the study.

Patients who met the study criteria underwent computed tomography with a Siemens Somaton DR2 whole body scanner as soon as possible in the seven days after assessment. Unenhanced examinations were complemented by enhancement when clinically required; this was achieved by intravenous injection of 50 ml Conray 420 (May and Baker, United Kingdom).

Electrocardiograms obtained in each case were assessed for the presence of left ventricular hypertrophy or strain using standard criteria.[8] Echocardiography was performed when clinically indicated. Serum was tested using rapid plasma reagin and *Treponema pallidum* haemagglutination tests (Wellcome, United Kingdom). Unless contraindicated by raised intracranial pressure lumbar punctures were performed after computed tomography. Cerebrospinal fluid was examined for protein and cells and was submitted to *T pallidum* haemagglutination testing.

Significance was evaluated using the χ^2 test of association or, when appropriate, Fisher's exact test.

Results

DEATHS OCCURRING BEFORE COMPUTED TOMOGRAPHY

During the study 100 patients underwent computed tomography. A further 13 patients met the study criteria but died before scanning could be performed. All 13 had hypertension. Postmortem information was available for two of them: both had intracerebral haemorrhage with subarachnoid extension. In a further five patients lumbar puncture yielded uniformly bloodstained cerebrospinal fluid. In one patient cerebrospinal fluid obtained shortly after death was clear. We had no definite information about the remaining four patients who died before scanning. They were all thought by the admitting clinicians to have suffered cerebral haemorrhage.

PATIENTS UNDERGOING COMPUTED TOMOGRAPHY

The 100 patients who underwent computed tomography comprised 37 women and 63 men. The mean age of the whole group was 52: 39 patients were younger than 50 and 28 were 65 or older. There was a significant preponderance of women in the older group: 17 (46%) of the 37 women in the study were 65 or older compared with only 12 (19%) of the 62 men ($\chi^2=7{\cdot}46$, $p<0{\cdot}01$; one man was excluded as his age was unknown) (table I).

TABLE I—*Age and sex distributions of patients undergoing computed tomography*

	Age (years)					
	<20	20-34	35-45	50-64	≥65	Total
Men*	1	5	22	22	12	62
Women	1	6	3	10	17	37
Total	2	11	25	32	29	99

* One man was excluded as his age was unknown.

FINDINGS ON COMPUTED TOMOGRAPHY

Table II shows the computed tomography findings and associated clinical conditions. Cerebral infarction was the most common finding, followed by haemorrhage and non-stroke lesions.

TABLE II—*Findings on computed tomography and associated conditions in 100 patients presenting with stroke*

		No (%) with associated conditions		
Computed tomography findings	No of patients	Hypertension	Cardiac source of embolism	Neurosyphilis*
Cerebral infarction	62	20 (32)	12 (19)	8 (13)
Cerebral haemorrhage	29	27 (93)	1† (3)	
Non-stroke lesions	7	‡		
Subarachnoid haemorrhage	2	2 (100)		
Total	100	49 (49)	13 (13)	8 (8)

* Defined as increased protein or cells in cerebrospinal fluid in the presence of a positive serum *T pallidum* haemagglutination or rapid plasma reagin test and without another clear cause for stroke.
† Haemorrhage from a ruptured mycotic aneurysm.
‡ Two patients with non-stroke lesions had increased blood pressures on admission that were not sustained.

Cerebral infarction—Cerebral infarction was diagnosed in 62 patients. In 50 of these there was evidence of focal infarction, but in the remaining 12 no lesion or only atrophy was seen on computed tomography. In 12 patients with infarction a clear cardiac source of embolism (atrial fibrillation, cardiomyopathy, or valvular heart disease) was present. None of the patients studied showed any clinical evidence of atheromatous carotid artery disease. After exclusion of obvious sources of embolism 50 of the scanned patients were assumed to have suffered thrombotic strokes. Twenty of this group were hypertensive and two had diabetes. Ten of the 62 patients with cerebral infarction yielded positive results on serum rapid plasma reagin or *T pallidum* haemagglutination testing, or both. In two of these patients stroke could be attributed to other causes (cardiac disease and hypertension), though one had an increased cerebrospinal fluid protein concentration. The remaining eight patients, who had no other cause of the stroke, were thought to have neurosyphilis because of positive blood findings together with increased concentrations of protein or cells, or both in their cerebrospinal fluid. The result of the *T pallidum* haemagglutination test in cerebrospinal fluid was positive in seven of this group. Only one of the 38 patients who had not suffered infarction had positive results for the *T pallidum* haemagglutination and rapid plasma reagin tests in serum, though the result of the *T pallidum* haemagglutination test in cerebrospinal fluid was negative.

Intracerebral haemorrhage—Twenty nine patients who underwent computed tomography showed intracerebral haemorrhage. The mean age of this group was 49. Twenty seven of this group had hypertension. Four of these patients also showed areas of old focal infarction in addition to fresh haematomas. In one of the two normotensive patients the bleeding originated from a mycotic aneurysm. In one patient the cause of the haemorrhage was not found, though arteriography was not performed.

Non-stroke lesions—Non-stroke lesions were found in seven patients who underwent computed tomography. Subdural haematomas were found in four of these patients, cysticercosis in one, and tumours (one cystic glioma and one metastatic deposit) in two (table III). All of these patients had focal neurological deficits, and two had increased blood pressure on initial examination. We were satisfied that the onset of neurological deficit was acute in five. In two patients (one with a subdural haematoma and one with a glioma) the neurological episodes were thought to be acute by the admitting doctor, but eyewitness accounts obtained several days later suggested a longer history of behavioural change. Four of the patients with non-stroke lesions had a history of convulsions from the onset of weakness, whereas no patient with cerebral infarction or haemorrhage had suffered convulsions (Fisher's exact test, $p<0{\cdot}0001$). Two of the four patients with subdural haematomas underwent surgery and recovered motor function, one patient would not consent to surgery, and the fourth died before the operation could be performed. The patient with a malignant glioma underwent operative exploration but subsequently deteriorated.

TABLE III—*Non-stroke lesions and associated findings*

		No with associated condition		
Non-stroke lesion	No of patients	Hemiplegia or monoplegia	Dysphasia or coma	Convulsions
Subdural haematoma	4	4	2	1
Cysticercosis	1	1		1
Malignant glioma	1	1		1
Secondary deposit	1	1		1
Total	7	7	2	4

RISK FACTORS

Hypertension was present in 49 of the patients. This represents 53% of the 93 patients with true stroke lesions. Twenty two had previously been diagnosed as having hypertension but had dropped out of treatment. For another 22 hypertension was diagnosed for the first time during the admission for stroke. Thirty nine of the patients with hypertension showed clinical or electrocardiographic evidence of left ventricular hypertrophy or strain, or both, while 10 did not. There were no significant differences in the distributions of these cardiovascular findings, sex, or age between patients with hypertension who had suffered cerebral infarction and those who had suffered haemorrhage. Thus 23 (80%) of the 29 patients with hypertension and intracerebral subarachnoid haemorrhage and 16 (80%) of the 20 patients with hypertension and cerebral infarction showed evidence of left ventricular abnormalities.

Fourteen patients were cigarette smokers, though only two smoked more than 20 cigarettes a day. There was no significant difference in the proportions of smokers between those who had suffered thrombosis and those who had suffered a haemorrhage. Three patients had diabetes. No patient in the study was taking oral contraceptives.

Discussion

Strokes are a common cause of admissions to general medical wards in Harare. In about six months 113 cases were seen. Most of our patients were potentially economically active: their mean age was 52, and less than a third were 65 or older. In comparison, 54% of all patients in the World Health Organisation multicentre stroke registry were older than 64, and the proportion of older patients was even greater in Scandinavia and Ireland.[7] This obviously reflects the difference in age structure between Zimbabwe and developed countries. Nevertheless, by affecting younger people strokes in Zimbabwe may impose even greater economic burdens on families and society than in the West.

Although our numbers are relatively small, the pathological findings make an interesting comparison with the findings of studies in the developed world.[9,10] Most striking is the high proportion of cerebral haemorrhage in Zimbabwe and the relatively large number of non-stroke lesions presenting as a stroke. Seven of our 100 patients were found to have non-stroke cerebral lesions. This compares with 5% in Allen's hospital based series[11] and 1·5% in the Oxfordshire Community Stroke Project.[10] Sandercock *et al* empha-

sised that taking complete histories, including eyewitness accounts, will minimise misdiagnosis.[10] This is not always possible, particularly in dysphasic or comatose patients. Moreover, eyewitness accounts, if not obtained on admission, may not always be readily available to us because of difficulties in contacting rurally based relatives.

But for this study, the diagnosis of stroke would most probably not have been changed in all seven patients with non-stroke lesions; in only two cases did a careful review of the history, taken some days later, elicit atypical features. The diagnosis of non-stroke lesions remains a problem in developing countries, where computed tomography and carotid arteriography are costly and not always available. The diagnosis is important: five of our patients (four with subdural haematomas and one with cysticercosis) had potentially treatable lesions, and two recovered completely after surgery. This also implies that attempts to delineate clinical features of the group are necessary. A careful history of the event probably remains the most important part of assessment. In our sample a history of convulsions predicted non-stroke lesions. We have begun to investigate more extensively patients who have convulsions with "stroke."

The strokes in 62 of our patients were due to cerebral infarction. In contrast, infarcts were found in 84% of patients in the Harvard stroke registry and in 86% of Oxfordshire patients who underwent computed tomography.[9,10] Not only are infarcts less common in developing countries, but the causes may differ from those in developed countries. Cerebral embolism accounted for 12 strokes in our study, and in all cases the emboli were thought to have arisen from a cardiac source. Reports on strokes in developed countries show a higher incidence of embolism, almost all of the emboli arising from atheromatous extracranial carotid artery disease.[9] No carotid bruits or clinical evidence of atherosclerosis was detected in any of our patients. By the same clinical criteria 37% of patients in the Harvard registry had atherosclerosis. Pathological and clinical studies have shown a low incidence of atherosclerosis and coronary artery disease in black people in southern and central Africa,[12,13] and we believe that this largely accounts for the different pattern of cerebral infarction.

Neurosyphilis accounted for at least eight of the 62 cases of cerebral infarction in this study. This represents a large proportion of the patients with a remediable cause for stroke. The results and progress of this group are being analysed further and will be the subject of a further report. Nevertheless, it is apparent that the possibility of neurosyphilis should be investigated when no obvious cause of stroke is evident, particularly in developing countries.

Hypertension was the most important risk factor for thrombosis, being present in 20 of 50 patients with thrombotic strokes. Other risk factors such as smoking and diabetes were uncommon. No patient was taking oral contraceptives, but there were few women of childbearing age in the study population. In common with other African countries, however, smoking is increasing in Zimbabwe[14] and the recent expansion of child spacing services should substantially increase the number of oral contraceptive users.

The proportion of patients with intracerebral haemorrhage might seem high because of the relatively small number of patients with infarction. More probably, however it results from a high prevalence of untreated hypertension in the community. Twenty seven patients who suffered cerebral haemorrhage had hypertension, as did all patients who died before undergoing computed tomography. Overall, hypertension was the most important risk factor for stroke and was present in 49 of the 93 scanned patients with stroke lesions. Twenty two patients in this group had undiagnosed hypertension, and another 22 had defaulted from treatment. Thus the overall picture is of undetected or poorly controlled hypertension.

Epidemiological studies have shown a high prevalence of hypertension in black South Africans[15]; detection programmes are uncommon and rates of default high.[16] If this continues we would expect the incidence of haemorrhagic strokes, and accompanying mortality, to remain high. As we have indicated, risk factors for thrombosis may be increasing. Thus in the absence of good control of hypertension the incidence of and mortality from stroke may continue to rise in countries such as Zimbabwe, in contrast with declining morbidity and mortality from stroke in developed countries.[17,18]

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A patient blames her 35 year old daughter's mentally handicapped state on pertussis vaccine. When was the pertussis vaccine first introduced and routinely used in the United Kingdom and could this unlikely connection be ruled out if we show that the vaccine was not available in 1951?

Pertussis vaccines were evaluated in the United Kingdom by the Medical Research Council by field trials in 1942-4 and in a subsequent controlled trial in 1946-50 in which 3801 children plus controls received three doses of vaccine with no severe local or general reactions, no convulsions, and a 78% reduction of whooping cough.[1] Fourteen preparations of vaccine were then compared in trials on 28 799 children at six centres in 1948-54 with no severe local or general reactions: several children developed convulsions, eight of them within 72 hours after injection, but showed no "gross cerebral damage" in the two year follow up.[2] From the early 1950s local authorities were permitted to use the vaccine at their own discretion and it came into routine use as "triple vaccine" from 1957 in the United Kingdom.[3] From the dates alone the possibility that the patient's daughter might have received one of these vaccines cannot be excluded, though it is possible that any administration of vaccine might be traceable from trial records. Whether, if so, this had any connection with the mental handicap is even less likely to be identifiable.—N R GRIST, emeritus professor of infectious diseases, Glasgow.

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