



THE CENTRAL AFRICAN JOURNAL OF MEDICINE

Vol. 41, No. 10

CONTENTS

October, 1995

ORIGINAL ARTICLES

- | | | |
|---|---|-----|
| Acute myocardial infarction in Zimbabwe: the changing scene of coronary artery disease | JG Hakim, MG Odwee, S Siziya, I Ternouth, JA Matenga | 303 |
| Cholelithiasis in Dar es Salaam, Tanzania | MR Aziz, WBC Wandwi | 308 |
| Zinc, hydrochlorothiazide and sexual dysfunction ... | SM Khedun, T Naicker, B Maharaj | 312 |
| Neuropsychiatric HIV-1 infection study: in Kenya and Zaire cross-sectional phase I and II | MB Sebit | 315 |
| Malignant solid tumours in Nigerian children | KA Adelusola, WO Odesanmi, O Adejuyigbe, OA Rufai, MA Durosinmi, NO Akinola | 322 |
| The diagnosis and management of gall bladder in private patients in Harare. | CT Faranisi | 326 |

CASE REPORTS

- | | | |
|---|---------------------------|-----|
| A case of dual chloroquine and halofantrine treatment failure in Zimbabwe | S Mharakurwa | 327 |
| Kaposi's sarcoma in a two-week-old infant born to a mother with Kaposi's sarcoma/AIDS | KA McCarty, Z Bungu | 330 |
| A simple decannulating method for suprapubic trocar and cannula without a side slit | OA Fadiran | 331 |

LETTERS TO THE EDITOR

- | | | |
|---|-------------------|-----|
| The effects of sickle cell disease on the families of affected children | G A Oyediji | 333 |
|---|-------------------|-----|

NOTES AND NEWS

- | | | |
|---|---|-----|
| Important notice to all readers: Polio eradication initiative | Zimbabwe Expanded Programme on Immunization | 334 |
|---|---|-----|

ERRATUM

- | | | |
|--|--|-----|
| Impact of primary health care on child morbidity and mortality in rural Ghana: the Gomoa experience | Cent Afr J Med;1995;41,1995;41(5):148-53 | 335 |
|--|--|-----|

THE CENTRAL AFRICAN JOURNAL OF MEDICINE

ORIGINAL ARTICLES

UNIVERSITY OF ZIMBABWE

MEDICAL LIBRARY

PERIODICALS

Acute myocardial infarction in Zimbabwe: the changing scene of coronary artery disease

JG HAKIM, MG ODWEE, S SIZIYA,
I TERNOOUTH, J MATENGA

SUMMARY

From 1988 to 1993 (six years), 127 suspected cases of acute myocardial infarction (AMI) were admitted to the Parirenyatwa Hospital coronary care unit. AMI was confirmed in 76 cases, 37 were Black, 27 White, six Indian and six Coloured. For Blacks the male to female ratio was 5:1. The clinical and laboratory features and complications of AMI were similar in all ethnic groups. Compared to other groups, Blacks presented to hospital late, an observation which has important implications for thrombolytic therapy. With the increasing number of cases of AMI now being seen among Black Zimbabweans, the time has come for the evaluation of the changing risk factor profile and the initiation of education and intervention programmes which could contain this rise before it spirals into a major health problem.

INTRODUCTION

Several studies have alluded to the rarity of coronary artery disease (CAD) and its complications such as acute myocardial infarction (AMI) among Blacks in

Zimbabwe.¹⁻⁴ Most clinical descriptions of AMI have been in the form of single case reports.⁵⁻⁷ Contrary to the paucity of CAD among Blacks, AMI and complications of CAD have been reported in a much larger number of White Zimbabweans.^{4,8}

Parirenyatwa Hospital is the major referral centre in Zimbabwe. An analysis of cases of AMI admitted to this hospital between 1988 and 1993 is made with the aim of establishing clinical and laboratory characteristics of AMI among Blacks and other ethnic groups.

MATERIALS AND METHODS

Records of all patients admitted to the coronary care unit (CCU) between 1988 and 1993 with a provisional diagnosis of AMI were analyzed. The following information was abstracted; demographic data (age, gender, race), risk factors for CAD, clinical features, information on previous evidence of CAD (angina and old myocardial infarction), time from onset of symptoms to presentation at hospital, use of thrombolytic agents, complications, final diagnosis and outcome of hospitalization. Serial electrocardiograms (ECG) and cardiac enzymes were analyzed.

Definitions: AMI was diagnosed if two of the following three criteria were found:-

- (a) A typical history of chest pain – usually central, radiating to arm, jaw or neck and associated with sweating, nausea or vomiting.
- (b) A characteristic evolutionary change in serial ECGs consistent with AMI.
- (c) A typical pattern of cardiac enzyme rise.⁹

Hypertension was diagnosed if the patient: (a) was on antihypertensive treatment, or (b) had a persistent blood pressure of > 160/95 mmHg during hospitalization, or (c) showed fundal, ECG or echocardiographic evidence of hypertension.¹⁰

*Departments of Medicine and Community Medicine
University of Zimbabwe
PO Box A 178, Avondale
Harare, Zimbabwe
Correspondence to:
Dr JG Hakim*

Diabetes mellitus was diagnosed if the patient was on dietary, oral hypoglycaemic or insulin treatment or there was a persistently elevated random blood sugar > 11,1 mmol/l, or a fasting blood sugar of > 7,8 mmol/l.¹¹

Statistical analysis: The unpaired t-test was used to test for statistically significant differences in means, while the Chi-squared and Fisher's exact tests were used to test for statistically significant differences in proportions. A p value of < 0,05 was considered stati-

stically significant.

RESULTS

Between 1988 and 1993 a total of 127 patients were admitted to the CCU with a suspicion of AMI. Of these, 76 (59,8 pc) were confirmed to have AMI, comprising 37 Blacks, 27 Whites, six Indians and six Coloureds. The age distribution according to race and sex is shown in Table I. Whites were on average 15,7 (standard error

Table I: Risk factors in patients with acute myocardial infarction (AMI).

	Blacks			Whites			Others		
	men	women	all	men	women	all	men	women	all
AMI (pc)*	31 (63,3)	6 (54,5)	37	17 (58,6)	10 (62,5)	27	7 (53,8)	5 (55,6)	12
Age mean(SD)	51,2(12,6)	55,5 (10,7)	51,9 (12,3)	62,5(11,8)	76,3 (7,4)	67,6(12,3)	53,6(14,7)	66,6(14,5)	59(15,5)
Smoking	17	0	17 (50,0)	10	2	12 (52,5)	6	1	7 (63,6)
Hypertension	15	4	19 (52,8)	6	4	10 (41,7)	6	4	10 (83,3)
Diabetes	6	0	6 (16,7)	2	0	2 (8,3)	3	2	5 (45,5)

*Percentage of coronary care unit admissions confirmed to have AMI.

SD - Standard deviation.

Note: Information is not available on some factors. Percentage of available data is presented.

Table II: Clinical and laboratory features in patients with acute myocardial infarction.

Feature	Blacks	Whites	Others
	Total = 37 N (pc)	Total = 27 N (pc)	Total = 12 N (pc)
Chest pain	37 (100,0)	26 (96,3)	11 (91,7)
Left ventricular failure	*14 (38,9)	13 (48,1)	5 (41,7)
Arrhythmias at presentation	*15 (41,7)	9 (33,3)	7 (58,3)
Syncope	2 (5,4)	1 (3,7)	2 (16,7)
Elevated enzymes	!32 (91,4)	#18 (85,7)	!10 (100,0)
Abnormal ECG	37 (100,0)	*26 (100,0)	12 (100,0)
History of angina	*12 (33,3)	*7 (26,9)	4 (33,3)
History of myocardial infarction	*7 (19,4)	*5 (19,2)	5 (41,7)

N - total number of patients evaluated for a characteristic.

ECG - electrocardiogram.

* Information not available on one patient.

! Information not available on two patients.

Information not available on six patients.

Table III: Discharge diagnosis in patients not shown to have acute myocardial infarction following admission to the coronary care unit.

Diagnosis	Blacks	Whites	Others
Angina	0	5	3
Non-specific chest pain	3	2	4
Cardiac failure	4	2	0
Arrhythmias	3	2	0
Gastro-intestinal	3	1	1
Musculoskeletal pain	1	2	0
Pericarditis	3	0	0
Dissecting aneurysm	1	1	0
Respiratory	1	2	1
Cerebrovascular disease	1	1	0
Others	3	0	1

(SE) 3, 11 years older than Blacks ($t = 5,05$, $df = 62$, $p < 0,001$). The male to female ratio for Blacks was 5:1, but the difference in mean age between males and females was not statistically significant.

Risk factors: The distribution of risk factors of CAD according to race and sex is shown in Table I. The proportion of people who smoked was similar in all groups. None of the Black women smoked. Hypertension was present in a similar proportion of Blacks and Whites (52,8 pc vs 41,7 pc $X^2 = 0,70$; $df = 1$, $p = 0,403$). Serum cholesterol was estimated in only eight Black, one White and four Indian and Coloured patients. One

Table IV: Time from onset of symptoms to hospitalization in patients with acute myocardial infarction.

Hours	Blacks Total = 37 N (pc)	Whites Total = 27 N (pc)	Others Total = 12 N (pc)
< 6	7 (18,9)	10 (37,0)	6 (50,0)
6 - 24	8 (21,6)	8 (29,6)	2 (16,7)
> 24	17 (45,9)	4 (14,8)	4 (33,3)
Unknown	5 (13,5)	5 (18,5)	0

N - number of patients.

patient in each group had a level > 6,5 mmol/l. A positive family history of CAD was obtained in four out of seven Whites, but in none of the 16 Black patients for whom this data was available. Three White female patients had myxoedema.

Clinical and laboratory features: Chest pain was the commonest mode of presentation, found in more than 90 pc of patients in each group (Table II). The proportion of patients presenting with left ventricular failure and arrhythmias was similar in all groups. Elevated cardiac enzymes and abnormal ECGs were equally common in all the three groups. The discharge diagnoses for patients initially suspected of having AMI are shown in Table III. No Black patient was discharged with a diagnosis of angina.

Table V: Complications in patients with acute myocardial infarction.

Complication	Blacks Total = 37 N (pc)	Whites Total = 27 N (pc)	Others Total = 12 N (pc)
Arrhythmias	*14 (38,9)	8 (29,6)	5 (41,7)
Heart failure	*9 (25,0)	8 (29,6)	3 (25)
Pericarditis	1 (2,7)	0	0
Post myocardial infarction angina	10 (27,0)	4 (14,8)	4 (33,3)
Arterial embolism	3 (8,1)	1 (3,7)	1 (8,3)
Shock	7 (18,9)	7 (25,9)	3 (25,0)
Death	9 (24,3)	12 (44,4)	5 (41,7)

*Information was available on 36 patients.

N - number of patients with a complication.

Time to presentation: Table IV shows the time from onset of symptoms of AMI to hospitalization. A large proportion of Blacks (45,9 pc) presented at hospital later than 24 hours after the onset of symptoms. The association between time from onset of symptoms to hospitalization in patients with AMI and race (Black or White) was statistically significant ($X^2 = 6,96$, $df = 2$; $p = 0,031$).

Infarct localization: For Blacks, anterior AMI was found in 21 (56,8 pc), inferior in five (13,5 pc), combined anterior and inferior in six (16,2 pc) and non-Q infarction in five (13,5 pc) patients. ECG tracings were not available for two White patients. Of the rest, 12 (48 pc) had anterior, 11 (44 pc) inferior and two (8 pc) non-Q infarctions. In the Indian and Coloured group four (33, pc) had anterior, five (41,7 pc) inferior, one (8,3 pc) combined anterior and inferior and two (16,7 pc) non-Q infarctions.

Complications: Complications of AMI during hospitalization were similar in the three groups (Table V). The highest crude in-hospital mortality rates were observed in Whites (44,4 pc) and in the Indian and Coloured group (41,7 pc), but these did not differ statistically from the rate of 24,3 in Blacks ($X^2 = 2,82$; $df = 1$, $p = 0,093$ and Fisher's exact test, $p = 0,213$ respectively).

Arrhythmias during admission and hospitalization: Persistent tachycardia and premature ventricular contractions were each observed in eight Black patients. In Whites and the Indian and Coloured group sinus bradycardia was the commonest arrhythmia, seen in eight and five cases respectively.

Thrombolysis: None of the patients in this study received a thrombolytic agent.

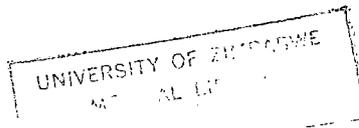
Duration of hospitalization: For Black patients discharged alive after AMI, 11 (39,3 pc) went in less than 10 days, seven (25 pc) between 11 and 14 days, nine (32,1 pc) between 15 and 21 days and one after a stay of over 21 days. For white patients eight (53,3 pc) were discharged in less than 10 days, two (13,3 pc) between 11 and 14 days and five (33,3 pc) between 15 and 21 days. For the Indian and Coloured group, five (71,4 pc) were discharged in less than 10 days and two (28,6 pc) between 15 and 21 days. Indians and Coloureds had a shorter duration of hospitalization (< 10 days), but this was not statistically different from the other races (Fisher's exact test, $p = 0,173$).

DISCUSSION

Several authors have observed or predicted a gradual increase in the prevalence of CAD and its sequelae among Blacks in Zimbabwe.^{4,12-14} Ross in an autopsy series of cases of sudden or unnatural death showed an increasing prevalence of CAD in Blacks, with 16 (1 pc) out of 1 604 autopsies demonstrating advanced coronary artery lesions.¹² However, there was no clinical, ECG or cardiac enzyme correlation, making it difficult to determine the importance of the coronary artery lesions seen. The 37 cases of AMI seen in Black Zimbabweans over six years in this study, is a small number, but it is a much larger clinical series than the earlier case reports.⁵⁻⁷ This might be an indication of a true increase in incidence or merely a reflection of better diagnosis and increasing awareness of AMI among Blacks. Whatever the case may be, this means more cases of AMI are now being seen.

Studies in several countries in sub-Saharan Africa attest to the low prevalence of CAD in Blacks. In a study in Uganda of 449 patients attending a cardiac clinic, only three cases of CAD were found.¹⁵ In a Malawian study of 114 cardiac patients no cases of CAD were seen.¹⁶ A study in the Ivory Coast comparing hospitalized African and European patients found the proportion of AMI to be 0,18 pc and 17,2 pc respectively.¹⁷ In six years only 54 Black patients were admitted to the Baragwanath Hospital with AMI.¹⁸ Many studies have speculated on the reasons for the rarity of CAD among Blacks in Africa,^{19,20} but it would appear that socio-economic, nutritional and metabolic factors play the most important role in determining the risk of developing CAD.^{13,21,22}

In this study, smoking is a common risk factor in all groups, but none of the Black women were smokers. Hypertension is also an important risk factor in all races, especially among the Indian and Coloured group. The small number of patients in this group is, however, inadequate to draw conclusions. Diabetes is an important risk factor in all groups, but conspicuously none of the Black or White women were diabetic. Again small numbers, do not allow meaningful interpretation of the latter finding. It is noteworthy that six Indians were diabetic, a finding which is in keeping with the observation of a high prevalence of diabetes and CAD among immigrant Indians.²³



Among Blacks admitted to the CCU and discharged with a diagnosis other than AMI, none had a diagnosis of angina, a further indication of the relative low frequency of CAD among Blacks. The clinical features of AMI are similar in all groups. The similarity of clinical, electrocardiographic and laboratory features of AMI in Blacks and other racial groups is well documented.^{18,24}

The late presentation to hospital by Black patients following the onset of the symptoms of AMI has important implications for the use of thrombolytic agents, as these are of the greatest benefit to patients who are treated soon after the onset of AMI.²⁵ Reasons for this late presentation to hospital include; lack of adequate diagnostic facilities and less awareness by health workers and the public of AMI in Blacks, and hence a delay in consultation, referral, diagnosis and admission to hospital. Blacks in general are of a lower socio-economic status than their White counterparts¹³ and they tend to live further from the centrally located referral hospital, hence transport constraints could have contributed to late arrival in hospital.

No patient in this study received thrombolytic agents, despite the fact that this was already established treatment for AMI.²⁵ Cost considerations must obviously feature highly in this omission, but we believe part of the problem was the lack of awareness of this and other newer additions to the treatment of AMI among health personnel, or alternatively its slow adaptation by doctors. The cost of thrombolytic agents, especially streptokinase is well within the financial means of many patients and health institutions. Fortunately it is now standard practice to use thrombolytic agents in the CCU.

The pattern of complications during hospitalization and the duration of admission in hospital did not show any marked differences between the various races. The trend toward a higher mortality observed in the White, Indian and Coloured groups was possibly because they were older than Blacks.

Limitations of this study: The limitations of retrospective studies with respect to the absence and reliability of data and the accuracy of information is well known, and we believe this is a major drawback of this study. The lack of information on occupation, income and diet, makes it difficult to compare the various ethnic groups in their proper socio-economic context.

The AMI cases analyzed represent only those referred to one government institution; cases admitted to private hospitals may have different characteristics. Indeed, a private hospital which was established in 1983 admits many cases of AMI from the well-to-do members of all races.

Conclusions: Most of the reports of CAD among Blacks in Zimbabwe were written more than two decades ago. Major developments have since occurred in the socio-economic status of Blacks with increasing life expectancy, adaption of Western diets and escalation of smoking. The low prevalence of CAD had hitherto made it difficult and costly to mount prospective, clinically oriented studies to evaluate its characteristics among the Black population. But, it is evident that AMI is now being seen more frequently and appropriate measures must be taken to recognize and address the problem. Various studies have provided evidence to show that socio-economic, nutritional and metabolic factors rather than racial or ethnic considerations are more important in determining the prevalence of CAD in a community.^{13,20,21} Studies should now address the changing risk factor profile predisposing to CAD among Zimbabwean Blacks and intervention programmes must be initiated to contain the rise before it spirals into a major health problem.¹⁴

The encouragement of healthy life styles through schools, primary health care facilities and media-based campaigns among Blacks is a cost-effective measure that has been advocated to avert an epidemic of CAD.²⁶

ACKNOWLEDGEMENTS

We would like to thank medical students Maggie Ambayi and Floyd Sekeramayi for assistance in data collection. They were supported by Grant No. RB/659/93 from the University of Zimbabwe Research Board.

REFERENCES

1. Baldachin BJ. Cardiovascular disease in the African in Matebeleland. *Cent Afr J Med* 1963;9: 463-9.
2. Gelfand M. Heart disease in the elderly African. *Br Heart J* 1961;23:387-92.
3. Shee JC. Myocardial infarction in Southern Rhodesia. *Br Heart J* 1963;25:25-9.
4. Levy LF. Atherosclerosis in Rhodesia. *Afr J Med Sci* 1971;2:229-38.

5. Gelfand M, Kaplan M. Bantu coronary insufficiency: report of a possible case. *Cent Afr J Med* 1958;4:157-9.
6. Davis JCA. Myocardial infarction in an African man. *Cent Afr J Med* 1964;10:173-6.
7. Buchanan WM. Coronary artery occlusion in an African male of 21 years. *Cent Afr J Med* 1968;14:80.
8. Forbes JJ, Newey W. Myocardial infarction in the African. *S Afr Med J* 1964;38:786-8.
9. Pasternak RC, Braunwald E, Sobel BE. Acute myocardial infarction. In: Braunwald E, editor. Heart disease: a textbook of cardiovascular medicine. Philadelphia: WB Saunders, 1988; 1222-1313.
10. WHO Expert Committee. Arterial hypertension. Geneva: World Health Organization, 1978.
11. World Health Organization. *Diabetes mellitus*. Geneva: WHO, 1985;9-12.
12. Ross MD. Death due to coronary atheroma in Rhodesian Africans. *Cent Afr J Med* 1969;15: 247-9.
13. Castle WM. Coronary heart disease risk factors in Black and White men in Zimbabwe and the effect of living standards. *S Afr Med J* 1982;61: 926-9.
14. Anonymous. The threat of coronary artery disease. *Cent Afr J Med* 1984;30:133.
15. D'Arbella PG, Kanyerezi RB, Tulloch SA. A study of heart disease in the Mulago Hospital, Kampala, Uganda. *Trans R Soc Trop Med* 1966; 60:782-90.
16. Brown KGE, Willis WH. Cardiac disease in Malawi. *S Afr Med J* 1975;49:926-30.
17. Bertrand E, Aye H, Beda B, Barabe P, Lebras M, Dellone M. Quelques aspects de coronarites observées à Abidjan, chez des noirs Africains. *Afr J Med* 1971;2:217-28.
18. Di Bisceglie AM, Miller MT, Blumsohn D. Myocardial infarction in an intensive care unit for Blacks. *S Afr Med J* 1982;61:902-4.
19. Wainwright J. Atheroma in the African (Bantu) in Natal. *Lancet* 1961;i:366-8.
20. Seftel HC. The rarity of coronary heart disease in South African Blacks. *S Afr Med J* 1978;54:99-105.
21. Seedat YK, Mayet FG, Latiff GH, Joubert G. Risk factors and coronary heart disease in Durban Blacks - the missing links. *S Afr Med J* 1992; 82:251-6.
22. Baker D. Poverty and ischaemic heart disease: the missing links. *Lancet* 1994;343:496.
23. Seedat YK, Mayet FG, Khan S, Somers SR, Joubert G. Risk factors for coronary heart disease in the Indians of Durban. *S Afr Med J* 1990; 78:447-54.
24. Falase AO, Cole TO, Osuntokun BO. Myocardial infarction in Nigerians. *Trop Geogr Med* 1973;25:147-50.
25. Anderson HV, Willerson JT. Current concepts: thrombolysis in acute myocardial infarction. *N Engl J Med* 1993;329:703-9.
26. Steyn K, Jooste PL, Bourne L, Fourie J, Badenhorst CJ, Bourne DE, et al. Risk factors for coronary heart disease in the Black population of the Cape Peninsula. The BRISK study. *S Afr Med J* 1991;79:480-5.



This work is licensed under a
Creative Commons
Attribution – NonCommercial - NoDerivs 3.0 License.

To view a copy of the license please see:
<http://creativecommons.org/licenses/by-nc-nd/3.0/>

This is a download from the BLDS Digital Library on OpenDocs
<http://opendocs.ids.ac.uk/opendocs/>