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CASE REPORTS

IS THE INCIDENCE OF ACUTE MYOCARDIAL INFARCTION IN NIGERIANS INCREASING?

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Abstract
Myocardial infarction is thought to be rare in Nigerians. This assumption may not be tenable any longer. We report 4 cases of acute myocardial infarction within one year out of which 3 were Nigerians. This seemingly increasing incidence may not be unconnected with the increase in the number of trained cardiologists and diagnostic tools, the rising incidence of the predisposing factors to myocardial infarction such as hypertension, diabetes mellitus, hyperlipidaemia etc. This report is meant to further increase our awareness of the slow but rising incidence of the disease. There is therefore the need for adequate preventive and control measures targeted at the predisposing factors.

Mots clés : Infarctus du myocarde, incidence, Nigérians

Résumé
L'infarctus du myocarde est considéré d'être rare parmi les Nigérians. Cette prétention parait n'être plus défendable. Nous rapportons 4 cas d'infarctus du myocarde aigu dans une période d'un an dont 3 étaient des Nigérians. Cette incidence apparemment croissante est tributaire à l'augmentation du nombre de cardiologues qualifiés et d'outils diagnostiques, l'augmentation des incidences des facteurs prédisposant à l'infarctus du myocarde tels que l'hypertension, diabète sucré, et hyperlipidémie. Ce rapport vise, de plus, à augmenter notre conscience de l'incidence lente mais montante de la maladie. Il y a donc le besoin de mesures préventives adéquates et de contrôles visés aux facteurs prédisposant.

Introduction

The incidence of myocardial infarction (MI) has remained a major public health problem in the industrialized world and is becoming an increasingly important problem in developing countries.[1] The World Heart Federation Projections indicate that the burden of disease in developing countries will become more closely aligned with that now afflicting developed countries.[2, 3] Previous studies across Nigeria from the sixties to late nineties showed MI to be relatively rare disease among Nigerians. [4 - 8] In Zaria, the first case of MI was reported by Adesanya et al *in* 1977.[5]In a ten-year review after that (1985-1995), Danbauchi reported 10 cases of ischaemic heart disease (IHD) out of which 7 cases had MI giving an average of one case or less per year.

With the decline in infectious disease-related deaths, the incidence of non-communicable diseases has been shown to be on the increase.[9] This has been attributed to accelerated economic development and lifestyle change promoting atherosclerosis in the last three decades. We report 4 cases of MI in one year alone out of which 3 cases were Nigerians who have been living in Nigeria.

Case 1

A 32 year old male Banker, a Nigerian presented in May 2003, with a 7 hours history of sudden onset of left-sided chest pain while driving to work. He described the discomfort as a heavy stone placed on his chest. There was no radiation to any other part of the body including the left arm, root of the neck, epigastric region etc. Patient vomited once after the onset of pain. He was not known to be hypertensive or diabetic but father is hypertensive and diabetic. He neither drank alcohol nor smoked. Physical examination revealed an anxious-looking young man

in pain with hand placed on the left precordial area and sweating. He was not in respiratory distress. The cardiovascular examination revealed a pulse rate of 100/min, regular and moderate volume. All other peripheral pulses were palpable and had similar character with no evidence of arterial wall thickening. The blood pressure (BP) was 144/116 mmHg supine and the jugular venous pressure was not raised. The apex beat was at the fifth left intercostal space, mid-clavicular line. The heart sounds were normal S1 and S2, no murmurs. The chest was clinically clear and no remarkable findings in the abdomen and central nervous system. A diagnosis of acute myocardial infarction was made.

The resting 12-lead ECG showed a sinus rhythm, ST segment elevation in V5 V6 with poor R-wave progression. Repeat ECG on day 3 showed Q waves in V2, T-wave inversion in chest leads, ST segment elevation in AVL and V2. The chest x-ray showed mild cardiomegally, CTR = 0.57. Lipid profiles showed elevated total cholesterol, 6.7 mmol/l (2.5-6.0) and LDL cholesterol 4.9 mmol/l (0.5-2.3). The fasting blood sugar, urea and electrolytes were normal.

Patient was given intravenous morphine 10 mg to relieve pain on admission and subsequently placed on atenolol 50 mg daily, lisinopril 2.5 mg daily, aspirin 75 mg daily, after an initial dose of 325mg and fluvastatin 20 mg daily. Patient improved with treatment and bed rest and later discharged for follow-up in cardiac clinic.

A 2D echocardiography after 6 months showed a thin and hypokinetic apical segment and interventricular septum, however, there was good global contractility. Left heart catheterization and coronary angiography at 6 months showed a normal coronary arterial system without significant angiographic lesions.

Case 2

A 70 year old Lecturer, Indian National who had lived in Nigeria for over 30 years presented in June 2003 with a 2-day history of sudden onset, central tightening chest pain associated with epigastric discomfort, excessive perspiration and paraesthesias in both upper limbs. There was no vomiting, breathlessness or body swelling. Patient was diagnosed hypertensive for 8 years with erratic medication. No history of diabetes mellitus. His father and two elder brothers had heart attacks. He never smoked nor drank alcohol. Physical examination revealed an elderly, anxious-looking man who was in pain. He was not dyspnoeic but was cyanosed and had ankle oedema. The pulse rate was 88/min regular and full volume, the BP was 100/70 mmHg, the JVP was not raised and the apex beat was located at the fifth left intercostal space lateral to midclavicular line. The heart sounds were soft S1 and S2; there were no murmurs. The Chest was clinically clear and the abdomen and the CNS examinations were essentially normal.

A diagnosis of acute myocardial infarction was made. ECG showed features of acute myocardial infarction involving the antero-septal region. A 2D echocardiography showed a grossly hypocontractile heart with mild left ventricular hypertrophy. Chest x-ray, U&E and Lipid profile could not be done before patient died on the second day of admission while trying to brush his teeth in the morning. Before he died, patient was on bed rest, tramal, isosorbide dinitrate 5 mg daily, ramipril 2.5 mg daily, atenolol 50 mg daily and aspirin 75 mg daily after an initial dose of 325mg. Streptokinase/Heparin injections were not available.

Case 3

A 47 year- old Technician presented in October, 2003 with a 2-day history of severe precordial pain which was sudden in onset, non-radiating and associated with diaphoresis vomiting and dizzy spells. Patient was diagnosed Type 2 diabetic 12 years earlier with erratic medication and clinic attendance. There was no history of hypertension, significant smoking or alcohol consumption. There was no family history of diabetes mellitus, hypertension or sudden death. Physical examination showed a middle-aged man with android obesity, dyspnoeic at rest, cyanosed and sweating profusely. The pulse rate was 120/min, regular, full volume with all other peripheral pulses palpable and synchronous. The BP was 120/90 mmHg in the supine position; the jugular venous pressure was not raised and the apex beat was difficult to locate because of thick anterior chest wall. The heart sounds were normal S1 and S2, and a gallop rhythm. Chest examination showed tachypnoea and few basal crepitations. Abdominal and CNS examination were unremarkable.

The diagnosis of AMI with left ventricular failure in a diabetic was made. ECG showed sinus rhythm, ST segment elevation in Leads 1, V2-V6, poor R-wave progression with QS pattern in V5 and V6 suggestive of antero-lateral infarct. A repeat ECG six days later showed flattening of the ST segment and Q-waves in AVL. Chest x-ray showed mild cardiomegally. Random blood sugar on admission was 14.7 mmol/l (3.0-8.3). Lipid profiles, cardiac enzymes could not be assessed because reagents were not immediately available. Patient showed remarkable improvement following commencement of treatment with frusemide 40 mg daily, atenolol 50 mg daily, captopril 6.25 mg daily, isosorbide dinitrate 5 mg twice daily, aspirin 325mg stat, followed by 75 mg daily and antidiabetic drugs. He remained stable but died suddenly on the 14th day of admission. Before he died patient was also on low molecular weight heparin (clexane) 40mg subcutaneously daily for prophylactic anticoagulation.

Case 4

A 74 year old retired Civil servant was seen in May, 2004 with a 6-day history of sudden onset of excruciating left precordial pain, sharp and radiating down the epigastric region. Pain was severe enough to prevent patient from lying down or sitting and lasted throughout the night. There was associated diaphoresis but no vomiting or symptoms suggestive

of cardiac failure. Patient had several large volumes of antacids because he was erroneously thought to have acute severe dyspepsia by the first general practitioner. Patient had been hypertensive for more than 20 years but not diabetic.

Physical examination showed an elderly man with android obesity, pulse rate was 80/min, regular, full volume; BP was 160/90 mmHg, and JVP was not raised, apex beat was at the 6th left intercostal space anterior axillary line, the heart sounds were normal S1 and S2; no murmurs. The chest, abdomen and CNS examinations were unremarkable. Diagnosis of acute myocardial infarction was made.

ECG showed sinus rhythm, Q-waves in leads II, aVF and V6, ST segment elevation in II, V4, V5, V6. T-wave inversion in V4, V5, V6. Chest x-ray showed gross cardiomegally with markedly unfolded aorta. Lipid profile showed raised triglycerides 4.0(1.3-3.7) mmol/l only. U&E, creatinine, random blood sugar were normal. Again, cardiac enzymes could not be done because of lack of reagents. Patient was treated with atenolol 50 mg, lisinopril 2.5 mg daily, Aspirin 75 mg daily after an initial dose of 325mg, and fluvastatin 20 mg daily while on absolute bed rest. Following much improvement patient was discharged for follow-up at the Cardiac clinic.

Table 1: Predisposing factors to myocardial infarction

Predisposing factors	Case 1	Case 2	Case 3	Case 4
Diabetes mellitus	-	-	+	-
Hypertension	+	+	-	+
Sedentary life style	+	+	+	+
Strong family history (hypertension/heart attack/diabetes mellitus)	+	+	-	-
Hyperlipidaemia	+	NA	NA	+
Atherosclerosis	-	-	-	-
Cigarette smoking	-	-	-	-
BMI (kg/m^2)	30	25	34	35

- =Negative; + =Positive; NA=Not available; BMI = Body mass index

Discussion

The incidence of MI in Nigerians has been reported to be very low, even though, Nigerians suffer diseases known to predispose to ischaemic heart disease such as hypertension and diabetes mellitus.[10,11] Coronary heart disease (CHD) is said to be still rare representing only 6% of all cardiovascular diseases in black Africans despite its increased incidence in recent years.[12] Also, a recent study in Ibadan,[8] revealed that though the incidence of MI is still relatively low, there has been a definite increase. The slight increase in the incidence has been attributed to urbanization, low level of physical activity and acquisition of unhealthy habits and diet of westernized population.

The first case of myocardial infarction in Zaria was reported by Adesanya et al[5] in 1977 in a 44 year old male accountant. This was followed by a ten year (1985-1995) review by Danbauchi who reported 10 cases of IHD with 7 presenting as MI[6]. Of this number, less than 4 cases were Nigerians. In this study, we also report that the incidence of MI in Nigerians is not static. When compared to previous reports from this centre[5, 6] it becomes obvious that the incidence of MI is on the increase with a total of four cases in one year, out of which three were Nigerians.

All the patients reported in this study belonged to the higher socio-economic group including the technician who could afford diet rich in animal protein and fat, with none engaging in hard manual labour. Predisposing factors such as hypertension, diabetes mellitus, sedentary lifestyle, obesity and hyperlipidaemia were identified in these patients in various combinations (Table 1). These findings are similar to those of Danbauchi[6] and Falase et al[8]and as seen in the Caucasians.[12]

The classic World Health Organizations (WHO) criteria for the diagnosis of acute myocardial infarction (AMI) require that at least two of the following three elements be present: I) a history of ischaemic-type chest discomfort, ii) evolutionary changes on serially obtained ECG tracings, and iii) a rise and fall in serum cardiac markers.[13] Our diagnosis of MI in this report was based on only the first two criteria as facilities for assessing cardiac markers were not available. Cardiac enzymes such as creatinine phosphokinase (CPK), lactic dehydrogenase (LDH) and serum glutamic oxaloacetic transaminase (SGOT), though not as specific as the troponins, could also not be measured because of lack of facilities. This is one of the limitations of this study. It is known that there is considerable variability in the pattern of presentation of AMI with respect to these three elements. While some patients do not present with classic chest pain, and therefore, the event would go unrecognized unless ECG was recorded fortuitously in temporal proximity to the infarction, others may have non-diagnostic ECG recordings. The diagnosis of AMI in Case 4 would have been missed if the ECG was not done. The patient was wrongly diagnosed as having acute dyspepsia and was being managed with antacids because he presented with overwhelming epigastric pain. A high index of suspicion of AMI in a patient presenting with chest pain, epigastric discomfort/pain, backed by the necessary

investigations will lead to improved diagnostic accuracy. It follows, therefore, that the presently reported incidence is probably more as a result of missed cases due to silent MI and sudden death before arrival at the emergency room for proper evaluation.

Coronary angiography and histopathological examinations are also useful in confirming the diagnosis of AMI, but unfortunately the facilities for angiography are not available in our center. Again, consent for post mortem examination is difficult to obtain in our area of practice because of religion.

The management of AMI can be quite challenging in terms of facilities and cost. Our patients for example could not receive the best due to limitations in facilities including some emergency drugs. Cardiac catheterization and coronary angiography which is crucial in the total evaluation of these patients for possible surgical intervention is not available locally. One of the patients reported in this series, had left heart catheterization and coronary angiography in a neighbouring West African country. Others could not afford it because of the high cost. Despite the increase in number of trained cardiologists in many centres in Nigeria, properly equipped and functional coronary care units are non-existent. This is understandable because of the previously reported low incidence of CHD and huge cost of setting up such units.

This report shows that the incidence of MI, though relatively low, is on the increase. A high index of suspicion is therefore required of every physician if the correct diagnosis must be made. Since we lack full functional coronary care units, due to high cost, attention should be directed at the prevention of the disease. This will involve the individual, the community, the government and non-governmental organizations' efforts at promoting lifestyle modifications which will in turn lead to a decrease in the incidence of the predisposing factors[9] of MI.

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