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Case Report

Acute myocardial infarction in a resource-poor setting: a case report in Awka, Nigeria

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ABSTRACT

The incidence of acute coronary syndrome (ACS) is increasing in Sub-Saharan Africa. Predisposing factors to ACS in Nigeria are not completely known. The management of ACS is influenced by its timely detection and availability of medical and reperfusion intervention facilities. We thus document a case of acute myocardial infarction (AMI) in 3 years that was successfully managed medically in Awka, Nigeria, albeit a debacle of resource-poor setting. The patient was a 42 year-old man who presented with retrosternal, stabbing chest pain of 4 hours duration. He has obesity, hypertension and a sedentary life style. He was in painful and respiratory distress; blood pressure was 140/80mmHg. Oxygen therapy, intravenous morphine 10mg was given. Oral Isosorbide dinitrate 20mg bd, oral Clopidogrel 300mg bd initially then 75mg daily, oral Aspirin 150mg bd, oral Simvastatin 20mg daily and oral Lisinopril 2.5mg were instituted. Resting electrocardiography showed evidence of ST elevations in the inferior leads. Cardiac enzyme markers were elevated. Following medical therapy, his condition improved. On the 3rd day he was discharged and subsequently followed up in the clinic. This case of AMI in a man who, has risk factors for AMI and, presented as the first case in three years in a Cardiology Unit in a tertiary hospital in Awka suggests that AMI is rare in this area. A resource-poor setting such as ours might still provide valuable medical therapy to ACS patients especially with prompt referrals from peripheral hospitals.

Keywords: Awka, Acute myocardial infarction, Nigeria, Resource-poor setting, Risk factors

INTRODUCTION

The incidence of acute coronary syndrome (ACS) is increasing in Sub-Saharan African countries including Nigeria.[1] Acute coronary syndrome is an ischemic heart disease marked by inadequate perfusion of heart muscles resulting from inadequate blood supply to, or increase in demand of oxygen and nutrients by, heart muscles. Ischemia to heart muscles may be regional or global.[2-4] Regional ischemia usually follows an area of heart muscles supplied by a given coronary vessel or its branches.[2-4] Acute myocardial infarction and unstable angina constitute ACS.[2-4] Factors that predispose to ACS are not completely known but include hypertension, diabetes mellitus, obesity, cigarette smoking, alcohol, sedentary life-style, high socio-economic status, among others.[2-4]

The management of ACS includes medical and reperfusion interventions.[4] There are variable outcomes in the management of ACS largely dictated by the severity of the injury, availability of facilities, time lapse between injury and intervention, and interventional expertise.[4]

We thus report a case of AMI occurring in a 42 year-old man with some of its predisposing factors, who presented

as the first case of AMI in three years in the Cardiology Unit of Chukwuemeka Odumegwu Ojukwu University Teaching Hospital, Awka, Nigeria, and was managed medically, without reperfusion surgery, with good outcome, albeit our resource-poor setting.

CASE REPORT

Patient is a 42 year-old man, a medical practitioner, who hails and resides in Orumba South LGA, Anambra State, Nigeria. He is a Christian of Roman Catholic denomination. He presented at the Accident and Emergency Unit of Chukwuemeka Odumegwu Ojukwu University Teaching Hospital, Awka, Nigeria, with chest pain and shortness of breath of 4 hours duration. He was apparently well until 4 hours prior to presentation when he developed sudden, progressive stabbing, retrosternal, continuous chest pain. Chest pain was exacerbated by meal; it radiated to his left shoulder and arm and was worse on exertion, and waned at rest. About the same time, he developed breathlessness, also worse on exertion. He has associated nausea and palpitation but no fever, cough, wheeze, orthopnea, leg swelling, sweatiness, or vomiting. There was no associated trauma. He has hypertensive and was compliant with his medications; he has no diabetes mellitus or peptic ulcer. There was no history of a similar chest pain in the past.

Patient's parents and siblings are all alive and well. There was family history of hypertension and obesity but no heart disease. Patient is married and has 4 children, all alive and well. He never used alcohol nor smoked cigarette. However, he has a sedentary life style.

Physical examination showed a middle-aged, obese man, conscious, in obvious respiratory distress, afebrile, not pale, anicteric, acyanosed, not dehydrated, and has no peripheral edema. His weight was 102kg, height 1.7m and body mass index 35.3kg/m2. Pulse was 120 beats/minute, moderate volume and regular. There was no peripheral arterial wall thickening. Dorsalis pedis and posterior tibia arterial pulses were present and adequate. Blood pressure was 140/80mmHg sitting. Jugular venous pressure was normal. Precordium was active. Apex beat was at the 5th left intercostal space in the mid-clavicular line. Heart sounds, S1 and S2 were heard. There was no murmur or any other adventitious heart sound. His respiratory rate was 32 cycles/minute. There were diffuse rhonchi but no crepitation. Abdominal and central nervous system examination was unremarkable.

A working clinical diagnosis of ACS on a background of obesity and hypertension, was made. He was given oxygen therapy and intravenous morphine 10mg on presentation, and placed on oral Isosorbide dinitrate 20mg bd, oral Clopidogrel 300mg bd initially then 75 daily, oral Aspirin 150mg bd initially then 75mg daily, oral Simvastatin 20mg daily, oral Lisinopril 2.5mg daily and oral Amlodipine 10mg daily. Intranasal oxygen was discontinued after some hours when patient's condition improved. The resting 12-lead ECG revealed a sinus rhythm, ST elevation in Leads II, III, AVF. Electrocariography repeated on the third day was normal. Chest X-ray showed normal CTR <0.5, and aorta was not unfolded. Fasting serum lipid profiles were within normal limits (total cholesterol 4.2mmol/l, high density lipoprotein cholesterol 0.7mmol/l, low density lipoprotein cholesterol 3.2mmol/l, triglyceride 0.8mmol/l). Fasting blood sugar, serum urea, electrolytes and creatinine were normal. Cardiac markers done about 24 hours of presentation were elevated: myoglobin <30ng/ml (reference <70ng/ml), CK-MB 7.36ng/ml (reference <5ng/ml), Troponin I 4ng/ml (reference <1ng/ml), Troponin T 1.40ng/ml (reference <0.03ng/ml), D-Dimer 0.212ng/ml (reference <0.5ng/ml). BNP was not done. Full blood count was normal (hemoglobin 14.2g/dl, white blood cells count 5900cells/ml, neutrophils 60%, lymphocytes 40%), platelet count $284x10^9$cells/ml. Erythrocytes sedimentation rate 101/1st hour. C-reactive protein was not done. Urinalysis showed normal findings. Echocardiography done on day 3 showed hypokinetic inferior wall motions.

He remained stable while on admission and was discharged on the 3rd day. On the 7th day on follow-up, he has no complaint. ECG done on the third day showed no Q-wave in the inferior leads. He was educated on life style modifications, continued on Simvastatin, Clopidogrel, Lisinopril and Aspirin, and advised on monthly clinic check-up.

DISCUSSION

Present index patient was the only case of AMI we documented in a space of three years in our Cardiology Unit. Variable prevalence of AMI have been reported in Africa: a Nigerian study documented 0.2%, a Senegalese 0.1%, and a Sudanese 10.4%.[5-7] However, the study populations were heterogeneous and not clearly defined. One of these studies evaluated AMI in an emergency department, and one in inpatients.[5,6] Present patient presented as an emergency case in the hospital. The occurrence of only one case in three years suggests that the prevalence of AMI is also low in Awka, Nigeria. Researchers have raised the possibility of inflammatory profiles or protective genetic factors to account for these low prevalence. The absence of high quality data relating to AMI in Sub-Saharan Africa, perhaps, makes this assertion speculative.[8,9]

We found that our index patient belonged to the high socioeconomic group, has obesity, hypertension, and a sedentary life style, all of which have been reported as predisposing factors to ACS in Nigeria.[4]

The diagnosis of AMI usually employs the classic World Health Organization criteria requiring that at least two of the following conditions be present: a) a history of ischemic-type chest discomfort, b) evolutionary changes in serially obtained ECG tracings, and c) a rise and fall in

serum cardiac markers.[10] We made a diagnosis of AMI in this report based on all the three criteria. However, we were not able to do tests for serial rise and subsequent fall in serum cardiac markers and serial ECG, making these a limitation of present report. The presentation of AMI may run an aggressive course leading to death before the patient may be seen in hospital. No doubt, many cases would be missed. Furthermore, autopsies are not routinely done here as a result of religious belief, making it all the more difficult for all cases of AMI to be identified and reported.

Facilities for surgical reperfusion interventions were not available in our center. Should our patient have required them, the outcome would have been unpleasant. We were limited by dearth of facilities given that we belong to the low income group countries, overburdened by diseases. Even when ACS is identified early, the patients might be constrained by poor resources to seek for treatments in areas where the facilities are available.

CONCLUSION

This case of AMI in a man who has its risk factors and presented as the first case in three years in a Cardiology Unit in a tertiary hospital in Awka suggests that AMI is rare in this area. A resource-poor setting such as ours might still provide valuable medical therapy to ACS patients especially with prompt referrals from peripheral hospitals to Cardiology Units.

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