West African Journal of Radiology
April 2009 Vol. 16 No. 1

Acute Myocardial Infarction Presenting in a Woman with Multiple Cardiovascular Risk Factors: Case Report

Dr. S. O. Ike, Dr. B. J. C. Onwubere, Dr. C. C. Okwara,
Department of Medicine, University of Nigeria Teaching Hospital,
Enugu, Nigeria.

Correspondence to: Dr. Samuel, O. Ike, Dept. of Medicine University of Nigeria Teaching Hospital, PMB 01129 Enugu (400001) Nigeria, Email: sobiajuluike@gmail.com

INTRODUCTION

Myocardial infarction occurs when cardiac myocytes die due to myocardial ischaemia[1]. Acute myocardial infarction (AMI) is part of the spectrum of acute coronary syndrome[1]. Its incidence is variable all over the world. In the United States, approximately 650,000 patients experience a new Acute Myocardial Infarction, and 450,000 experience a recurrent AMI, each year[2]. In United Kingdom its incidence is 300,000 new cases per year[3]. In Kenya, Eastern Africa, increase in the prevalence and risk factors has been reported[4]. In Nigeria, the actual prevalence is unknown. However, in a study done at Aminu Kano teaching Hospital, Kano, Nigeria, the reported prevalence was 0.4% of all medical admissions[5].

The incidence of case fatality following myocardial infarction appears to be on the decline in the USA and in developed countries[6]. This is attributable to introduction of effective intervention therapies and reduction of identifiable major risk factors. Acute myocardial infarction may not be as rare as has been thought in our environment, especially in the light of changing lifestyles to match western habits. It is likely that awareness of its existence is not recognized early enough and/or is still low in our environment. The authors think that a high level of suspicion should be developed by doctors and opportunities to create awareness for non-medical members of our community should be embarked on. This is the reason for this case report.

CASE SUMMARY

Mrs. CC, a 58 year old female trader, presented with chest pain and shortness of breath of 24 hours duration, through the Accident and Emergency unite of University of Nigeria Teaching Hospital, Enugu, on the 21st February, 2008. The chest pain was central and retrosternal, felt as heaviness, usually lasted more than 30 minutes and was not relieved by rest. She had associated palpitation, excessive sweat and vomiting. She had similar chest pain 3 days earlier which was relieved by rest and aspirin prescribed by a doctor at a peripheral hospital. She was not known to be hypertensive nor diabetic. She however had a strong family history of hypertension and diabetes mellitus. She did not take alcohol or tobacco.

Examination revealed a middle aged woman in painful distress, tachypnoeic (RR = 35 c/min.), and obese (BMI = 30.1kg/m^2). Her blood pressure was 170/100mmHg. She had I, II, & 4th heart sounds, but nor murmur. The rest of the examination was normal. Accompanying ECG showed evidence of inferior myocardial infarction (Q waves in lead II, III, Avf). The provisional diagnosis was recent myocardial infarction in a hypertensive patient.

She was admitted into the Intensive Care Unit (ICU) and commenced on oral Lisinopril 5mg daily, Atenolol 100mg daily, cap Tramadol 100mg b.i.d and subcutaneous Enoxaparin 40mg daily. Due to the incidental finding of hyperglycemia, soluble insulin was started. She made steady improvement from the 2nd to the 4th day of admission. However, by the 5th day she developed sudden onset of severe shortness of breath and cough productive of whitish frothy sputum; suggestive of acute pulmonary edema. There was elevation of the blood pressure. Serial ECG revealed left atrial abnormality and anteroseptal infarction on a background of recent inferior infarction.

The consciousness was impaired. This was successfully treated with i.v Frusemide, oral Lisinopril, i.m Morphine and dietary approach (DASH).

She made steady improvement over the next one week. However, by the 12th day she had a dramatic deterioration clinically, with progressive and rapid drop in BP, pulse and mental state, as monitored with Glasgow coma scale, with death following. Investigation results are as shown in table 1.

DISCUSSION

Chest *pain* is the most common presenting complaint in patients with ST segment elevation acute myocardial infarction. The pain is deep and visceral; adjectives commonly used to describe it are *heavy, squeezing,* and *crushing*[1]. Studies have shown that women and patients with diabetes mellitus are less likely to report chest pain or discomfort[7]. Breathlessness, vomiting and collapse or syncope are common features. Greater number of acute MI occurs in the early morning hours than any other time of the day. When patients with prolonged ischaemic discomfort at rest are first seen, the working clinical diagnosis is that they are suffering from an acute coronary syndrome[2]. The most common risk factors are smoking, hypertension, dyslipidemia, Diabetes Mellitus and obesity.

Traditionally, male gender was considered to be an important risk factor. During the past 15 years, however, females have overtaken males with respect to the annual number of cardiovascular deaths[8]. Studies have also shown that case fatality following Acute MI is higher among women[9 10 11]. Adjusted in-hospital mortality was similar among white women and black men compared with white men but was higher among black women and was unchanged overtime[12]. Female patients with myocardial infarction are typically older, have a higher prevalence of risk factors and a lower functional status than their male counter parts[12]. Women appear to be at higher risk than men when DM and dyslipidemia are present.

Diagnosis of acute MI was in the past based on typical chest pain, characteristic ECG changes and elevation of enzyme markers. CK MB is elevated within six hours of onset, peaks at 12 hours and returns to baseline within 48 72 hours. ECG changes typically are acute SET segment elevation occurring within minutes, progressive loss of R-wave and developing q waves within hours, appearance of Q waves and T wave inversion within days, and persistence of Q wave with near normal T wave in weeks to months. The recently introduced cardiac troponins are highly sensitive and specific markers for myocardial cell necrosis. This has led to the new definition for acute MI[13]. The complications of AMI commonly seen are (1). Sudden death, (which occurs in 50% within the first 2 hours and is usually due to ventricular fibrillation) (2) Heart failure (3). Recurrence and extension of the infarct and (4). Cardiogenic shock.

The presence of symptoms and older age in our index patient is in keeping with literature review findings[12]. Her presentation 24 hours after onset of symptoms reflects the prevalent unawareness of emergency nature of the cause of her symptoms in our environment. Also evident in her was the presence of multiple risk factors which were obesity, hypertension, diabetes mellitus, and dyslipidemia. Thus, the occurrence of acute myocardial infarction and multiplicity of complications were not unexpected in this patient. She displayed such complications as recurrence with extension, acute left ventricular failure, cardiogenic shock and death. The serum CK-MB was normal in our index patient. This is expected when the time of the sample collection is factored in. Investigation results of the patient could not be available promptly due to logistic problems associated with carrying out the tests.

CONCLUSION

The authors have brought this case to focus because we feel that AMI may not be as rare as presumed in our environment, especially in the light of changing life styles to match western habits. We suggest that a high index of suspicion for this entity should be developed by medical practitioners.

TABLE 1: RESULTS OF INVESTIGATIONS

1.	Serum total cholesterol was 230mg/d1 (140-200)
2.	Prothrombin time (27/02/2008) (test) = 9.45sec.; PT (control) = 11.1sec International normalized ratio was 0.8.
3.	Echocardiography report (22/2/2008): Dilated left ventricle. Other chamber sizes were normal. Normal valve leaflets. No stenosis or regurgitation. Normal wall thickness. Hypokinesia, especially of interventricular septum. Poor left ventricular contractility. Normal transmural diastolic flow. Normal pericardium. Conclusion: Dilated poorly contracting ventricles. Possibilities are: (1) Ischaemic heart disease. (2) Dilated cardiomyopathy.
4.	Cardiac enzymes: a. Troponin reported as positive b. CKMB isoenzyme 3.49 (0-24) c. LDH 220.0 (103-227).

Fig. 1 Patients' ECG tracing taken on the 21/02/08 showing sinus rhythm, with heart rate of 60/min, widened p-wave that is biphasic in V_1, and significant Q waves in lead II, III, aVF.

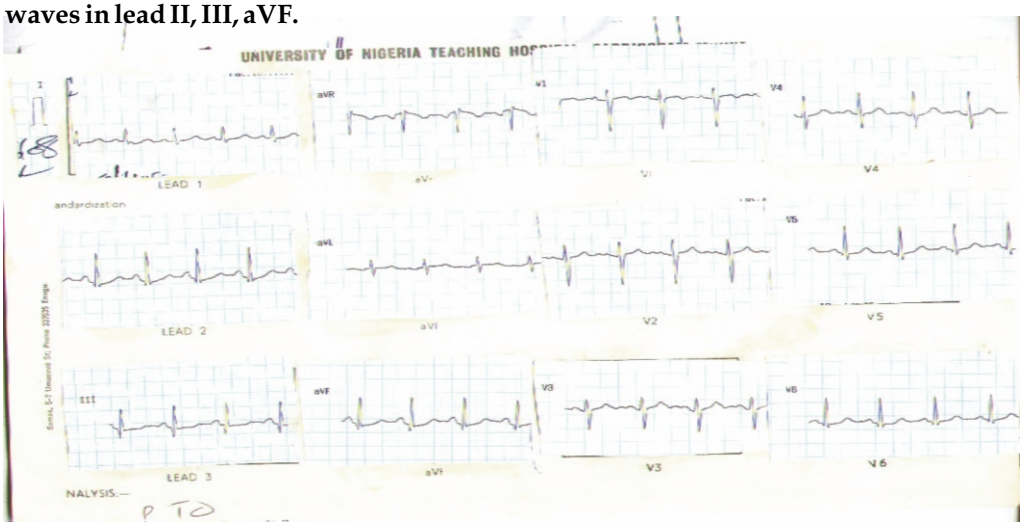
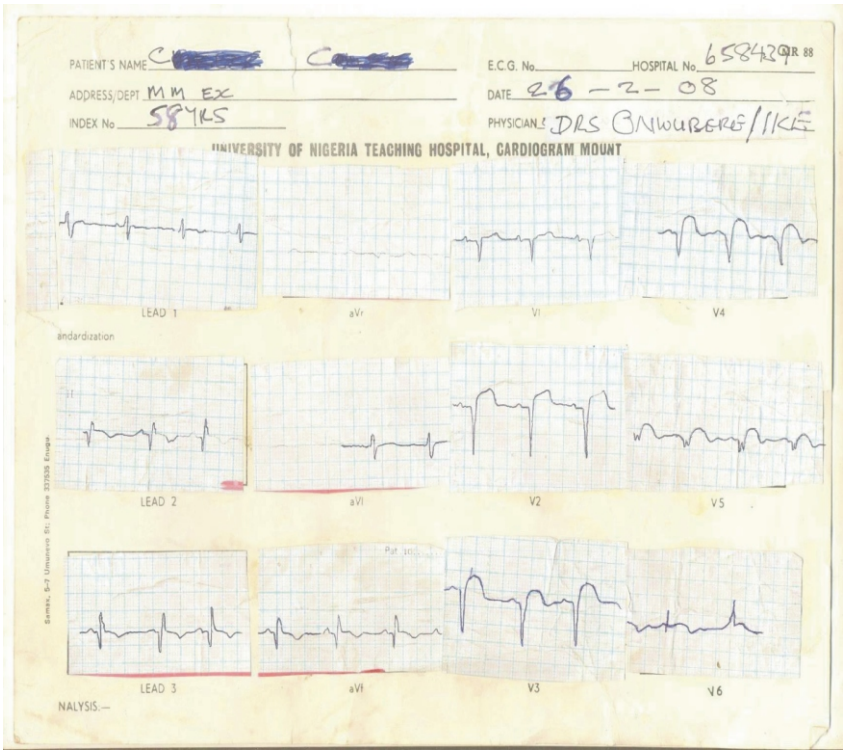


Fig. 2 Patients' ECG tracing taken on the 26/02/08 and showing sinus rhythm, heart rate of 83/min, QRS axis = O, biphasic p wave in v1, PR interval 0.16sec. Q waves in lead II, III, aVF, QT interval 0.32sec, T wave inversion lead II, III aVF, significant convex ST segment elevation in V_2-V_5

Conclusion:

a. left atrial abnormality
b. recent inferior myocardial infarction
c. acute anteroseptal myocardial infarction



REFERENCES

1. Kumar P, Clark M. (eds). Acute Coronary Syndrome. In: Clinical Medicine 6th edition. Edinburgh; Elsevier Saunders. 2005: 808-812.
2. Elliot MA, Eugene B. ST-segment elevation in myocardial infarction. In: Dennis LK, Eugene B, Anthony SF, Stephen LH, Dan LL, Jameson JL, (eds). Harrison's Principles of Internal Medicine. 16th edition. New York. McGraw-Hill; 2005. 1448-1453.
3. Kumar P, Clark M. (eds). Myocardial Infarction. In: Clinical Medicine. 5th edition. Edinburgh; Elsevier Saunders. 2002. 774-778.
4. Jablonski-Cohen MS, Kosgei RJ, Rerimoi AJ, Mamlin JJ. Prevalence of myocardial infarction in Kenya. East Afr J Med 2003; 53:80 (6); 293-297.
5. Sani MU. Pattern of medical admissions at the Aminu Kano Teaching Hospital. Niger J Med 2006; 15 (2): 128 131.
6. Klein W. Cardiovascular disease at the turn of the millennium: focus on Europe. Eur Heart J. 2001; Suppl 3: M2-M6.
7. Canto JG, Goldberg RJ, Hand MM, Bonow RO, Sokpo G, Pepine CJ, et al. Gender differences in myocardial infarction. Arch Intern Med. 2007; 167: (22) 2405-2413.
8. Anderson RD, Pepine CJ. Gender differences in the treatment for acute myocardial infarction: Bias or Biology? Circulation. 2007; 115:823-826.
9. Anyian JZ, Increased mortality among middle-aged women after myocardial Infarction. Ann Intern Med. 2001; 134:239-241.
10. Andreotti F, Marchese N. Women and coronary disease. Heart. 2008; 94:108-116.

11. Griffith D, Hamilton K, Norrie J, Isles C. Early and late mortality after myocardial infarction in men and women: prospective observational study. Heart. 2005; 91:305-307.
12. Vaccarino V, Rathore SS, Wenger NK, Frederick PD, Abramson JL, Barron HV, et al. Sex and racial differences in the management of acute myocardial infarction, 1994 through 2002. N Engl J Med. 2005; 353:671-682.
13. Antman EM, Tanasijevic MJ, Thompson B, Schactman M, McCabe CH, Cannon CP, et al. Cardiac-specific troponin 1 levels to predict the risk of mortality in patients with acute coronary syndromes. N Engl J Med. 1996; 335:1342-1349.