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M. S. JABLONSKI-COHEN, R. J. KOSGEI, A. J. RERIMOI and J. J. MAMLIN

ABSTRACT

Objective: **To review the literature on coronary heart disease (CHD) and its electrocardiogram (ECG) manifestations in Eastern Africa and provide medical education by increasing awareness and strengthening recognition skills of myocardial infarction (MI) through discussion of key features from representative ECGs selected from the Moi Teaching and Referral Hospital (MTRH) ECG service in Eldoret, Kenya.**
Data source: **Peer reviewed published articles found using a Medline search. ECGs were reproduced with one complex from each of the 12 standard leads, without patient's name or other identifying information.**
Conclusion: **CHD and its risk factors are increasing in prevalence in Eastern Africa over recent years. The ECG remains integral to the diagnosis of acute coronary syndromes, including MI. Representative ECGs from MTRH demonstrate the various features of the common anatomical distributions of MI, enabling medical education. Recognition of CHD and its ECG manifestations is one step on the path to decreasing resultant morbidity and mortality.**

INTRODUCTION

Although coronary heart disease is a well established leading cause of morbidity and mortality in the developed world, previous studies have shown that it has been uncommon in many parts of Africa(1,2). However, in several African countries, CHD and its ramifications are being increasingly recognised(3,4). This increase is likely secondary in part to the impact of urbanization, and with it the change of traditional diet and lifestyles evidenced most rapidly by more affluent segments of the population(4-6), as well as acquisition of diagnostic technologies and increasing numbers of tertiary health care centers(3). Undeniably, a majority of the population in Africa suffers from infectious diseases and malnutrition, but with the development of modern cities in African countries, a health population afflicted mainly by diseases of lifestyle and affluence, such as stroke and CHD, becomes increasingly prevalent(1).

Comparative to other cardiac diseases, there is a paucity of literature regarding coronary artery disease in Kenya and even less on ECG manifestations of the same. A Medline search conducted for this review produced only a few articles. More prominent causes of heart disease in black Africans are hypertension, rheumatic fever, congenital heart disease, and cardiomyopathy, with CHD responsible for a smaller but growing proportion(7,8), up to 6% of all cardiovascular diseases(9). The lack of data is likely due in part to limitations in technology and availability of diagnostic tests.

Much epidemiological research has been done worldwide in identification of coronary artery disease risk factors in order to prevent morbidity and mortality. The major risk factors include hypertension, dyslipidemia, and tobacco use, particularly cigarette smoking. Many experts also consider physical inactivity, obesity, and diabetes mellitus to be major risk factors. Additional associated risk factors include; male gender, heavy alcohol use, family history of premature coronary disease, left ventricular hypertrophy, and elevated levels of homocysteine or fibrinogen. There are investigations currently being undertaken to investigate the association of various other factors like hyperuricaemia with an increased risk of cardiovascular disease.

Recent literature from studies done at hospitals in Kenya, have shown that both risk factors for CHD and CHD itself are increasing(1). Yonga *et al* found a high prevalence of obesity, hypercholesterolaemia, cigarette smoking, and ECG evidence of left ventricular hypertrophy (LVH) in a hospital-based report of risk factors in Nairobi(1). An epidemiological survey of 145 admitted patients in October 2001 conducted at a referral hospital in Kenya found that hypertension was the most common discharge diagnosis assigned to

15.9% of patients (Cohen, 2003; personal communication). In this same study by Cohen, diabetes was diagnosed in 6.9% of patients. A risk factor thought to be pertinent to Africans specifically is haemoglobin S or C trait, especially in patients with CHD but normal angiography(9). Haemoglobin S trait is thought to have a role in the formation of microthrombi, but this as well as the role of haemoglobin C needs to be further investigated(9).

Coronary heart disease is typically manifested clinically by myocardial infarction, angina pectoris, congestive heart failure, and cardiac death. Atherosclerosis is linked to almost all causes of CHD, beginning with fatty streaks in adolescence and progressing to plaques in early adult life, finally resulting in coronary occlusions and cardiac events in middle and later adulthood. Myocardial infarction (MI) usually results from coronary artery occlusion from rupture of an atheromatous plaque. However, in some patients, MI results from coronary emboli, thrombotic disease, coronary vasculitis, vasospasm, ostial occlusion, congenital anomalies, or trauma. MI in indigenous Africans shows similar characteristics to patients less than 40 years of age in the west; infarction is frequently the first manifestation of disease, and normal coronary angiography is often found(7,9).

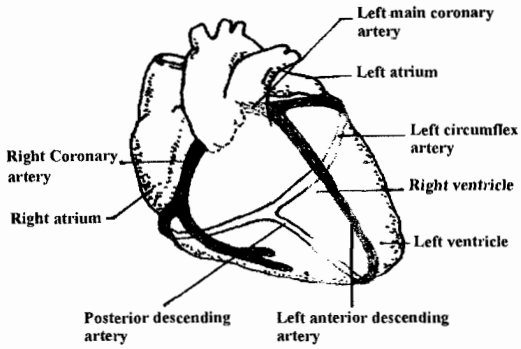
Myocardial infarction and ECG manifestations: Myocardial infarction often clinically presents as acute substernal chest pain lasting more than 30 minutes. Patients often describe the pain as being severe, crushing or squeezing; often there is radiation down the left arm, into the neck, teeth, or jaw. Associated symptoms of nausea, dyspnoea, diaphoresis, weakness, palpitations or a sense of impending doom may be present. In some cases, especially in elderly or diabetic patients, MI may occur without chest pain, also known as a "silent MI". In these patients symptoms may include mental status changes or gastrointestinal symptoms. Diagnosis of MI is based on history, physical examination, ECG findings, and a rise in cardiac enzymes.

The ECG remains integral to the diagnosis of acute coronary syndromes. Through reading the ECG, one is able to get an idea of the infarct's scope (transmural vs subendocardial), localization (anterior versus inferior), and timing (acute versus chronic). Persistent dynamic changes on serial EKGs consistent with ischaemia and infarction improve the diagnostic ability to determine the presence of MI.

The ECG of a normal heart usually has an isoelectric ST segment. With transmural infarcts, the ST segment is usually elevated. When ischaemia and infarct occur in the subendocardium, the overlying ECG leads usually demonstrate ST segment depression. The ECG is helpful in localizing the region of infarct in the heart, and subsequently which of the main coronary arteries was involved. Figure 1 illustrates the anatomy of the coronary arteries and the different areas of the heart that are supplied. It is important to note that in approximately 90% of people there is right dominance with the posterior descending artery (PDA) branching off the right coronary artery (RCA). In the remaining 10% of people, the PDA branches off the left circumflex artery (LCA). This obviously has implications for the resulting pattern of infarct seen on the ECG.

Figure 1

Anatomy of the major coronary arteries



In general, several statements can be made about the pattern of transmural infarction on ECG. With acute transmural anterior wall infarct one or more of the precordial leads (V1- V6) are affected. To break this down further, if leads V1- V3 are solely affected, an anteroseptal infarct has occurred supplied by the left anterior descending artery (LAD). Leads V4-V6 are representative of apical or anterolateral ischaemia supplied by the LCA; leads 1 and aVL, V5 and V6 are indicative of a lateral infarct, also supplied by the LCA. Both the LAD and the LCA branch of the left main coronary artery. Leads II, III, and aVF are affected with inferior infarct supplied by the PDA. Posterior wall infarctions, which are sometimes seen with inferior wall infarctions and are caused by lesions in either the RCA or the LCA, would show ST elevations in leads placed over the back of the heart (V7-V9). However, in the standard 12 lead ECG, posterior infarction is indirectly recognised by reciprocal ST depressions in leads V1-V3 and tall R waves in leads V1 and V2. Right-sided precordial leads (V4R, V5R) are demonstrative with ST elevations when there is right ventricular infarct supplied by the RCA. With the exception of right ventricular infarct, the other infarct patterns reviewed pertain to left ventricular infarcts.

The ECG is also helpful in determining the

approximate time of infarct. An acute transmural infarct is usually characterised by elevation of the ST segment, however the earliest change, which is not always seen, is the development of hyperacute or peaked T waves. Reciprocal changes are sometimes seen during the initial period, often seen as depressions of the ST segments in the leads opposite to the area of acute infarction. For example, reciprocal changes are seen in the inferior leads with anterior wall myocardial infarctions. Then there is ST segment elevation, with initial Q wave development and loss of R wave amplitude. As time passes, the infarct evolves on the ECG. Usually, the ST segment returns to the isoelectric baseline, R wave amplitude becomes reduced and the Q waves deepen. T waves often become inverted; this may resolve after days or weeks or persist for an indefinite period. These changes occur within the first hours to few weeks after the event.

An infarction of indeterminate age or that occurred remotely, is characterised by Q waves in the area of infarction that are typically deep (>1 mm) and broad (>0.04 seconds). There are often associated inverted or flattened T waves. In some cases, there may be no Q waves; in these cases there is usually loss of R wave amplitude or in the case of the precordial leads, poor R wave progression (R wave amplitude does not increase from leads V1-V6). In most cases, the ST segment is isoelectric, however, occasionally ST segments can remain elevated from the baseline. Possibilities to consider are aneurysmal formation or wall motion disorders (akinesis or dyskinesis). An old posterior wall infarction is characterised by tall R waves in V1-V2. However, other causes for tall R waves in these leads must first be excluded. The findings of Q waves in the posterior leads (V7- V9) can help confirm this infarct location.

One must be careful to distinguish ischaemia and infarction from other conditions that can produce Q waves, T wave changes, or ST segment elevations or depressions. Q waves can also be seen in patients with cardiomyopathies of non-ischaemic origin, ventricular hypertrophy, and ventricular conduction disorders like left bundle branch block. Some causes of T wave inversions include ventricular hypertrophy, cardiomyopathy, and cerebrovascular injury. Digitalis, ventricular hypertrophy, and hypokalemia can all cause ST depression. ST segment elevations can occur with acute pericarditis or myocarditis, or as a normal variant.

Discussion of ECGs at MTRH: In order to decrease morbidity and mortality from CHD, awareness, recognition and diagnosis are essential. Therefore, we present this review of ECG manifestations of myocardial infarction and ischaemia of select ECGs from the service at MTRH in Eldoret, Kenya.

Figure 2 shows a 12 lead ECG from a 52-year-old man illustrating an old anterolateral infarct in the distribution of the LCA. Note the Q waves in leads I, aVL, V4- V6 along with T wave inversions in this same distribution. Accordingly there is lack of R wave progression precordially.

Figure 2

Old anterolateral infarct in the distribution of the LCA. Note Q waves in leads I, avl, V4-V6 and T wave inversions. Poor R wave progression precordially

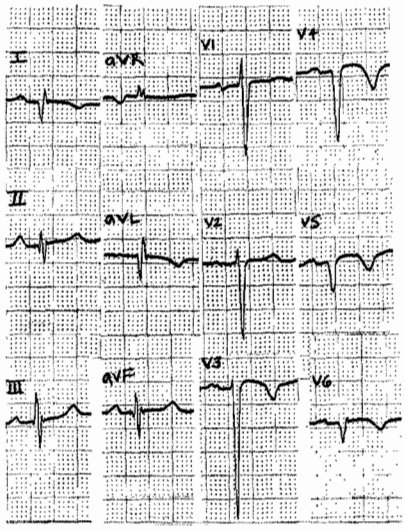


Figure 3

Acute inferior wall infarct. Note ST elevations in leads II, III and aVF and Q waves in leads III and aVF. Reciprocal changes are present anteriorly (ST depressions VI- V5 and aVL)

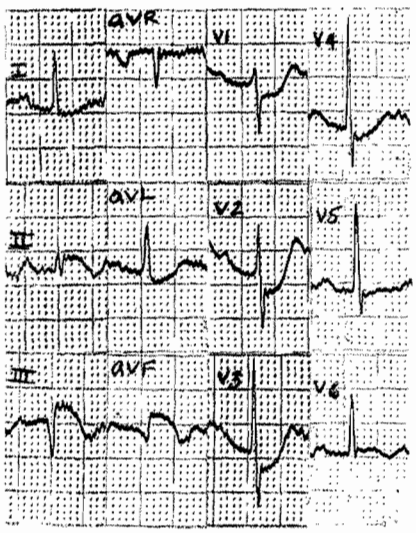


Figure 3 is an example of a 12 lead ECG from a 77-year-old woman with an acute inferior wall infarct resulting from occlusion of the PDA with right coronary dominance. The patient was admitted to the medical ward with complaints of diaphoresis and epigastric pain and found to be hypertensive with an elevated blood sugar level. Of note, one of the authors (M.J.) was leading rounds when this patient was presented; MI was not one of the differential diagnoses formulated by the team. The ECG was done immediately as the patient

was still symptomatic and also complaining of left leg numbness and dyspnoea. Note the ST elevations in leads II, III, and aVF and Q waves in leads III and aVF. Reciprocal changes are also present anteriorly as evidenced by the ST depressions in leads V1 - V5 and aVL. Also, there is a prolonged PR interval representing first degree atrioventricular block which can be seen with inferior infarctions as a result of increased vagal tone.

Figure 4 shows a 12 lead ECG from a 64-year-old man demonstrating a bifasicular block (right bundle branch block and left anterior hemiblock) and an old anterior infarction likely secondary to occlusion in the left main coronary artery. Note the Q waves in leads V1- V5. As the infarct has affected a large portion of the anterior wall, it is most likely responsible for the bifasicular block.

Figure 5 shows a 12 lead ECG from a 60-year-old male with an old anteroseptal infarct most likely secondary to occlusion in the LAD. Note the presence of Q waves in leads V1 - V3 and poor R wave progression.

Figure 4

Old anterior infarction with bifasicular block. Note Q waves in leads VI-V5

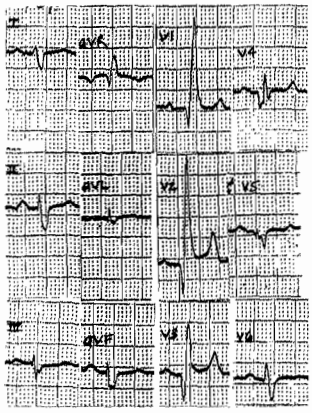


Figure 5

Old anteroseptal infarct. Note Q waves in leads VI-V3 and poor R wave progression

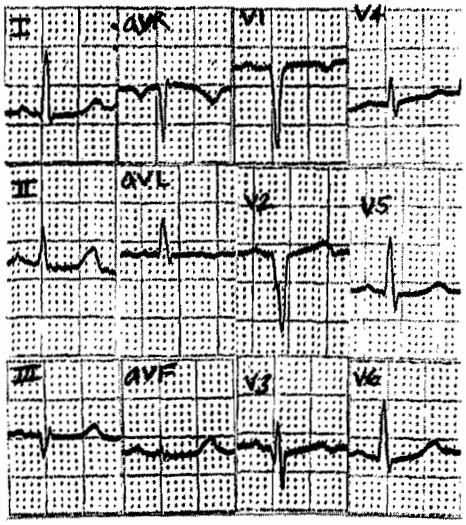


Figure 6 shows a 12 lead ECG that illustrates diffuse ST elevations. Infarct is unlikely, however, as the changes fail to follow the anatomical distribution of the coronary arteries and there are no reciprocal changes or Q waves. PR segment depression and tachycardia are also present further supporting a diagnosis of pericarditis.

Figure 6

Pericarditis. Note diffuse ST elevations PR segment depression and tachycardia

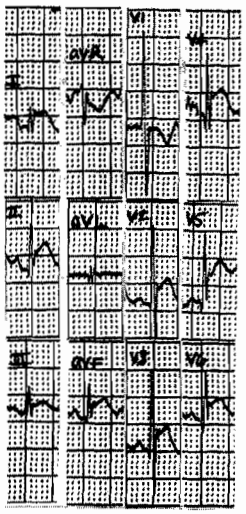
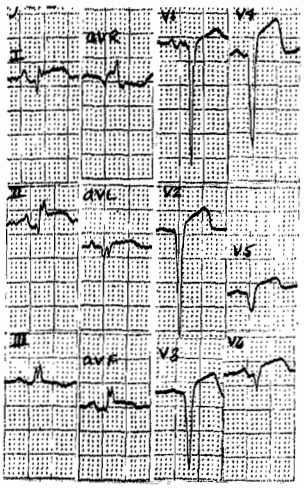


Figure 7 shows a 12 lead ECG from a 39-year-old male outpatient with a history of diabetes mellitus, hypertension, and complaint of chest pain. It demonstrates an acute massive infarct in the distribution of the left main coronary artery with left dominance. Note the ST elevations in leads I, aVL, II, aVF, V2 - V6 and Q waves in the same distribution. Though the changes are diffuse, this should not be confused with pericarditis, in which Q waves are not present.

Figure 7

Acute massive infract versus myocarditis. Note ST elevations in leads I, aVL, II aVF, V2-V6 and Q waves in the same distribution



Without serial ECGs, a diagnosis of myocarditis cannot be excluded. Myocarditis can simulate an infarct pattern on ECG in some patients with both regional ST elevations and Q waves. In several of the leads, notably I, II, and aVF, there appears to be some depression of the PR segment. Some possibilities include an atrial current of injury or Dressler's syndrome, an infrequently occurring post-myocardial injury pericarditis which may begin from a few days to six weeks after an MI.

Figure 8

Anterolateral subendocardial infarction. Note deep ST segment depressions in leads V4-V6 and mild ST depression in the inferior lead (II, II, aVF)

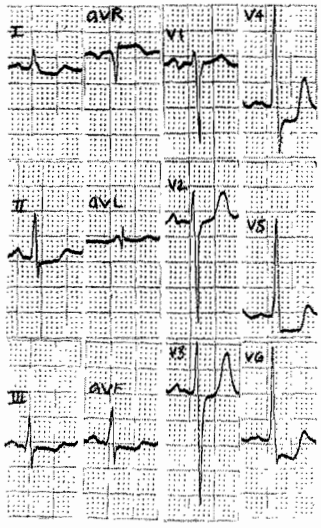


Figure 8 shows a 12 lead ECG from an adult woman which demonstrates changes consistent with an anterolateral subendocardial infarction with decreased blood supply from the LCA. Note the deep ST segment depressions in leads V4 - V6. There are some mild ST depressions in the inferior leads (II, III, aVF) which may be significant of ischaemia, perhaps with left coronary dominance, or may be non-specific.

CONCLUSIONS

Before Africa becomes part of the epidemic affecting the developed world, measures should be taken to prevent CHD from becoming a major health problem. Along with the obvious goals of public education campaigns and preventive medicine to enable primary and secondary risk factor reduction, accurate diagnosis is essential so that effective treatment can be delivered. With the increasing prevalence of CHD as discussed above, and as more diagnostic centers and testing become available, it is essential to have physicians and medical personnel well trained in the recognition of both the clinical and ECG manifestations of ischaemia and infarction. At MTRH, greater than 90% of myocardial infarctions were not suspected prior to diagnosis on ECG (Mamlin, 2003; personal communication). Clearly, there is a need for increased awareness of the acute coronary syndromes. Eventual goals for western Kenya and Africa, in general, include increasing the availability of therapeutic facilities such as telemetry and coronary care units (CCUs). Morbidity and mortality from acute MI has been significantly reduced by the introduction of CCUs (10). Attention must be given to this growing health problem so that detection of CHD occurs before MI or sudden death.

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