Incidence and Profile of Myocardial Infarction in Young Age Patients by Echocardiography

Thesis

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Introduction

Acute myocardial infarction is defined as death or necrosis of myocardial cells. It is a diagnosis at the end of spectrum of myocardial ischemia or acute coronary syndromes. Myocardial infarction occurs when myocardial ischemic exceeds a critical threshold.

Myocardial infarction is the leading cause of death in the United States as well as in most industrialized patients throughout the world. In general myocardial infarction can occur at any age but its incidence raises with age. The actual incidence is dependent upon predisposing risk factors for atherosclerosis. Approximately 10% of myocardial infarctions can occur in patients less than 45 years ***(Anderson et al., 2008).***

A number of studies in recent years have discussed the epidemiologic characteristic of acute myocardial infarction in a variety selected populations. However few reports have compared acute myocardial infarction in men and women, particularly less than 45 years of old with their older counterparts.

Acute myocardial infarction at a young age (<45 years) is characterized by low mortality rate, less extensive coronary artery disease (CAD), good residual left ventricular function and a favorable prognosis. Implication of family history of

myocardial infarction in young women is more frequent ***(Patel, 2007).***

Infarct size is a major determinant of prognosis patients with acute myocardial infarction. Early reperfusion (Within a few hours) and sustained patency of occluded artery by thrombolytic therapy limit the infarct size and thereby preserve left ventricular function and improve survival. Infarct sizes and left ventricular function are associated with extend and duration of myocardial ischemia ***(Karel Moons et al., 1999).***

Aim of the Study

Is to explore prevalence the clinical and echo characteristics in patients with acute MI aged below 45 years as compared to those aged 45 years or more.

Prevalence of Acute Myocardial Infarction

Acute myocardial infarction is rare in teenagers and young adults. The pathophysiology of their infarct is varied but not usually due to atherosclerotic plaque rupture except for those with genetically predetermined or familial hyperlipidemia ***(osula et al., 2002).***

Some investigators theorize that this increased incidence may be related to the circadian variation in cortical production affections the concentrations of various cytokines and other mediators of inflammation ***(Fantidis et al., 2002)***.

In India, cardiovascular disease (CVD) is the leading cause of death ***(Mukherjee, 1995).*** The deaths due to CVD in India were 32% of all deaths in 2007 and are expected to rise from 1.17 million in 1990 and 1.59 million in 2000 to 2.03 million in 2010 ***(Ghaffar et al., 2004)***

Although a relatively new epidemic in India, it has quickly become a major health issue with deaths due to CVD expected to double during 1985-2015 ***(Rastogi et al., 2004 and Gupta, 2007).***

Mortality estimates due to CVD vary widely by state, ranging from 10% in Meghalaya to 49% in Punjab (percentage of all deaths). Punjab (49%), Goa (42%), Tamil

Nadu (36%) and Andhra Pradesh (31%) have the highest CVD related mortality estimates ***(Gupta et al., 2006).***

Moderate physical exercise is associated with reduced incidence of CVD in India (those who exercise have less than half the risk of those who don't) ***(Rastogi et al., 2004).*** CVD also affects Indians at a younger age (in their 30s and 40s) than is typical in other countries.

The clincal trial data from studies of fibrinolysis show that the elderly (75 years old or older) continue to suffer a mortality rate four times that of the younger patients ***(Ahmed et al., 2006)***.

Pathology

MI is defined as myocardial cell death due to prolonged ischemia. Cell death is categorized pathologically as either coagulation or contraction band necrosis, or both which usually evolves through oncosis, but can result to a lesser degree from apotosis. Carful analysis of histological section by an experienced observer is essential to distinguish these entities .

After the onst of myocardial ischemia, cell death is not immediate but takes, a definite period to develop (as little as 15 minutes in some animal models, but even this may be overestimated. It takes 6 hours before myocardial necrosis is identified by standard macroscopic or microscopic postmortem examination complete necrosis of all myocardial cells at risk requires at least 4 hours to 6 hours or longer depending on the presence of collateral blood flow into the ischemic zone, persistent of intermittent coronary artery occlusion and sensitivity of the myocytes ***(Saraste et al., 1997)***.

Infarcts are usually classified by size: microscopic (foca) necrosis small (<10% or the left ventricle) as well by location (Anterior, lateral, inferior, posterior, septal or a combination of all). The pathologic identification of myocardial necrosis is made without references to morphological changes is epicardial

coronary arteries tree or to the clinical history ***(Bardales et al., 1996)***.

The term MI in a pathological extent should be preceded by the words acute, healing or healed. Infarcts are classified temporally according to the pathologic appearance as follows:

Acute (6 hours to 7 days), healing 7 to 28 days, and healed (29 days or more). An acute or evolving infarction is characterized by the presence of polymorph nuclear leukocytes if the interval between the onst of infarction and myocardial cell death is brief (e. g. 6 hours), minimal or no polymorph nuclear leukocyte maybe seen **(Saraste et al., 1997)**.

The presence of mononuclear cells and fibroblasts and the absence of polymorph nuclear leukocytes may be seen. healed infarction is manifested as scar tissue without cellular infiltration the entire process leading to a healed infarction usually requires five to six weeks or more.

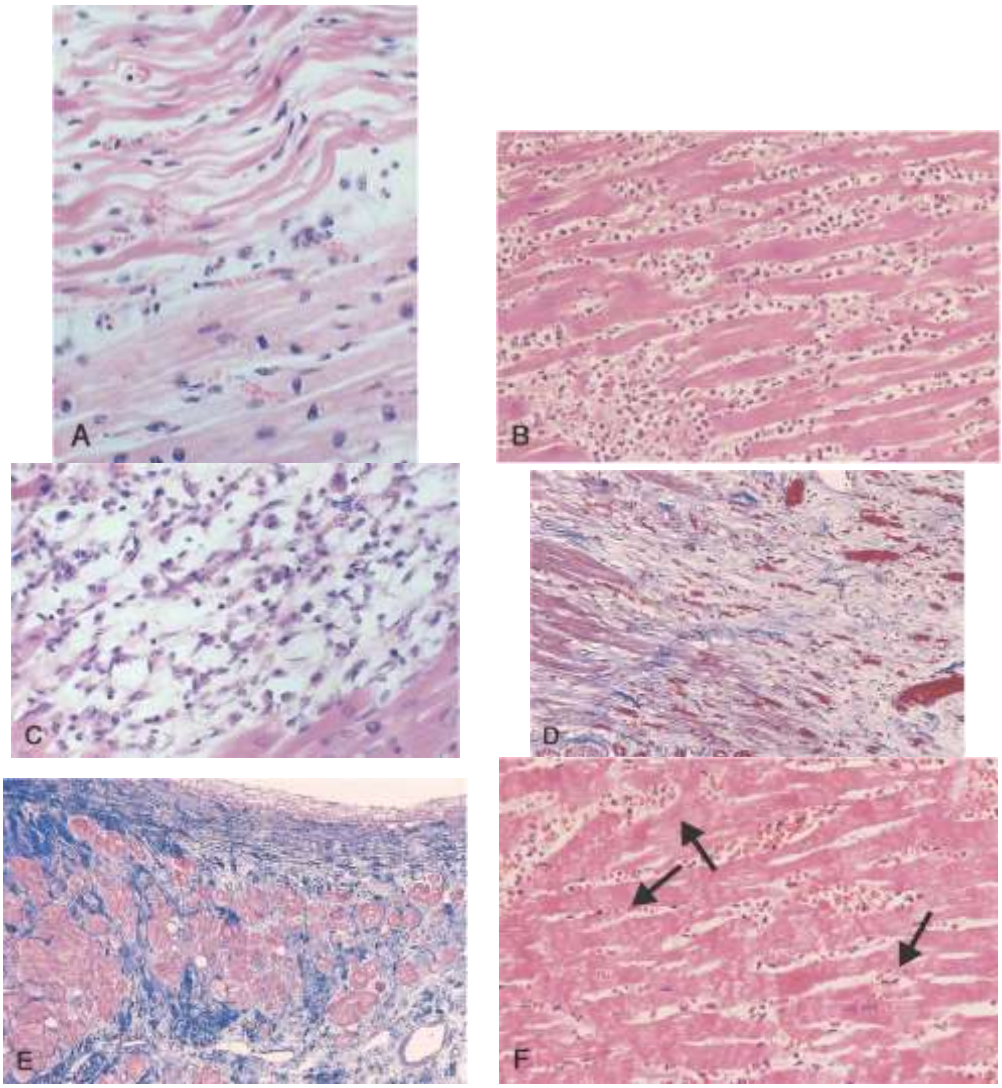

Figure (1): Microscopic features of Consequences of myocardial infarction ***(Kumar et al., 2005)***.

A One day old infarctshowing coagulative necrosis ,wavy fibres with elongation ,and narrowing ,compared with adjacentnormal fibres (lower right) widened spaces between the dead fibres conain edema fluid and scattered neutrophils

.B dense polymorphonuclear leukcytic infiltrate in an area of acute myocardial infarction of 3to 4 days duration

.C nearly complete removal of necrotic myocytes by phagocytosis (=7 to 10 days)

.D granulation tissue with arich vascular network and early collagen deposition ,approximately 3 weeks after infarction

.E well healed myocardial infarct with replacement of necrotic fibres by dense collagenous scar .Afew residddddual cardiac muscle cells are present .(in D and E collagen is highlighted as blue in this Masson trichrome stain)

.F myocardial necrosis with hemorrhage and contraction bands ,visible as dark bands spanning some myofibers (arrows).

The causes of myocardial infarction in young patients aged less than 45 years can be divided into four groups:

1- Atheromatous coronary heart disease.

2- Non atheromatous coronary heart disease.

3- Hypercoaguable states.

4- Myocardial infarction related to substance abuse ***(Egred et al., 2005)***.

The pathology of myocardial infarction in the presence of normal coronary arteries remains unclear but can be explained on the basis of coronary artery thrombosis, embolization spasm or compination of these processes ***(Manzar et al., 1997)***.

Proteinuria associated with nephritic syndrome results in the loss of low molecular weight proteins which in coagulation factors.

Thus, factors 1X, X1 and X11 are decreased due to urinary excretion increased synthesis of factors II, VII, VIII, X, XIII and fibrinogen as a compensatory mechanism resulting in increase in their blood levels ***(Angelini et al., 1999)***.

Antiphospholipid syndrome (Hughes syndrome) arterial and venous thrombosis is a prominent feature of this syndrome together with antiphospholipid antibodies, and miscariages of pregenancy. Antiphospholipid antibodies are associated with autoimmune disease such as systemic lupus erythematoses, but when they occur in isolation, this is known as primary antiphospholipid syndrome. The main antiphospholipid antibodies implicated in thrombosis and atherosclerosis are the anticardiolipin antibody, the lupus anticoagulant, and IgG antibodies against plasmophospholipid finding proteins such as b2-glycoprotein 1 and prothrombin ***(vaarala et al., 1996)***.

There is in vitro evidence that the anticardiolipin antibody increase platelet adhesiveness ***(Reverter et al., 1998)***.

It is possible that the antiphospholipid antibodies predisposes to premature atherosclerosis compounding the risk for infarction with this syndrome ***(Vaaral, 2000)***.