STUDY OF STRESS HYPERGLYCEMIA AS A PROGNOSTIC FACTOR IN ACUTE MYOCARDIAL INFARCTION PATIENTS

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BONAFIDE CERTIFICATE

This is to certify that this dissertation entitled ''STUDY OF STRESS HYPERGLYCEMIA AS A PROGNOSTIC FACTOR IN ACUTE MYOCARDIAL INFARCTION PATIENTS'' submitted by Dr. J.Vijaya kumar to the faculty of GeneralMedicine ,The Tamil Nadu Dr.M.G.R.Medical University ,Chennai in partial fulfillment of the requirement for the award of M.D.Degree Branch 1[General Medicine] is a bonafide research work carried out by him under my direct supervision and guidance.

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INTRODUCTION

An unusually high prevalence of glycosuria in non-diabetic patients who have acute myocardial infarction was noted as early as 1931. Stress hyperglycemia after MI is associated with an increased risk of in hospital mortality in patients with & without diabetes. More over a positive association between hyperglycemia at the time of the event and subsequent mortality from MI has been reported.

The factors of hyperglycemia in acute MI patients without previously noted abnormal glucose homeostasis are different. Transitory hyperglycemia and glycosuria are common during the acute stage of MI and usually explained as a manifestation of stress.

Acute MI has an extremely unfavourable effect on glucose metabolism. In such situations there is very often an increase in catecholamine concentration suppression of insulin release , development of peripheral insulin resistance and increase in growth hormone and cortisol concentrations.

A decreased secretion of insulin can be dangerous. During ischemia the passage of glucose into cells become critical, since the heart switches from aerobic metabolism with primary utilization of fatty acids to anaerobic metabolism that depends on glucose as energy source. Insulin facilitates glucose uptake, whereas ketones, FFA and their oxidation products that develop during insulin deficiency inhibit its transfer across the cell membrane. The resulting hormonal & physiological changes during acute MI is intolerance which in such cases is usual even in non-diabetics.

In the present report an attempt has been made to predict blood sugar response in acute myocardial infarction and correlate with selected parameters.

AIM OF THE STUDY

1. To find out the prevalence of hyperglycemia on acute myocardial infarction in non-diabetic population.

2. To identify the selected risk factors and correlate with blood sugar response.

3. To correlate blood sugar level within hospital outcome.

REVIEW OF LITERATURE

INTRODUCTION

Historically metabolic response to injury was first noticed by John Hunter in 1794 .The literature is rich in conditions where stress hyperglycemia has been observed. The etiology can be physical , mental, psychological or physiological but all these factors share one common determinant i.e., stress resulting in a sequence of events culminating as hyperglycemia.

ETIOLOGICAL FACTORS

Stress hyperglycemia has been observed in such trivial conditions like simple visit to a physician's office(white coat hypertension).The range of stressful condition varies from mild to severe stress like myocardial infarction cerebrovascular accident, physical trauma, crush syndrome, hemorrhagic hypovolemia and shock.

Stress hyperglycemia has been documented in cancer patients . Psychological stress also leads to insulin resistance, raising blood glucose level .Sustained hyperglycemia was observed in rats subjected to hyper G stress or swimming .Electroconvulsive therapy in psychiatric patients is a stressful event leading to hyperglucagonemia, suppression of insulin and a sustained rise in glucose concentration. Hyperglycemia is also observed during exercise in hot weather , emotional stress associated with solitary confinement and environmental novelty.

Glycemia was noticed with drug therapy and certain toxicological state. The anti-hypertensive drug sodium nitroprusside induced hypotension is a powerful stress producing factor leading to glucose intolerance.Overdosage or poisoning of verapamil , a calcium antagonist,causes peripheral insulin resistance leading to sustained elevation of blood glucose. Hyperglycemia has been observed in response to anaesthetic agents thiopental, propofol and diazoxide. It has also been seen in severe carbon monoxide poisoning due to stress of cerebral damage. Similarly L-dopa therapy , atropine injection , severe dehydration hypoxia and water intoxication are also associated with glucose intolerance.

The neurotoxin fraction of Egyptian cobra venom (Naja Naja) stimulates glucocorticoid release from adrenocortical cells. This lead to increased gluconeogenesis and inhibition of glucose utilization by the peripheral tissues, thus maintainig hyperglycemia during the period of stress.Stress hyperglycemia has been noticed in pediatric practice also. The pediatric emergencies are frequently associated with raised blood glucose level, where its magnitude is directly related to the severity of the illness. Stress hyperglycemia is seen in severely stressed premature infants on parenteral nutrition due to increased peripheral insulin resistance. Acute respiratory distress syndrome in children is one example associated with markedly raised blood glucose levels. Moreover

,hyperglycemia has been observed in 5.5% of all critically ill infants ,when they are infused with 10%dextrose solution.

Diarrhoea is frequently noticed in day-to-day practice. Interestingly hyperglycemia is a recognised complication of diarrhoea associated with hemolytic uremic syndrome and high mortality. The hyperglycemia in diarrhoeal states is a stress response to severe dehydration and hypovolemia.

PATHOPHYSIOLOGY

Carbohydrates serve as major fuel for the tissues of mammals except ruminant and universal fuels for fetus , metabolized as first line substrate for energy production. In health ,blood glucose level is maintained in a narrow range by a complex interplay of many counterbalancing processes. The factors which tend to raise the blood glucose level ,include diet, glycogenolysis and gluconeogenesis.

Liver and muscles are the organs where glycogen is brokendown. However, liver glycogen yields glucose in the blood , as the first line fuel, if the need arises e.g.,during fasting. Gluconeogenesis from noncarbohydrate sources like lactate , glycerol, alanine and propianate takes place in the liver and kidneys ,as these organs contains all enzymes involved in the gluconeogenic pathway.

Glucose is removed from the blood principally by five pathways :

(i) uptake and subsequent glcolysis of glucose by kreb's cycle in all cells of the body

(ii) stored in the liver and muscles as glycogen by glycogenesis,

(ii) excess is converted via glycerol-3-phosphate pathway to acylglycerol in the adipose tissues i.e.,lipogenesis,

(iii) remaining excess is converted into non essential amino acids,

(iv) loss in the urine ,which is normally an insignificant route of glucose removal in health.

During stress states catecholamines and a number of other hormones contribute to the development of hyperglycemia by directly stimulating glycogenolysis in the liver . In normal circumstances hyperglycemia stimulates the secretion of insulin and inhibits the secretion of glucagon . Insulin in turn tends to lower the blood glucose through its uptake by the liver and adipose tissue.

In stress , the key role of catecholamines is to interfere with insulin mediated glucose uptake by peripheral tissues and at the same time to inhibit the glucose –induced stimulation of insulin released by the pancreas.

The ability of catecholamines to interfere with each component of the glucoregulatory response i.e., stimulation of splanchnic and peripheral glucose uptake and suppression of hepatic glucose production, accounts for sustained hyperglycemia and makes catecholamines an important contributor to stress induced hyperglycemia. The diabetics ,when exposed to surgical stress ,demonstrate greater degree of hyperglycemia than normal people due to decreased metabolic clearance rate of glucose because of insulin deficiency and /or increased peripheral resistance .On the other hand the counter regulatory response to hypoglycemic stress in diabetics is abolished due to increased somatostatin: glucagon ratio in their pancreas with the result that there is no catecholamine –mediated raise in their blood glucose levels .Thus ,hypoglycemic stress can be life threatening in diabetic patients. Cytokines like interleukin -1 produced endogenously within the brain represent an important compenent of central regulation of metabolic response to stress as intra –cerebroventricular injection of this cytokine produces hyperglycemia and sustained elevation of glucagons and corticosterone.

<u>DIFFERENTIAL DIAGNOSIS</u>

Having learned about stress hyperglycemia and its cause along with its consequence ,it has to be distinguished from underlying true diabetic status by the following means .

a) ***by measuring islet antibodies ,insulin antibodies and stimulated insulin release especially in children***

b) ***by measuring fructosamine,***

c) ***by measuring glycosylated haemoglobin in blood***

CLINICAL FORMS OF STRESS HYPERGLYCEMIA

<u>CARDIOVASCULAR DISEASE AND CENTRAL NERVOUS SYSTEM</u>

Patients with cardiovascular disease commonly have a type of stress hyperglycemia during the acute phase of myocardial infarction or stroke or during any prolonged hypotensive response related to impaired cardiac performance or bleeding episode , altered carbohydrate metabolism is likely. At the onset of an acute myocardial infarction and acute stroke , there is an immediate increase of plasma catecholamines followed by increase of the other counter regulatory hormones , such as glucagon , cortisol and growth hormone . Impaired insulin secretion is prominent in these patients . In addition to the release of catecholamines from central sympathetic stimulation , there is some evidence that local damage to cardiovascular neurons may be a source of circulating catecholamines during infarction. This may explain the relationship between the degree of hyperglycemia and the eventual prognosis of acute cardiovascular and CNS events. Peripheral vascular occlusive disease

probably activates the system by stimulation of pain fibers , whereas cerebrovascular occlusive disease or cerebral hemorrhage may produce activation of central neurons by a local hypoxic stimulus. since adrenergic system is fully active and the metabolic effects are quite different from those seen in systemic hypoxia .

Since diabetics often have myocardial infarction and /or chronic cardiovascular disease and stroke, the pathophysiology of non insulin dependent diabetes and pathophysiology of neuroendocrine related sress hyperglcemia may infact not be different. Therefore hyperglycemia should not be ascribed to non insulin dependent diabetes during any acute cardiovascular event.

PAIN AND TRAUMA

Hyperglycemia and impaired glucose tolerance are common during any kind of traumatic injury. The mechanism of the stimulus to the autonomic nervous system and the activation of the stress response appears to be the pain associated with trauma. The major effect is to produce a hypercatabolic state characterized by high glucose levels, impaired insulin secretion, increased gluconeogenesis, increased muscle proteolysis and increased mobilization of fatty acids from adipose tissue . It would appear that reduction of catecholamines and increased insulin secretion are the two major goals to be achieved.

SURGERY AND ANAESTHESIA

Understanding the contribution of sympathetic activation to the metabolic response to surgical trauma is complicated by the effects of anaesthetic agents themselves on the sympathetic nervous system. The depth of anaesthesia is also an important factor .Decreased parasympathetic activity and activation of pituitary hormones promote hyperglycemia.

BURNS

Major burns are associated with a hypercatabolic state, that includes elevations of cortisol, growth hormone and glucagon, as well as impaired insulin secretion due to activation of sympathetic neurons and epinephrine release. Since this burn injury is often associated with sepsis,it is possible that leukocyte endogenous mediators may be playing a role. It is possible that the massive reabsorption of tissue and increased levels of plasma aminoacids are used as a source for gluconeogenesis and may be important to this continued heat production.

SEPSIS

Carbohydrate metabolism is often altered by sepsis. Usually carbohydrate intolerance and increased levels of counter-regulatory hormones like glucagon , cortisol and growth hormone have been reported. Mechanisms for the increased secretion of these hormones during uncomplicated infections are not certain, but it has been suggested that they may be related to the production of endotoxin like substances from the leukocytes. These

substances,which may be related or identical to the interleukins, may explain both the pyrogenic and metabolic responses to bacterial sepsis. These leukocytes has been shown to increase glucagon secretion to raise plasma glucose.

HYPOXIA

Sudden reduction of oxygen tension due to any cause is a potent stimulant of sympathetic nervous system activity. Hypertension is common due to sympathetic vasoconstriction .The explanation for this paradox appears to lie in two factors :(1)there may be simultaneous activation of the vagus and (2)severe hypoxia appears to impair the ability of beta adrenergic receptors to respond.

HYPOTHERMIA AND COLD STRESS

Cold stress and hypothermia are both associated with severe carbohydrate intolerance. In cold, stress metabolism increases as a result of activation of sympathetic nervous system and by shivering. In hypothermia, shivering is usually suppressed , thus reducing metabolic heat production and leading to lowered body temperature .Therefore the two physiologic states, although related , are quite different because of the vastly different responses to cold exposure. Therefore , during cold exposure with shivering there may be no increase of plasma glucose levels even though glucose turnover is markedly increased.

HYPOGLYCEMIA

Hypoglycemia elicits a sympatho adrenal response; a vagally mediated parasympathetic response that activates the hypothalamo –pituitary system.While hyperglucagonemia during hypoglycemia can be modulated by adrenergic and cholinergic blockade in experimental animals , this has been difficult to show in intact human beings . In man ,recovery from hypoglycemia is usually normal in the absence of either a catecholamine or a glucagon response, but if both are missing. Thus attempts to block glucagon release lead to little impairment of counter-regulation of insulin induced hypoglycemia and attempts to block the effects of autonomic nervous stimulation by atropine and /or adrenergic blockade also produces impairment of recovery mechanism from insulin induced hypoglycemia.

STRESS HYPERGLYCEMIA IN DIABETES MELLITUS

INSULIN DEPENDENT DIABETES MELLITUS

IDDM is chatacterised by an islet lesion leading to markedly impaired insulin secretion with eventual death and loss of almost all of the islet B cells. Elevated levels of catecholamines , cortisol , growth hormone and glucagon are characteristic of DKA. When the hyperglycemia becomes sufficient in magnitude to lead to glycosuria and elctrolyte loss , it is the subsequent volume depletion associated with glycosuria that activates the counter-regulatory hormones epinephrine , glucagon, growth hormone and cortisol to produce the

full blown diabetic ketoacidosis syndrome. The rationalisation was that catecholamine induced lipolysis could be prevented so that episodes of poor diabetes control would be associated with marked reduction of frequency of hospitalisation for ketoacidosis, in contrast to other forms of therapy directed at reducing the environmental stress factors in these patient's lives. A variety of stress states are associated with increased need for insulin in IDDM . It is also a commonly observed phenomenon that deterioration of diabetes control is likely during episodes of sepsis , burns , hypoxia , hypotension or vascular accidents in insulin treated diabetes patients.

NON- INSULIN DEPENDENT DIABETES MELLITUS

Islet B cells play a key role in any hyperglycemic state .Due to the feedback nature of islet regulation , it appears that increased hepatic glucose production and /or decreased peripheral glucose utilization alone or together cannot lead to sustained hyperglycemia unless the islet fails to adapt .On theoretical grounds one would expect that hyperglycemia in non- insulin dependent diabetes must be associated with an abnormality of islet function. Thus any stimulus that tends to impair insulin action or accelerate hepatic glucose production will be more effective in elevation of glucose in NIDDM patients because it would require a greater degree of hyperglycemia for islet cell adaptation to occur . It has now become apparent that islet B cells are more sensitive to the ability of epinephrine to impair islet responses to glucose, with their attendant effects on the sensitivity of islet B cells to non glucose stimulants.

ACUTE MYOCARDIAL INFARCTION

HISTORY

Although the clinical syndrome of angina was described in the 1770s, it was not until 1912 that James B. Herrick described acute MI . This delay is largely attributed to the prevailing view that a sudden coronary occlusion would be uniformly fatal and to the lack of understanding of the pathophysiology. Just before Herrick reported the MI syndrome, two Russian physicians, Obrastzow and Straschesko, published cases of acute coronary thrombosis and the differential diagnosis of acute MI versus angina in the German literature . In the landmark paper by Herrick, he wrote, "The clinical manifestations of coronary obstruction will evidently vary greatly depending on the size, location and number of vessels occluded. The symptoms and end-result must also be influenced by blood pressure, by the condition of the myocardium not immediately affected by the obstruction, and by the ability of the remaining vessels to properly carry on their work, as determined by their health or disease"

For nearly 70 years, however, there was a failure to meaningfully build on Herrick's visionary paper. There was considerable reluctance to accept the notion that coronary thrombosis was the proximate cause. There were a few

isolated exceptions. Levine published a book on coronary thrombosis in 1929 .Fletcher and associates administered intravenous streptokinase (SK) in 24 patients with acute MI in 1958 and Boucek, using an aortic root injection, nonselectively administered fibrinolysin (SK and human plasmin) to eight patients after acute MI in 1960 . Later, in 1976, Chazov and a Russian team administered direct, intracoronary fibrinolysin to two patients, and the paper was published in Russian . Rentrop, in 1979, reported the use of intracoronary SK in five patients with evolving MI , and this ushered in the reperfusion era that initially used intracoronary SK in selected centers in the early 1980s.

Nearly simultaneous to the development of a reperfusion therapy for MI was the other critical insight to its pathophysiologic underpinning. The classic angiographic study of DeWood and colleagues from Spokane , showing thrombotic occlusion in 87% of patients within 4 hours of symptom onset , provided the backbone for a revolutionary change to aggressive management of acute MI. This trial was powerful in convincing the medical community that coronary thrombus was indeed the proximate cause in nearly all acute MI patients.

Incidence and Significance

The true incidence of acute MI is unknown. Beyond the significant proportion of patients who die before reaching the hospital,

estimated at 200,000 to 300,000 patients in the United States per year , it is estimated that approximately 1 million patients present to a hospital each year with some type of MI as the principal diagnosis . Of these, we know that roughly 200,000 patients receive reperfusion therapy in the United States per year, and that this represents only 20% to 30% of the patients who are assessed for eligibility for aggressive management . The breakdown of infarctions is also unclear, with a split between the classic ST-segment elevation and the non–ST-segment elevation (also known as non–Q wave). The latter group is difficult to differentiate from unstable angina on presentation, but it is estimated that approximately one-half of patients present with either significant ST-segment elevation (greater than 1 mm) or fall into the latter category with abnormal ST-segment depression or T-wave inversion.

The incidence and mortality resulting from MI is on the decline. Not only has the fatality rate been reduced as better therapies have evolved , but the absolute number of MI events has continued to decrease since the 1970s. This is likely attributable to the many advances in preventive cardiology, including treatment of hypertension, avoidance of smoking, management of hypercholesterolemia, improved diet and exercise, and the use of prophylactic aspirin. It may also be an outgrowth of the high rates of surgical and percutaneous coronary revascularization in the United States, where the treatment of angina is more apt to be bypass surgery or angioplasty, rather than medical management.

Although prevention of MI is not a prominent effect of coronary revascularization, intervening early in the course of atherosclerotic coronary disease may change its natural history. The most pronounced effect on reducing the incidence of MI appears to have resulted from the use of HMG-CoA reductase inhibitors , which has led to the concept that acute MI may even be "extinct" someday . Clearly, the most important initiatives for the condition of MI are in its prevention, because once the process of plaque rupture is unleashed, it is much more difficult to interrupt.

Pathophysiology

Coronary atherosclerotic disease is the underlying substrate in nearly all patients with acute MI. The initiating event is a crack or fissure in the diseased arterial wall, which occurs as a result of loss of integrity of the plaque cap, which is the fibrous tissue overlying the plaque and partitioning the atheroma from the arterial lumen . The fissure or even frank plaque rupture leads to exposure of subendothelial matrix elements such as collagen, stimulating platelet activation, and thrombus formation. Furthermore, tissue factor is released with the arterial injury, which directly activates the extrinsic coagulation cascade and promotes the formation of fibrin . If an occlusive thrombus forms, patients may develop an acute ST-segment elevation MI unless the subtended myocardium is richly collateralized. On the other hand, the

thrombus formed may not be occlusive, but rather mural, and the patient may develop unstable angina or non–ST-segment elevation changes on the ECG (ST-depression or T-wave changes), which denote the lack of a "current of injury" or full-thickness (subendocardial to epicardial) myocardial ischemia .

Understanding the reasons why plaques crack may provide a better means of preventing acute MI, rather than intervening at the late phase after the event has been initiated. Plaques that rupture or fissure tend to have a thin fibrous cap, a high lipid content, few smooth muscle cells, and a high proportion of macrophages and monocytes . These mononuclear cells are conceived as a major trigger in plaque rupture by their release of such proteases as monocyte chemotactic protein (MCP-1) and matrix metalloproteinases (examples are collagenases, stromelysin, elastases), which chemically digest the plaque cap. Of note, the 3-hydroxy-3-methylglutaryl coenzyme A (HMG-CoA) reductase inhibitors have been shown to reduce the incidence of MI, and this is likely related to reduction in lipid content, as well as a favorable antiinflammatory effect on the cellular plaque constituents and chemokines . Loss of integrity of the arterial wall and platelet thrombus, with cessation of coronary blood flow through the infarct-related artery, thus drives myocardial ischemia and injury. As elegantly described by Reimer and Jennings, the wavefront of necrosis extends from the subendocardium to the subepicardium , and the extent of necrosis varies as a function of collateral

flow, the length of time that coronary blood flow has halted, and the extent of diminution of coronary blood flow. In many patients, there is a stuttering quality of MI with severe pain often denoting the cutoff of blood supply, and less chest pain, with partial, albeit insufficient, reflow. This dynamic quality of the infarct vessel blood flow pattern in acute MI (altered vasomotor tone or spasm) is likely related to the release of vasoactive amines from the activated platelets and loss of endothelial function.

The thrombus that occludes the coronary artery is a mixture of white (platelet-rich) and red (fibrin- and erythrocyte-rich) clot. In some patients, there is a more dominant role of platelets, whereas in others predominantly fibrin-rich thrombus at the arterial injury site is found. Stagnation thrombosis results from the lack of blood flow through the infarct vessel, thus leading to calumniation of red thrombus proximal to the original occlusion site . There is rarely frank herniation of the plaque occluding the lumen, which is known as plaque disaster .

On the other hand, the mural thrombus in patients without ST-segment elevation MI is more apt to be platelet rich and not accompanied by stagnation as there is no sustained cutoff of coronary blood flow. Depending on the extent and duration of ischemia, the patient may not experience any myocardial necrosis (unstable angina) or develop myocardial damage (non–ST-elevation or non–Q-wave infarction). Beyond what is occurring at the arterial

injury site and proximal to it, there is the potential for embolization of atheroma constituents or platelet thrombus distally. This is not typically found at postmortem but requires careful histologic inspection . When this occurs, a further explanation to cessation of myocardial blood flow is provided.

Although there is considerable variability in time of presentation, the plaque fissure events are more likely to happen in the early morning hours, especially on awakening, owing to the circadian rhythm, with its temporal cyclic-enhanced platelet aggregation, and reduced fibrinolytic potential related to higher plasminogen activator inhibitor levels . Surges of epinephrine may also have a role, as evidenced by a considerably higher rate of MIs during Scud missile attacks in the Gulf War and during the Los Angeles earthquake . Beyond catecholamine excess, there are several factors that ultimately may precipitate plaque fissure, including infection or inflammation of the diseased coronary artery segment.

PATHOPHYSIOLOGY OF OTHER ORGAN SYSTEMS

ENDOCRINE FUNCTION

PANCREAS

Hyperglycemia and impaired glucose tolerance are common in patients with AMI. Although the absolute levels of blood insulin are often in the normal range,

they are usually inappropriately low for the level of blood sugar, and there may be relative insulin resistance as well. Stress-induced hyperglycemia in the setting of AMI is associated with an increased risk of mortality even in patients without diabetes mellitus. Patients with cardiogenic shock often demonstrate marked hyperglycemia and depressed levels of circulating insulin, often with complete suppression of insulin secretion in response to tolbutamide. These abnormalities in insulin secretion and the resultant impaired glucose tolerance appear to be secondary to a reduction in pancreatic blood flow as a consequence of splanchnic vasoconstriction accompanying severe LV failure. In addition, increased activity of the sympathetic nervous system with augmented circulating catecholamines inhibits insulin secretion and augments glycogenolysis, also contributing to the elevation of blood sugar.Glucose appears to be a more favorable energy source than free fatty acids for the ischemic myocardium by more efficiently replenishing the Krebs cycle and stimulating contractility .Because hypoxic heart muscle derives a considerable portion of its energy from the metabolism of glucose and because insulin is essential for the uptake of glucose by the myocardium as well as for myocardial protein synthesis and inhibition of lysosomal activity, the deleterious effects of insulin deficiency are clear. These metabolic considerations, combined with epidemiological observations that diabetic patients have a markedly worse prognosis, have served as the foundation for efforts to more aggressively administer insulin-glucose infusions to diabetics with AMI.

ADRENAL MEDULLA

Excessive secretion of catecholamines produces many of the characteristic signs and symptoms of AMI. The plasma and urinary catecholamine levels are highest during the first 24 hours after the onset of chest pain, with the greatest rise in plasma catecholamine secretion occurring during the first hour after the onset of MI. These high levels of circulating catecholamines in patients with AMI correlate with the occurrence of serious arrhythmias and result in an increase in myocardial oxygen consumption, both directly and indirectly, as a consequence of catecholamineinduced elevation of circulating free fatty acids. As might be anticipated, the concentration of circulating catecholamines correlates with the extent of myocardial damage and incidence of cardiogenic shock, as well as both early and late mortality rates.

Circulating catecholamines enhance platelet aggregation; when this occurs in the coronary microcirculation, the release of the potent vasoconstrictor thromboxane A_2 may further impair cardiac perfusion. The marked increase in sympathetic activity associated with AMI serves as the foundation for beta-adrenoceptor blocker regimens in the acute phase.

ADRENAL CORTEX

Plasma and urinary 17-hydroxycorticosteroids and ketosteroids, as well as aldosterone, are also markedly elevated in patients with AMI. Their concentrations correlate directly with the peak level of serum creatine kinase, implying that the stress imposed by larger infarcts is associated with greater secretion of adrenal steroids. The magnitude of the elevation of cortisol correlates with infarct size and mortality. Glucocorticosteroids also contribute to the impairment of glucose tolerance.

RENAL FUNCTION ***Both prerenal azotemia and acute renal failure can complicate the marked reduction of cardiac output that occurs in cardiogenic shock. On the other hand, an increase in circulating atrial natriuretic peptide occurs after AMI, an increase that is correlated with the severity of left ventricular failure.***

DIAGNOSIS ***Acute MI is a clinical syndrome for which a constellation of subjective and objective parameters need to be assessed. The diagnosis must be obtained rapidly and accurately, and misdiagnosis can have catastrophic sequelae. The individual components of making the diagnosis are discussed separately, but it is the integration of all of them that facilitates the accuracy and speed of the clinical syndrome recognition.***

History

The classic symptoms of MI are intense, oppressive, excruciating chest pressure, with an impending sense of doom and radiation of the pain to the left arm. However, the other symptoms of chest heaviness or burning, radiation to the jaw, neck, shoulder, back, or both arms may be encountered. Indigestion is common, especially with inferior wall MI. Nausea and vomiting, particularly the former, are typical. Profuse diaphoresis is also a frequent characteristic. Taken together, the patient with a clear-cut presentation is experiencing a unique, discrete, painful event that has induced fear. However, the subtleties of the history are more common and challenging. It is important to ask whether there were premonitory signs of chest discomfort (not necessarily pain) in the preceding week or two. Pain or discomfort may be completely localized to the arm or shoulder. Quite commonly, only the symptoms of indigestion and nausea prevail, such that the patient attributes the episode to heartburn and resorts to taking antacids.

The identification of risk factors, such as smoking, known cholesterol elevation, diabetes, hypertension, and family history, is a supportive

piece that helps to put the acute history into context. The chest discomfort that causes the patient to seek medical attention is usually sustained (greater than 20 minutes), but can be stuttering.

Other accompanying symptoms include dyspnea, which is of concern because it may denote incipient congestive heart failure or, alternatively, is an outgrowth of the patient's anxiety. Palpitations or syncope are unusual, but a history of lightheadedness or dizziness and presyncope often reflects the underlying vagotonia or bradyarrhythmias. When syncope, or an out-of-hospital arrest has occurred, there is a high likelihood of ventricular tachycardia as an explanation.

The differential diagnosis is quite broad (because many conditions can masquerade as acute MI) including aortic dissection, pericarditis, esophagitis, myocarditis, pneumonia, cholecystitis, and pancreatitis. Of these conditions, it is always worth considering that the patient has aortic dissection until proven otherwise, so that this diagnosis is not missed. Although considerably less common than acute MI, the therapies for the two conditions are entirely different and, for example, the use of fibrinolytic therapy for aortic dissection could be disastrous.

MOLECULAR MARKERS OF MYOCARDIAL INFARCTION

MARKER	*MW (D)*	*RANGE OF TIMES TO INITIAL ELEVATION (hr)*	*MEAN TIME TO PEAK ELEVATIONS (NONTHROMBOLYSIS)*	*TIME TO RETURN TO NORMAL RANGE*	*MOST COMMON SAMPLING SCHEDULE*
hFABP	*14,000-15,000*	*1.5*	*5–10 hr*	*24 hr*	*Adm &then 4 hr later*
Myoglobin	*17,800*	*1–4*	*6–7 hr*	*24 hr*	*Frequent; 1–2 hr after CP*
cTnI	*23,500*	*3–12*	*24 hr*	*5–10 d*	*Once at least 12 hr after CP*
cTnT	*33,000*	*3–12*	*12 hr–2 d*	*5–14 d*	*Once at least 12 hr after CP*
MB-CK	*86,000*	*3–12*	*24 hr*	*48–72 hr*	*Every 12 hr ×3*
MM-CK tissue isoform	*86,000*	*1–6*	*12 hr*	*38 hr*	*60–90 min after CP*
MB-CK tissue isoform	*86,000*	*2–6*	*18 hr*	*Unknown*	*60–90 min after CP*
LD	*135,000*	*10*	*24–48 hr*	*10–14 d*	*Once at least 24 hr after CP*
MHC	*400,000*	*48*	*5–6 d*	*14 d*	*Once at least >2 d after CP*

hFABP = heart fatty acid binding proteins;; cTnI = cardiac troponin I; cTnT = cardiac troponin T; MB-CK = MB isoenzyme of creatinine kinase (CK); MM-CK = MM isoenzyme of CK; LD = lactate dehydrogenase; MHC = myosin heavy chain; CP = chest pain

ANGIOGRAPHY

On occasion, even with all of the tools outlined, the diagnosis is uncertain. This may be the result of atypical symptoms and an ECG that is difficult to interpret. In a patient in whom reperfusion therapy is contemplated, an approach that can rapidly establish the diagnosis is emergency coronary angiography. By demonstrating an acutely occluded infarct vessel, with the characteristic appearance of thrombus or a cutoff sign at the point of occlusion, coupled with left ventriculography to ascertain the segmental wall motion profile, angiography can at times be helpful for a difficult diagnosis. Examples of patients who may present with ambiguity are those with an acute myocarditis with diffuse ECG changes, or patients with prior bypass surgery and previous MI, or those without a characteristic pattern on ECG. Furthermore, angiography can serve as the foundation for primary balloon angioplasty to achieve reperfusion.

Treatment

Analgesia and Supportive Measures

The first step in treating the patient, while more definitive therapy is being prepared (such as fibrinolytics or transfer to a cardiac catheterization laboratory), is to make the patient comfortable via supplemental oxygen (usually

nasal cannula at 2 L per minute) and morphine (2 to 4 mg intravenously and repeat as necessary). Before using morphine, it is helpful to have quickly tried sublingual nitroglycerin to determine whether there is a reversible component of the ischemia, pain, and ECG changes. Furthermore, if the patient is Killip class I or II and has no bradycardia or hypotension (systolic pressure less than 110 mm Hg), then use of intravenous beta-blockade can be considered as a means of reducing the extent of ischemia and lessening the need for narcotic analgesia.

Aspirin

The use of aspirin is a cornerstone of therapy for patients with acute coronary syndromes. It should be initiated as quickly as possible when the diagnosis is made, at a dose of 160 mg by chewable administration (2 80 mg "baby" aspirin) or a 325-mg orally administered tablet. No enteric coated formulation of aspirin should be used. The validation for the importance of aspirin in this setting is derived from the landmark International Studies of Infarct Survival (ISIS-2) trial , which showed it is lifesaving . Since this trial, aspirin has been used in all patients with acute MI, and other trials have provided strong evidence for its use in unstable angina and non–ST-elevation MI . The dose that has been routinely recommended is 325 mg per day, and it is continued indefinitely.

Clopidogrel

The Clopidogrel in Unstable Angina to Prevent Recurrent Events trial assessed the combination of aspirin plus clopidogrel compared with aspirin in patients without ST-segment elevation MI. With a 20% reduction of ischemic events, particularly MI (or reinfarction), there are supportive data to use this second antiplatelet .

Beta-Blockade

Nearly all of the data available for the use of beta-blockers in acute MI are derived from the prereperfusion era. There is extensive support for the usefulness of beta-blockade to reduce recurrent ischemia and arrhythmias and improve survival . The largest trial of intravenous beta-blockade, ISIS-1, suggested that the predominant benefit was mediated via the reduction of cardiac rupture events . Only two placebo-controlled trials in the contemporary era of reperfusion therapy have been conducted, and one of these showed reduction in recurrent ischemia with the use of early intravenous followed by oral metoprolol . The trial was relatively small, however, and the primary end point of preserving cardiac function was not significantly affected. Likewise, a trial of intravenous metoprolol conducted by Van de Werf et al. was negative . In

the GUSTO-I trial, the use of intravenous beta-blockade was associated with an adverse profile of clinical outcomes including worse mortality and congestive heart failure . In this trial, the use of intravenous beta-blockade in 44% of patients was higher than many other large-scale thrombolytic studies, and it remains possible that the ill effects were related to administering therapy to patients with a large infarction or with marginal left ventricular function . Current recommendations are to use beta-blockers orally in patients with a preserved left ventricular ejection fraction (estimated at >40%), and to initiate this on the first or second day after presentation. Intravenous beta-blockade is particularly useful in patients who are hyperdynamic and hyperadrenergic, representing the unusual subset of relatively young patients who have tachycardia and hypertension disproportionate to the smallness of their MI territory. But the potential for reducing intracerebral hemorrhage, as demonstrated in the TIMI-2 trial, has not been confirmed in subsequent large-scale efforts .

<u>Angiotensin-Converting Enzyme Inhibitors</u>

One of the most important advances in cardiovascular medicine in the last decade has been the demonstration of survival benefit for angiotensin-converting enzyme (ACE) inhibitors in patients with left ventricular dysfunction. In several trials of acute MI various ACE inhibitors have been tested and shown, in aggregate, to promote survival, reduce the incidence of

heart failure, and, in selected trials, reduce the incidence of reinfarction and the need for revascularization. ACE inhibitors can be recommended in the first 24 hours of an anterior MI (proximal or mid-LAD) or an MI complicated by either heart failure or ejection fraction less than 40%, or both. A graded dose schedule is used to avoid hypotension, especially with the first dose, with initiation of therapy on the first hospital day once the blood pressure is stabilized.

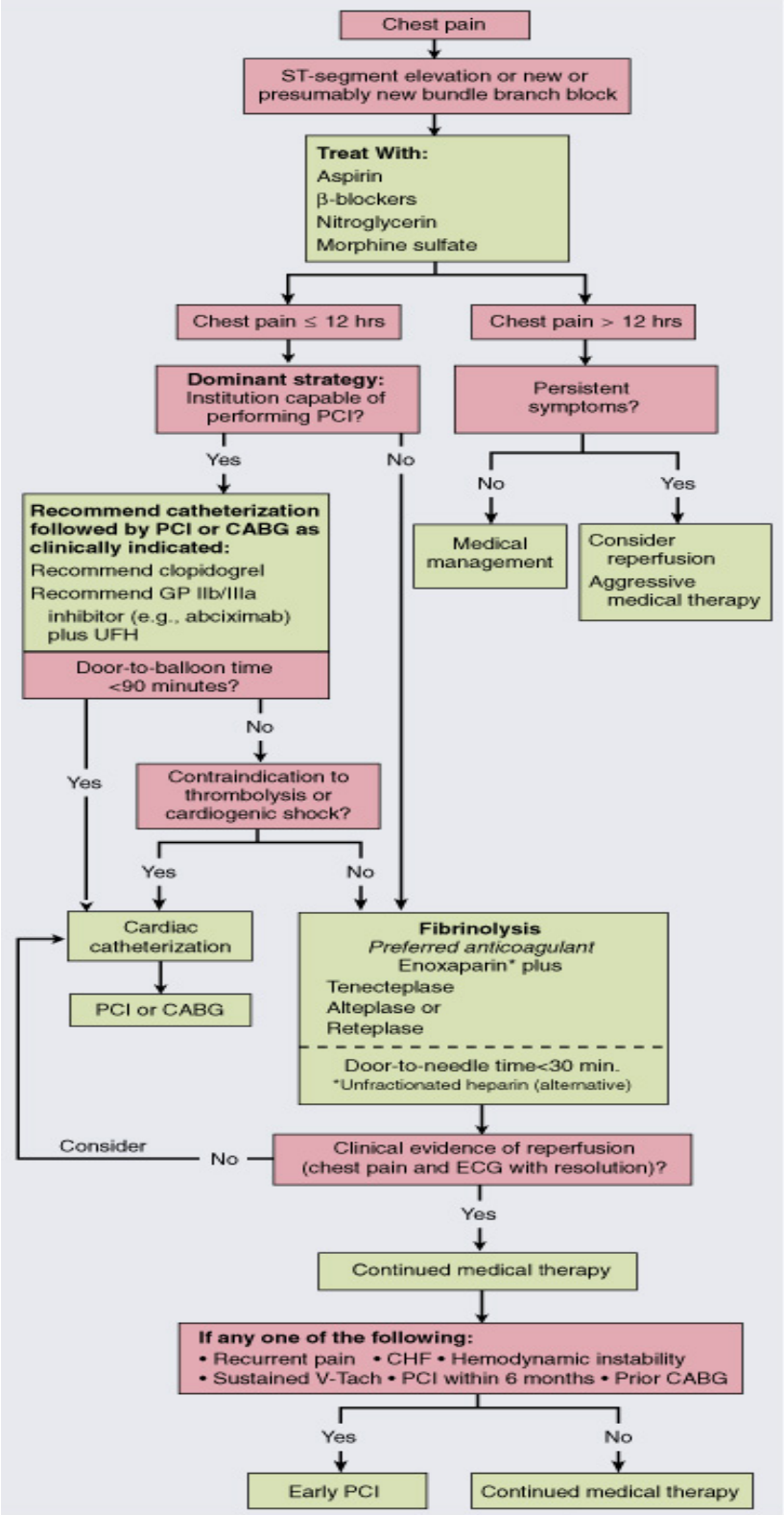
Management of Lipids

With the marked salutary effects on long-term survival, prevention of reinfarction, and subsequent coronary revascularization of HMG-CoA reductase drugs , it is important that a cholesterol panel (total, low-density lipoprotein, high-density lipoprotein) be obtained on admission and no later than the first 24 hours

Thrombolytics or Primary Mechanical Reperfusion

All patients with ST-segment elevation MI who present within 12 hours from the onset of symptoms should be considered for myocardial reperfusion therapy . The list of absolute and relative contraindications is provided in the table.

Contraindications and cautions for fibrinolytic use in myocardial infarction
Contraindications
Previous hemorrhagic stroke at any time; other strokes or cerebrovascular events within 1 yr
Known intracranial neoplasm
Active internal bleeding (does not include menses)
Suspected aortic dissection
Relative contraindications
Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg) or chronic history of severe hypertension
History of prior cerebrovascular accident or known intracerebral pathology not covered in contraindications
Current use of anticoagulants in therapeutic doses (international normalized ratio 2–3); known bleeding diathesis
Recent trauma (within 2–4 wk), including head trauma or traumatic or prolonged (>10 min) cardiopulmonary resuscitation or major surgery (<3 wk)
Noncompressible vascular punctures
Recent (within 2–4 wk) internal bleeding
For streptokinase/anistreplase: prior exposure (especially within 5 d–2 yr) or prior allergic reaction
Pregnancy
Active peptic ulcer


Chest pain
ST-segment elevation or new or presumably new bundle branch block
Treat With:
Aspirin
β-blockers
Nitroglycerin
Morphine sulfate
Chest pain ≤ 12 hrs
Chest pain > 12 hrs
Dominant strategy:
Institution capable of performing PCI?
Persistent symptoms?
Yes
No
No
Yes
Recommend catheterization followed by PCI or CABG as clinically indicated:
Recommend clopidogrel
Recommend GP IIb/IIIa inhibitor (e.g., abciximab) plus UFH
Medical management
Consider reperfusion
Aggressive medical therapy
Door-to-balloon time <90 minutes?
No
Yes
Contraindication to thrombolysis or cardiogenic shock?
Yes
No
Cardiac catheterization
PCI or CABG
Fibrinolysis
Preferred anticoagulant
Enoxaparin* plus
Tenecteplase
Alteplase or
Reteplase
Door-to-needle time<30 min.
*Unfractionated heparin (alternative)
Consider
No
Clinical evidence of reperfusion (chest pain and ECG with resolution)?
Yes
Continued medical therapy
If any one of the following:
• Recurrent pain • CHF • Hemodynamic instability
• Sustained V-Tach • PCI within 6 months • Prior CABG
Yes
No
Early PCI
Continued medical therapy

Acute MI is a clinical syndrome for which a constellation of subjective and objective parameters need to be assessed. The diagnosis must be obtained rapidly and accurately, and misdiagnosis can have catastrophic sequelae. The individual components of making the diagnosis are discussed separately, but it is the integration of all of them that facilitates the accuracy and speed of the clinical syndrome recognition.

RELATED STUDIES

RELATED STUDIES

1. ***Admission blood glucose level as risk indicator of death after myocardial infarction in patients with and without diabetes mellitus. (Archives of internal medicine 2004;164:982-988).***

   In this study 737 non-diabetics were analysed. Of them 101 had admission blood glucose levels around 200 mgs% and mortality in these patients was comparable to that in patients who had established diabetes.

2. ***Is blood glucose an independent predictor of mortality in acute myocardial l infarction in the thrombolytic era. (JOURNAL OF THE AMERICAN COLLEGE OF CARDIOLOGY-vol 40,no.10 2002).***

   In this review it was concluded that hyperglcemia in acute MI was associated with poor outcome even among patients without known diabetes.

3. ***Effects of stress hyperglycemia on acute myocardial infarction.(DIABETES CARE vol.26.no. 11,november 2003).***

   During MI ,hyperglycemia is associated with increased levels of inflammatory markers ,enhanced expression of cytotoxic T-cells,which are implicated in limiting immune process. An increased inflammatory immune response seems a likely mechanism linking acute hyperglycemia to poor outcome in MI patients.

4. Stress hyperglycemia and increased risk of death after MI in patients with and without diabetes ; a systematic review .(THE LANCET vol 355 , march 4,2000).

In this review 14 articles describing 15 studies were identified . patients without diabetes who has blood glucose concentrations ≥range 6.1 -8 mmol/L had a 3.9 fold higher risk of death more than patients without diabetes who had lower glucose concentration.

5. Hyperglycemia and acute MI in a non-diabetic population.(DIABETOLOGIA CROATICA 32-4 ,2003).

The study was done in 412 non-diabetic patients. During their hospital stay ,hyperglycemia was found in 27.43 % patients wihtout previously confirmed diabetes and had normalised in 90.27% of these patients by discharge.

MATERIALS AND METHODS

MATERIALS AND METHODS

Place of study	**Department of Medicine** **TMCH,Thanjavur.**
Type of study	**Prospective study.**
Period of study	**August 2005 – July 2006.**
Ethical Committee Approval	**The present project was approved by the ethical committee.**
Collaborating Department	**Department of Cardiology** **&** **Department of Diabetology.**
Consent	**Informed consent was obtained from the Participants.**

CASE SELECTION

INCLUSION CRITERIA

1. All patients admitted with acute onset of ST elevation Myocardial infarction.

2. Those admitted within 12 hours after the onset of Myocardial infarction.

EXCLUSION CRITERIA

1. Known diabetic, hypertensive or cardio-respiratory illness.

2. Patients on steroids, catecholamines .

3. Those with pancreatitis,acute surgical conditions,hypovolemia&any physical, Physiological or psychological stress.

4. Those treated outside before admission.

5. Those admitted 12 hours after the onset of Myocardial Infarction.

6. Those suffered from Myocardial Infarction earlier.

7. Those with metabolic disorders like acute/ chronic renal failure, hepatic failure,etc.

MATERIALS AND METHODS

A total of 50 consecutive patients admitted at TMCH,Thanjavur,with acute onset of ST elevation MI who satisfied the above criteria.Among these 10 non-diabetic euglycemic patients as a control for the purpose of analysis .

LIMITATIONS

1. ***Small number of patients.***
2. ***Other risk factors were not elicited.***
3. ***Insulin level was not measured.***

SAMPLE COLLECTION

1. ***Ethyl oxide sterilized syringe was used for collecting blood samples.***
2. ***Containers for collecting blood samples were marked, washed with distilled water & dried before collecting blood samples.***
3. ***Blood sugar taken at the time of admission &fasting blood sugar Taken at 24 hrs, 48 hrs & 72 hrs respectively.***

ECG

12 lead ECG was taken following standard methods.

ECHO

Echocardiogram was taken following standard methods.

BLOOD PRESSURE

Blood pressure taken on the day of admission & everyday during hospital stay. Patients with systolic BP>140 mm Hg & diastolic BP>90 mm Hg,were taken as hypertensives.

Criteria for diagnosing stress hyperglycemia

1. ***Blood sugar value of >150 mgs% at the time of admission.***
2. ***Euglycemic fasting blood sugar level on the next subsequent days.***

CATEGORIES

Based on the blood sugar values patients were categorized into 3 groups.

GROUP I Hyperglycemic non-diabetics.(cases)

GROUP II Hyperglycemic diabetics.(newly diagnosed)

GROUP III Euglycemic non-diabetics.(control)

STATISTICS

Significance was considered if the 'p' value is <0.05. For selected variables correletation studies were made.

PROFORMA

PROFORMA

NAME: **AGE: YRS** **SEX:M/F**
OCCUPATION: **I.P. NO:**
ADDRESS: **D.O.A:**
D.O.D:

SYMPTOMS AT THE TIME OF ADMISSION:
☐ CHEST PAIN / ☐ DYSPNOEA / ☐ SWEATING / ☐ PALPITATIONS
☐ GIDDINESS / ☐ OLIGURIA / ☐ PAIN & SWELLING IN JOINTS

PAST HISTORY:
☐ HYPERTENSION/ ☐ DIABETES MELLITUS / ☐ CHRONIC ALCOHOLISM/
☐ IHD/ ☐ DYSLIPIDEMIA/ ☐ DRUG INTAKE

ECG:

KILLIP'S CLASSIFICATION: I / II / III / IV

THROMBOLYSED : ☐ **NOT THROMBOLYSED** : ☐

INVESTIGATIONS:
a. Hb % : b. TC : c. DC :
d. URINE ➲ ALB: SUGAR: DEPOSITS:
e. BLOOD SUGAR: f: BLOOD UREA:
g. SERUM CREATININE:
SERUM ELECTROLYTES: Na+: K+:

i. ECHOCARDIOGRAM :

Sl. No.	VARIABLES	ON ADMISSION	DAY 2	DAY 3	ON DISCHARGE
1	BLOOD SUGAR (mg%)	(RANDOM)	(FASTING)	(FASTING)	(FASTING)
2	ARRYTHMIAS				
3.	CARDIOGENIC SHOCK				
4.	DEATH				

OBSERVATIONS

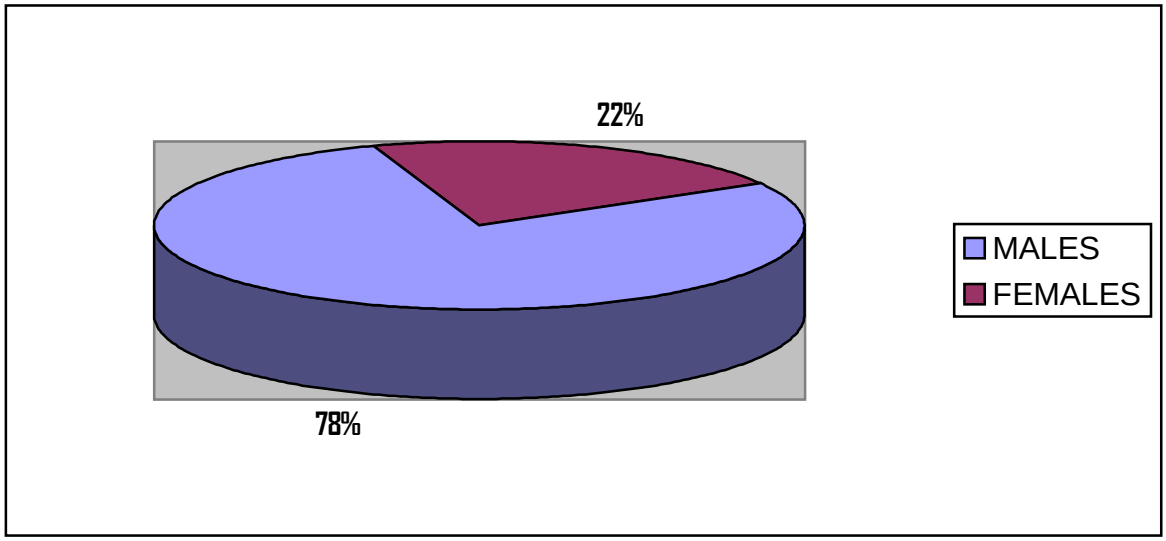
OBSERVATION

In the present study there were 39 males and 11 females out of 50 patients. 20 patients where under Group I with stress hyperglycemia & normal fasting blood glucose levels, 20 patients were under Group II with hyperglycemia & newly detected diabetes mellitus and 10 patients were under Group III with euglycemia and Non diabetics.

Distribution of patients according to group and sex

GROUP	NUMBER OF CASES	MALE	FEMALE	PERCENTAGE
I	20	15	5	40%
II	20	15	5	40%
III	10	9	1	20%

GENDER DISTRIBUTION

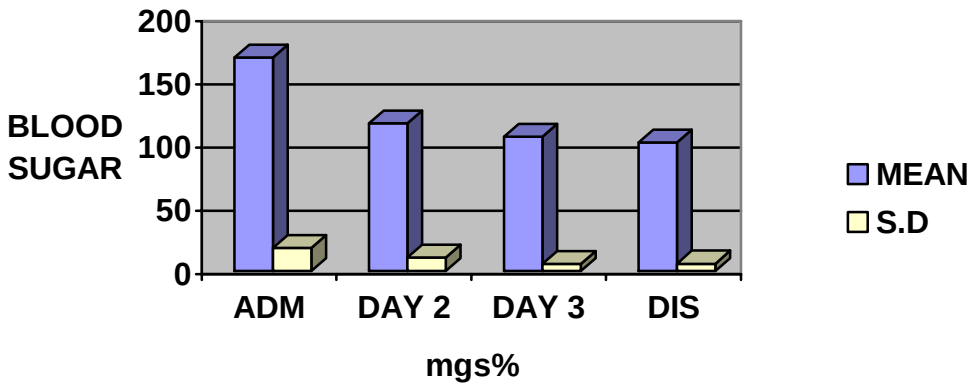


No significant correlation from each other .The age of the patients studied ranged from 32 yrs to 70 yrs. The mean age of patients with Group I , Group II and Group III were 56 ± 14 , 57 ± 13 ,52 ± 13.5 years respectively.

BLOOD SUGAR
GROUP I

NUMBER OF DAYS	RANGE (mg%)	MEAN (mg%)	S.D
ON ADMISSION (RANDOM)	152-218	168.6	18
DAY 2 (FASTING)	98-140	116.5	10.5
DAY 3 (FASTING)	100-116	106	5.3
ON DISCHARGE (FASTING)	94-110	101.3	5.7

BLOOD SUGAR RANGE FOR GROUP I

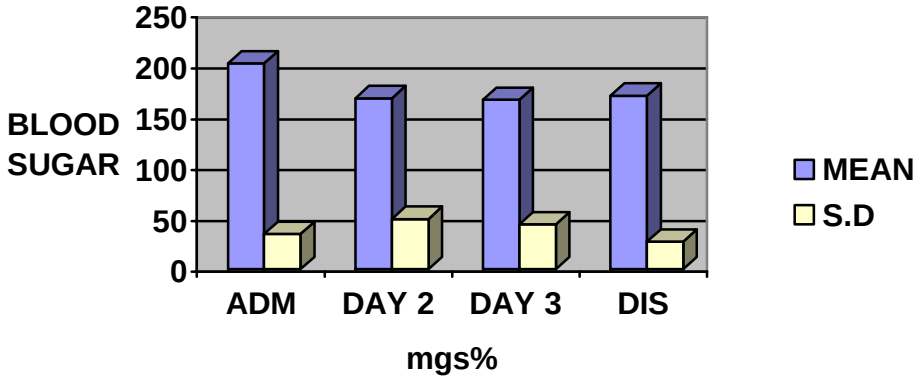


Under Group I blood sugar level at the of admission were in the range of 152-218 mgs% with a mean of 168.6 mgs%.
A reduction in the blood sugar was noticed on subsequent days and it was statistically significant (p value < 0.001).

GROUP II

NUMBER OF DAYS	RANGE (mg%)	MEAN (mg%)	S.D
ON ADMISSION (RANDOM)	154-272	201.85	34.4
DAY 2 (FASTING)	108-286	167.65	49.05
DAY 3 (FASTING)	108-276	165.95	43.5
ON DISCHARGE (FASTING)	132-218	170.23	26.73

BLOOD SUGAR RANGE FOR GROUP II

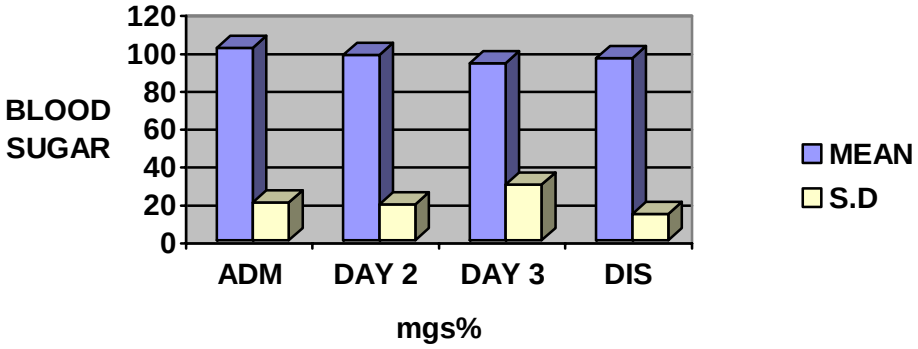


In this category , there is persistent elevation of fasting blood sugar level in the subsequent days.

GROUP III

NUMBER OF DAYS	RANGE (mg%)	MEAN (mg%)	S.D
ON ADMISSION (RANDOM)	68-124	101.6	19.9
DAY 2 (FASTING)	57-120	98	19.1
DAY 3 (FASTING)	46-122	93.7	20.4
ON DISCHARGE (FASTING)	80-116	96.2	14

BLOOD SUGAR RANGE FOR GROUP III



In this category no marked reduction in blood sugar level noted.In the above three groups there was a positive correlation between Group I & Group II and there was a negative correlation between Group I & III.

CLINICAL ASPECTS

Clinical aspects were analysed under the following headings.

1. ***Type of myocardial infarction***
2. ***Killip classification***
3. ***Complications***
4. ***Hospital stay***
5. ***Ejection fraction***
6. ***outcome***

TYPE OF MYOCARDIAL INFARCTION

GROUP	IWMI	AWMI	ASMI	ALMI	LWMI	PWMI
Group I	10	7	1	1	1	0
Group II	9	6	3	0	0	2
Group III	2	6	1	0	1	0

Out of 50 patients enrolled in the study 21(42%) of patients presented with acute IWMI ,19(38%) of patients presented with acute AWMI,5(10%) of patients presented with acute ASMI,2(4%) presented with LWMI and 2(4%) presented with true PWMI.

KILLIP CLASS

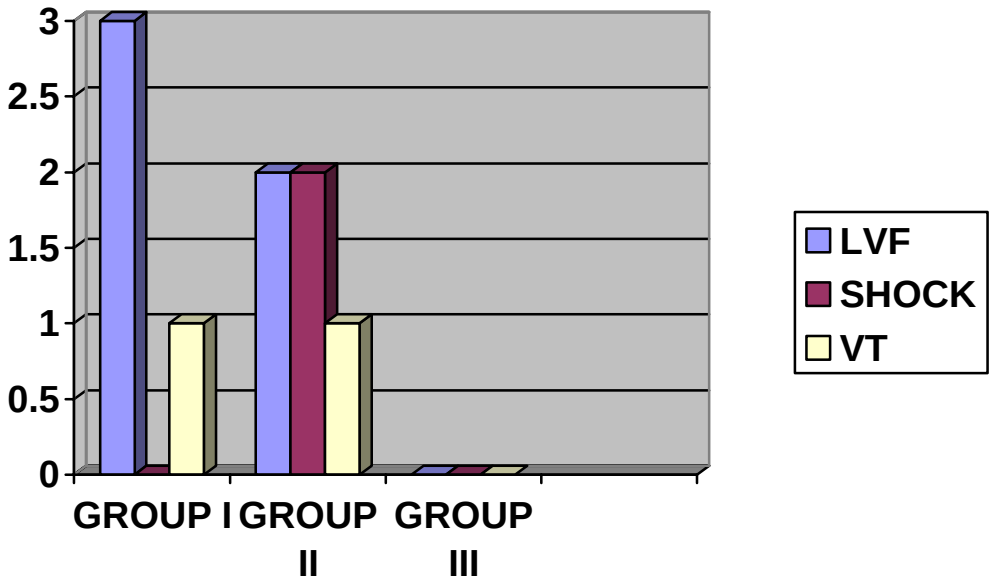
GROUP	I	II	III	IV
Group I	12	3	3	2
Group II	14	4	2	0
Group III	8	2	0	0

Most of the patients (68%) were coming under Killip class I , 18% under Killip class II ,10% under Killip class III and 4 % under Killip class IV. All patients with acute myocardial infarction were transferred to intensive coronary care unit ,where the patients were thrombolysed.9(18%) were not thrombolysed due to very long median delay after the onset of symptoms .

COMPLICATIONS

GROUP	LVF	SHOCK	VT
Group I	3	0	1
Group II	2	2	1
Group III	0	0	0

COMPLICATIONS OF MI

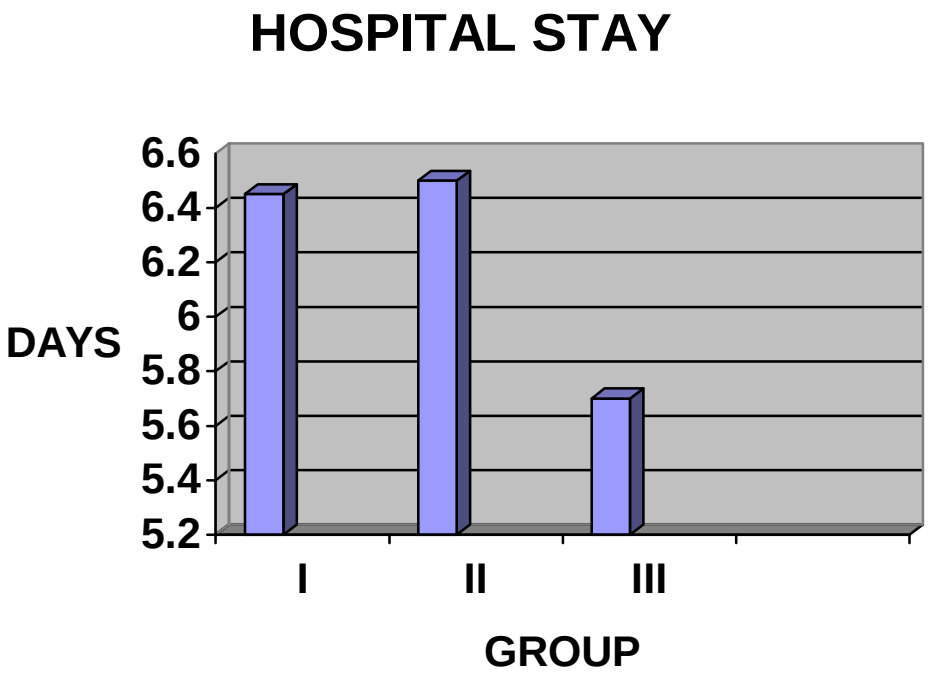


Out of 50 patients admitted with acute myocardial infarction 5(10%) of them developed LVF ,2(4%) of them developed cardiogenic shock, 2(4%) of them developed ventricular tachycardia.

After analysing the individual groups ,in group I 4(8%) of the patients developed complications like LVF,VT &shock. Under group II 5(10%) of the patients developed complications . Under group III no complications has been observed .There was a significant ($p < 0.05$) difference was noticed with reference to the complications among the Group I and Group III. There was no significant difference among the Group I and Group II patients.

HOSPITAL STAY

GROUP	NUMBER OF DAYS
Group I	6.45
Group II	6.5
Group III	5.7

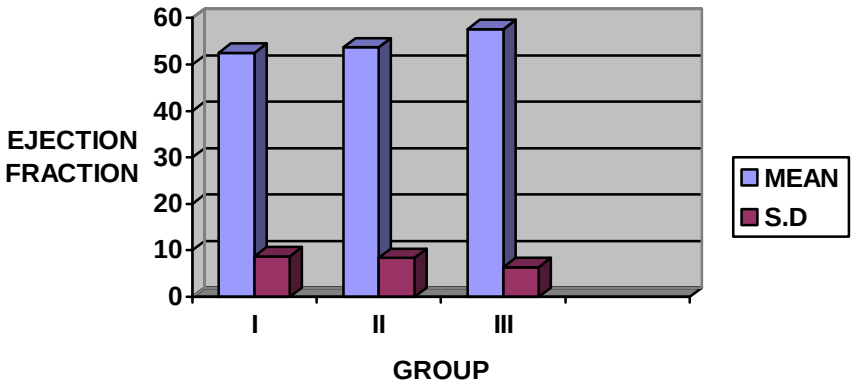


Hospital stay in Group I and Group II was prolonged due to development of complications.

EJECTION FRACTION

GROUP	*RANGE(%)*	*MEAN(%)*	*S.D*
Group I	*40-65*	*52.5*	*8.7*
Group II	*38-67*	*53.7*	*8.4*
Group III	*46-68*	*57.6*	*6.4*

EJECTION FRACTION

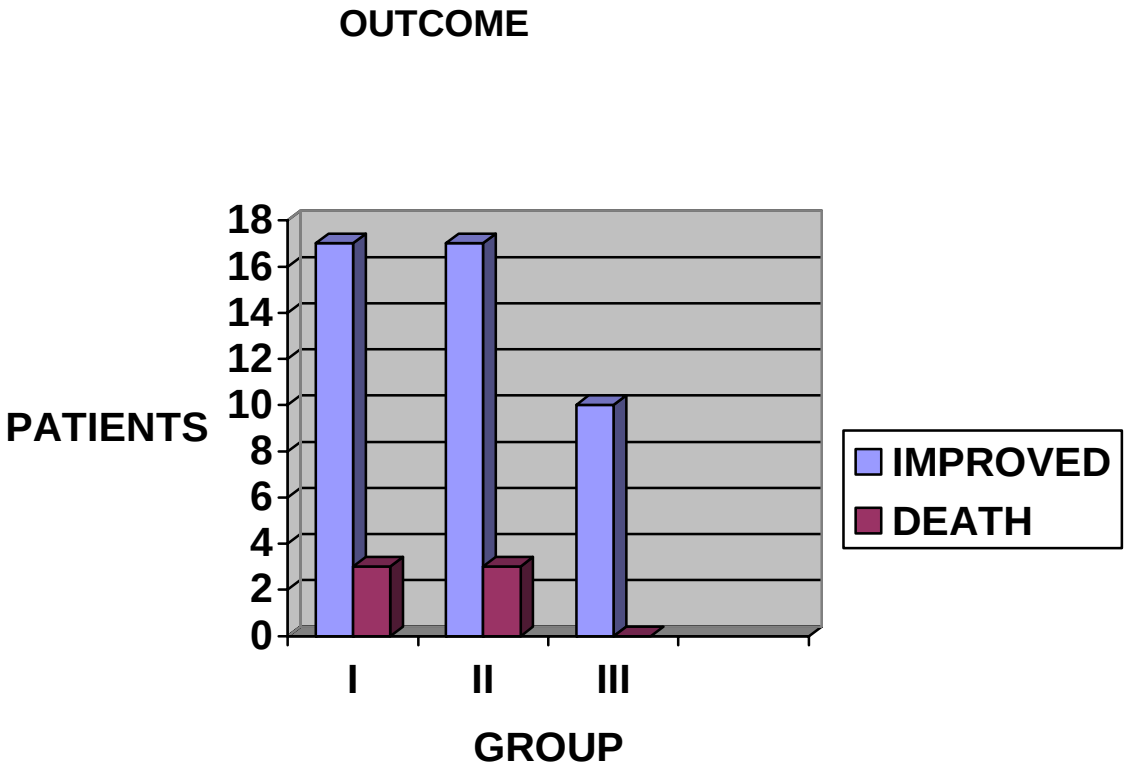


Under group I the mean value of ejection fraction was 52.5% ,under group II the mean value of ejection fraction was 53.7% and under group III the mean value of EF was 57.6%.On comparing group I and III was a significant ($p < 0.05$) reduction in the ejection fraction.There was no much of difference among group I and group II patients.

OUTCOME

Outcome was considererd based on their study in the hospital as improved or expired .the details are furnished in the table given below.

GROUP	IMPROVED	DEATH
Group I	17	3
Group II	17	3
Group III	10	0



DISCUSSION

DISCUSSION

Myocardial Infarction continues to have a devastating impact on public life as well as personal life savings of an individual and or family.Myocardial Infarction places a tremendous burden on the health resources all over the world.

AGE

Age group in this present study ranges as shown below in the three groups.

GROUP	*Age in years*			TOTAL
	Range	*Mean*	*SD*	
Group 1	*42-85*	*57.7*	*8.4*	*20*
Group 2	*38-70*	*57.4*	*9.6*	*20*
Group 3	*35-60*	*51.9*	*8.8*	*10*

A comparison was made with reference to age of the three groups and among each other and they were found to be not significant.

GENDER

There are about 39 males and 11 females in this study. The three groups were not statistically different from each other with reference to gender.

RISK FACTOR - HYPERTENSION

The details with reference to the study group are provided below.

GROUP	SYSTOLIC BP mmHg			DIASTOLIC BP mmHg			HYPERTENSIVES
	Range	Mean	SD	Range	Mean	SD	
Group 1	90-150	124.3	18.43	50-96	79	12	7
Group 2	80-196	135	35.9	50-120	86.1	20.9	9
Group 3	100-150	125	16.5	60-96	80.4	11.8	3

There is a negative correlation between blood sugar& hypertension.

So it is not statistically significant.

CLINICAL ASPECTS

At the time of admission patients coming under group1 & group 2 presented with complications like cardiogenic shock ,arrythmias and left ventricular failure.In this study , under group 1 , 3out of 20 patients developed LVF and 1 out of 20, VT when compared to euglycemic group III patients.

There was no correlation with the pattern of myocardial infarction and hyperglycemia.

EJECTION FRACTION

GROUP	Range	Mean	SD
Group I	40-65%	52.5%	8.7
Group II	38-67%	53.7%	8.4
Group III	46-68%	57.6%	6.4

In this study under group 1 the mean value of ejection fraction was (52.5 ±8.7)% and under group III was(57.6±6.4)%. On comparing group I & III patients there was a significant (p=0.001) reduction in the ejection fraction. There was not much difference among group I & II patients. There is a positive correlation between group I and ejection fraction(admission blood glucose affects the ejection fraction).

HOSPITAL STAY

GROUP	No.of.Days
Group I	6.45
Group II	6.5
Group III	5.7

In this study some of the group I and group II patients need to stay in the hospital for a prolonged period, because these hyperglycemic groups develop complications during their hospital stay. But it is not statistically significant.

OUTCOME

Outcome was considered based on their study in the hospital as improved or expired. The details are furnished in the table given below.

GROUP	IMPROVED	DEATH
Group I	17	3
Group II	17	3
Group III	10	0

Mortality was commonly noted in the hyperglycemic groups. 3 deaths were commonly noted in group I & II patients due to complications, but it was not statistically significant.

CONCLUSION

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CONCLUSION

1. In the previously undiagnosed healthy individuals admitted with acute myocardial infarction 80% of them had elevated blood sugar at the time of admission out of which 40% had stress hyperglycemia and it came down within 24-48 hours.

2. Elevated admission blood sugar is independent of factors like age ,sex, smoking, hypertension, alcoholism and clinical status in this study.

3. Admission blood sugar was elevated to the mean value of 168.6 mg% in non-diabetic stress hyperglycemic middle-aged and elderly individuals in contrast to the non-diabetic euglycemic group.

4. In this study death was significantly higher in diabetic and non-diabetic hyperglycemic individuals with acute myocardial infarction when compared to non-diabetic euglycemic group. Thus indicating that the admission blood glucose is a predictor of early outcome in acute myocardial infarction.

5. Ejection fraction was significantly reduced(53.1 ±8.55)% in hyperglycemic myocardial infarction patients.

SUMMARY

SUMMARY

A total of 50 consecutive previously stable active and independent individuals free from any drugs or overt illness with established features of acute myocardial infarction admitted within 12 hours were evaluated clinically and biochemically to find out the prevalence of elevated blood sugar response to correlate with hospital outcome as well as selected risk factors.

Blood sugar was elevated in 80% of patients and 40% had stress hyperglycemia.Elevated blood sugar was not related to demographic factors like age and sex,habits like smoking and alcohol, risk factors like hypertension and clinical status like type of myocardial infarction.There is a positive correlation between the blood sugar value and the ejection fraction.

Death was noted in both diabetic and non-diabetic hyperglycemic myocardial infarction patients. Thus indicating that the admission blood glucose level is a predictor of early outcome in acute myocardial infarction.

FUTURE PROSPECTIVES

Even though the prognosis in acute MI is greatly affected by the elevated blood glucose level, at present there are no related studies regarding the intervention of admission blood glucose level. So it needs further studies regarding the management protocol for stress hyperglycemia.

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MASTER CHART

(PLEASE VIEW IN NORMAL VIEW)

S.NO	NAME	IP NO	AGE	SEX	BLOOD SUGAR mgs%				DM	BP (mm/Hg)	MI
					ADM	DAY2	DAY3	DIS			
											GRO
1.	Nadarajan	881295	65	m	156	112	100	98	no	126/84	AV
2.	Sakuntala	872222	45	f	218	140	106	100	no	140/90	IW
3.	Jaithmbivi	860011	55	f	162	110	106	100	no	130/86	AV
4.	Cinnathal	879910	70	f	166	98	100	96	no	146/90	IW
5.	Selvaraj	885449	56	m	160	112	100	94	no	130/80	IW
6.	Gunasekar	880012	52	m	216	114	108	110	no	140/96	AV
7.	Venkat	867534	50	m	163	116	114	110	no	90/60	AV
8.	Meenatchi	879012	60	f	165	102	102	96	no	110/70	IW
9.	Lakshmi	861234	70	f	152	112	104	96	no	90/50	IW
10.	Ashok	874567	42	m	166	104	-----	----	no	120/80	IW
11.	Manickam	882125	76	m	184	124	106	104	no	130/84	IW
12.	Rajendran	881904	45	m	160	124	110	96	no	134/86	AV
13.	Chandran	876389	50	m	158	126	110	100	no	140/90	AV
14.	Elangovan	872451	52	m	164	118	114	106	no	150/96	IW
15.	Govindaraj	871045	70	m	156	116	104	96	no	140/92	AV
16.	David	871035	62	m	160	104	102	100	no	110/70	HI
17.	Nagaratnam	860952	67	m	166	116	108	110	no	90/60	AI
18.	Vijayan	862311	48	m	166	130	116	108	no	120/80	IW
19.	Nathan	881722	56	m	170	120	108	106	no	150/90	AS
20.	Prasannan	865232	45	m	164	132	96	98	no	110/70	IW
											GRO
1.	Rramayan	860591	45	m	158	192	130	208	yes	100/70	AV
2.	Ganapathi	860670	67	m	228	228	184	212	yes	130/80	AV
3.	Muniamuthu	862125	64	m	210	163	126	140	yes	126/70	IW
4.	Jameel	862367	55	m	217	132	108	170	yes	130/80	IW
5.	Kanagammal	864033	70	f	272	110	134	169	yes	130/80	IW
6.	Rajagopal	871084	50	m	180	114	136	148	yes	140/96	IW
7.	Mohammed	871209	52	m	164	140	180	165	yes	180/120	IW
8.	Jayaraman	872795	68	m	210	209	228	180	yes	140/90	IW
9.	Ilamaran	872993	60	m	180	108	139	-----	yes	80/60	IW
10.	Mangayarkarasi	872583	70	f	200	140	140	184	yes	130/86	PM
11.	Kannapillai	873749	70	m	232	286	276	-----	yes	80/50	AV
12.	Raja	877244	32	m	154	180	164	160	yes	150/96	AV
13.	Rajalakshmi	881552	50	f	180	130	140	136	yes	140/90	AV
14.	Kathija bivi	878910	60	f	208	148	160	170	yes	100/60	IW

15.	Selvam	881884	50	m	236	240	242	218	yes	160/100	PV
16.	Detchinamoorthi	862000	45	m	158	195	180	180	yes	160/110	IW
17.	Thumbusami	861493	65	m	180	136	146	136	yes	110/70	AS
18.	Gnathammal	861226	70	f	190	146	156	-----	yes	180/100	AS
19.	Kumaran	860016	60	m	260	220	210	186	yes	140/90	AS
20.	Rajan	869531	45	m	220	136	140	132	yes	130/86	AV
											GRO
1.	Manikandan	861506	48	m	124	100	75	98	no	130/86	AV
2.	Mathivanan	862097	55	m	123	115	46	80	no	110/70	AV
3.	Subramanian	862481	62	m	100	90	98	104	no	150/96	IW
4.	Manikandan	863825	61	m	103	80	94	86	no	140/90	IW
5.	Palanivel	869847	50	m	84	90	106	98	no	130/80	AV
6.	Paneerselvam	869892	48	m	120	110	98	116	no	100/80	AV
7.	Suresh	872920	35	m	68	57	96	86	no	110/76	AV
8.	Vedaratnam	876094	55	m	79	110	98	76	no	130/80	AV
9.	Kaliyaperumal	882080	45	m	119	120	122	116	no	140/96	HI
10.	Seethammal	862367	60	f	96	108	104	102	no	110/70	AS