"A STUDY ON STRESS HYPERGLYCEMIA IN MODERATE DEGREE BURNS"

Dissertation submitted to

THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY

CHENNAI

In partial fulfilment of regulations

for award of the degree of

M.D. (General Medicine)

Branch – I

KILPAUK MEDICAL COLLEGE,

CHENNAI

APRIL - 2013

BONAFIDE CERTIFICATE

This is to certify that **"A STUDY ON STRESS HYPERGLYCEMIA IN MODERATE DEGREE BURNS"** is a bonafide work performed by **Dr.H.ANURADHA,** post graduate student, Department of Internal Medicine, Kilpauk Medical College, Chennai – 10, under my guidance and supervision in fulfilment of regulations of the Tamilnadu Dr.M.G.R. Medical University for the award of M.D. Degree Branch I (General Medicine) during the academic period from May 2010 to April 2013.

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DECLARATION

I, **Dr.H.ANURADHA** solemnly declare that this dissertation "**A STUDY ON STRESS HYPERGLYCEMIA IN MODERATE DEGREE BURNS"** was prepared by me at Government Kilpauk Medical College and Hospital, Chennai, under the guidance and supervision of **Prof.Dr.G.BALAN, M.D,** Professor and Unit Chief, Department of Internal Medicine, Govt. Kilpauk Medical College and Hospital, Chennai.

This dissertation is submitted to **The Tamil Nadu Dr. M.G.R. Medical University, Chennai** in partial fulfillment of the University regulations for the award of the degree of **M.D. Branch I (General Medicine).**

Place : Chennai

Date : **(Dr. H.ANURADHA)**

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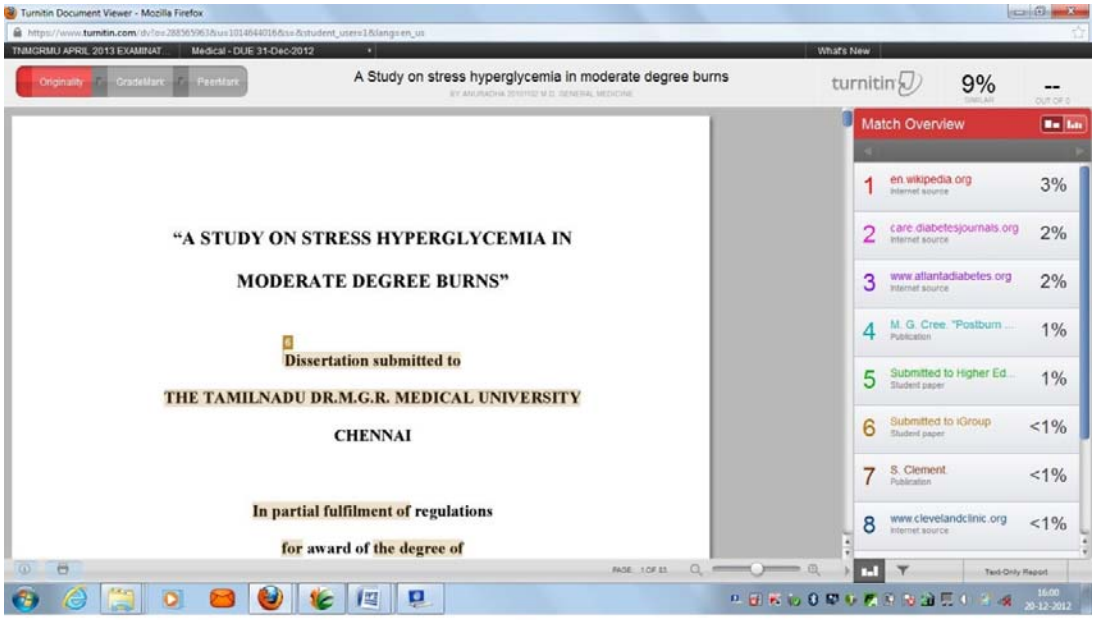
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CONTENTS

A STUDY ON STRESS HYPERGLYCEMIA IN MODERATE DEGREE BURNS

ABSTRACT

BACKGROUND - Stress hyperglycemia is a transient event but has its own morbidity and mortality. In this study we saw the prevalence of stress hyperglycemia in moderate degree burns and its impact on patient outcome.

MATERIALS AND METHODS: 150 patients were taken up for study.Patients with moderate degree burns 15-30%, age >18 years, non diabetics were taken up for study.Patients who diabetics ,age<18 years, gestational diabetes,on steroids were excluded from the study. Patients with random blood sugar >200 on admission were seen three days fasting sugar and one postprandial sugar.Patients were diagnosed as diabetic according to ADA criteria.Patients with hyperglycemia were seen HbA1c to rule out stress hyperglycemia and diabetes, since HbA1c showed last three months sugar status.

RESULTS: Among 150 patients 133patients were stress hyperglycaemic 17 were newly detected diabetes .Stress hyperglycaemia caused longer duration of stay >15 days,p value (0.009).Males had longer duration of stay,p value(0.034) .It was more common among young females aged less than 30

years,p value(0.048). More common among younger age group.Non pseudomonas organism(staphylococcus, klebsiella, proteus etc) was the most common organism found, p value(0.005). Staphylococcus was the common single organism and Staphylococcus and Klebsiella was combined organism.The hyperglycaemic sugar range was 200-250mg/dl .Non pseudomonas organism was more in younger age group p value (0.005). No mortality was seen in stress hyperglycaemic patients. Mortality was seen in three patients who were diabetic and they were infected with combined organism.

CONCLUSION: Thus from the study we see stress hyperglycemia causes increased morbidity in the form of longer duration of stay,infection ,and affects younger age group. 17new diabetic cases were detected.

1. INTRODUCTION

Stress hyperglycemia (stress diabetes or diabetes of injury) is a elevation of the blood glucose for any stress in the form of illness. It gets corrected spontaneously, but has to be differentiated from various forms of diabetes mellitus. Stress hyperglycemia indicates state of insulin secretory capacity which is reduced or a reduced sensitivity to the insulin, and is the first clue to incipient diabetes.

It occurs in acute stressful state which includes stroke, burns, myocardial infarction and any critical illness. It's due to the over activity and over production of counter regulatory hormones. Burns is a highly acute stressful state, caused by heat, electricity, chemicals, light, radiation or friction.[123] Following the injury, there appears to be varied metabolic changes , which includes energy expenditure, protein break down which is elevated, and alterations in fat metabolism.

Insulin resistance is the major factor for all effects, because more data have shown insulin resistance and its association with liver function derangement as a cause for increasing the morbidity and mortality which is seen in burned patients.[456] The metabolism of fat is altered postburn; which is related to changes in insulin resistance. The high sugar alters the outcome in burns in the form of graft uptake and sepsis. There are few studies

regarding burns and its hyperglycemia, either due to stress or diabetes. Therefore this study aims at finding out hyperglycemia due to stress or diabetes in burns and its outcome.

2. AIM OF STUDY

1. To find out prevalence of stress hyperglycemia in moderate degree burns patients.
2. To find out stress hyperglycaemic outcome in moderate degree burns patients.

3. REVIEW OF LITERATURE

The burns and hyperglycemia relationship has been dated back to the Viet Nam war.[7] Hyperglycemia due to insulin resistance may occur due to elevation of counter regulatory hormones, inflammatory cytokines, intravenous fluids in the form of dextrose support, and steroid treatment.[8] Even people who were not diabetic can become hyperglycemic and approximately 25% of traumatic patients with no history of diabetes can develop hyperglycemia.[910] Stress hyperglycemia in illness is elevation in the blood sugar due to the illness which is a stress. It corrects or get controlled after the stress but to rule out diabetes .

Most of the burns injury affect only the superficial skin.Rarely, the deeper structures of muscle, bone, and blood vessels be affected. In the patients with burn injury insulin resistance is what being recognized.

3.1 EPIDEMIOLOGY:-

The number of studies regarding stress hyperglycemia is less . Umpierrez et al.[11]showed prevalence of diabetes around 26% in hospitalized patients . Among them 12% of patients were newly diagnosed diabetes or stress related hyperglycemia. Levetan et al. [12] showed a 13% prevalence of hyperglycemia in 1,034 consecutively hospital inpatients. Based on hospital chart review, 64% of patients with hyperglycemia were known diabetic or were newly detected diabetesin the hospital stay.Thus average incidence is 12-13%.Till 2004, 11 million burn injured people were seeking hospital care throughout the world.[13] 90% of burns occurs in the emerging countries and among them 70% of them were pediatric aged. Mortality was more for burns with distribution of 40% of body area found in the developing countries [14].In India about 700,000 people suffer from injury of burns and getting treated from hospital, while very few seek care in higher instituitions.

3.2 BURNS:-

A **burn** is a injury to the body skin and flesh which is caused by heat contact, lightening, chemicals, electricity. The part of the body mostly affected is the skin dermis and epidermis. In severe burns deeper tissues may be affected like muscle and bone .It can also affect the blood vessels and can be more painful. It can be treated by simple methods out of hospital or can be treated at higher centres. The treatment involves removal of dead tissue, wound debridement, skin grafting, antibiotics and fluids. Burns can be very fatal but due to modern treatments prognosis is good.

Burns can be classified by the type of injury, depth of injury, extent and other co morbid conditions. Burns are classified according to the depth of injury to the skin and are categorized into four degrees. This classification was given by French barber-surgeon Ambroise Pare and till date, it is in use.[15]

The depth of burn is very difficult to determine. It is more difficult for second degree burns since it evolves over time. The difference between superficial thickness and partial thickness burns is important for treatment because superficial burns heal spontaneously but partial thickness burns requires surgical grafting.

The following tables describe degrees of burn injury

Names	**Layers involved**	**Appearance**	**Texture**	**Sensation**	**Time to healing**
First degree	Epidermis	Redness	Dry	Pain +	< 1weeks
Second degree	Extends into superficial dermis	Red colour blister.	Moist	Pain +	2-3weeks
	Extends into deep dermis	Red and white blisters. Blood +	Moist	Pain +	Weeks- may progress to third degree
Third degree (full thickness)	Extends till entire dermis	White or Brown	Dry, leathery	Pain absent	Excision Required
Fourth degree	Skin, subcutaneous tissue, muscle, bone	Charred, Black, Eschar +	Dry	Pain absent	Excision Required

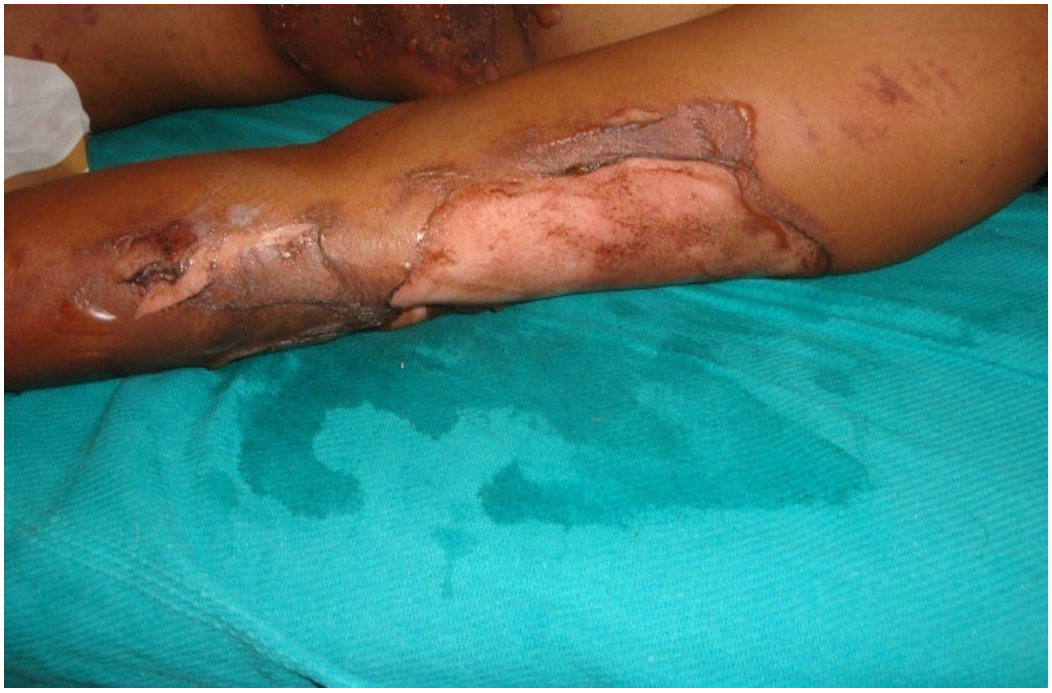


Figure - 1 First and Second Degree Burns

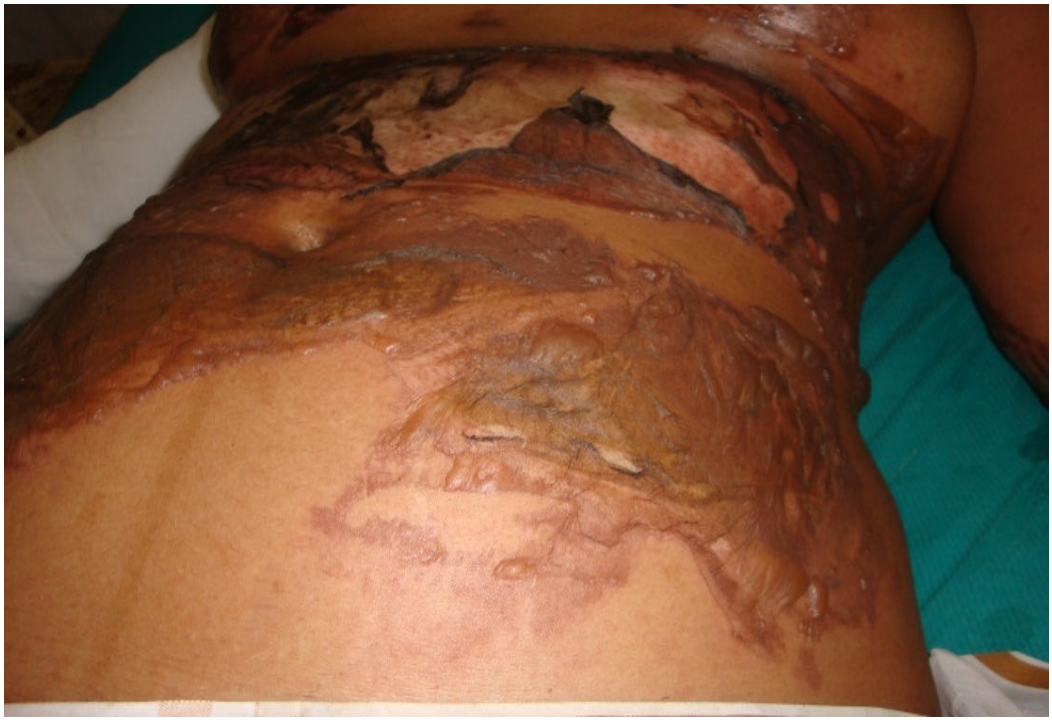


Figure - 2 Second Degree Burns

Figure- 3, Third-Fourth Degree Burns

3.3 CAUSES OF BURNS:-

There are number of causes of burns. They are chemical,electrical, radiation and scalding.

3.3.1 CHEMICAL: -

Among the chemicals it is caused by corrosives or caustic alkalis[16] The caustic compounds includes sodium hydroxide or silver nitrate, and acids such as sulfuric acid[17] Hydrofluoric acid results in deep burns and sometimes not quickly evident.[18] Chemical burns can be of any degree, depending on the nature of substance.

3.3.2 ELECTRICAL :-

Electrical burns are due to an electric shock or short circuit. Electrical burns most commonly include workplace injuries, domestic current injuries, defibrillation, or cardio version without a conductive gel. Though visible burn injury may not be seen with electrical burns, it could be fatal due to internal injury. It could be due to cardiac arrhythmias. Lightning is also a rare cause of electrical burns. It causes cardiac arrest and ventricular fibrillation but generates relatively low heat energy deposit into skin, thus producing few or no burn marks at all.[19] High voltage electricity, causes very deep seated wounds. It is common cause for deeper burns due to the

raised temperature and flashes associated with the voltages which is around thousand volts.

3.3.3 RADIATION:-

Radiation burns are caused by prolonged exposure to ultra violet light from sun, cancer treatment with radiation, sun lamps, radioactive fallout and X-rays. The most common radiation type of burn is due to sun exposure.It is due to the ultraviolet light A and B. B light wavelength is more dangerous. With more severe sun radiation it results in sun poisoning or "heatstroke". Tanning booths also cause burn injury. They cause skin irritation, reddish discoloration, swelling and inflammation. Microwave burns are caused by the thermal effects of microwave radiation.

3.3.4 SCALDING:-

Scalding (meaning hot in latin [20]) results from hot substances like liquids and gases. A so called *immersion scald* occurs when hands or legs are immersed in hot water, and is the most common burn seen in child abuse[21]. A blister is a serous fluid filled lesion in the skin. It occurs as a response to the heat and inflammatory reaction. The top of blister is peeled but the fluid has toxic substances. Scald type of burns are more often seen in children. Generally scald burns are first or second degree burns, third degree burns can occur when there is prolonged contact.

3.4 PATHOPHYSIOLOGY

3.4.1 BURNS :-

Burn injury results in a local inflammatory response and in larger burns there is a systemic inflammatory response. The lungs are affected due to smoke inhalation and venous return from the burned circulation. After burns injury,tachycardia and resistance increase peripherally. This is due to the release of epinephrine, nor epinephrine,etc from injured tissues, and the relative decrease in blood flow due to change in body fluids. Initially heart ejection of blood is reduced but after 24hours post burn injuries, ejection becomes normal if treated with adequate fluids. The output later meets the metabolic needs of the body.

High degree of heat injury results in various chemical reactions and alteration in protein structure. [22]

The pain fibres are stimulated due to heat and protein structures are altered which results in damage to body parts. The pain fibres which are stimulated release neuropeptides. Activation of complement occurs. Immune complex proteins attract the neutrophils which degranulate to release free radicals and proteases causing the damage. Mast cells when undergone degranulation releases the tumor necrosis factor – α (TNF – α, primary cytokine). This TNF – α is chemo attractant to other inflammatory

cells, and these inflammatory cells release secondary cytokines.[23] These cytokines which are secondary increases the permeability of blood vessel in burn area. This causes exudation of proteins and fluid into the adjacent interstitial tissue. Red cells are not extravasated. As a result, there is increase in the oncotic pressure in the interstitium. The volume of fluid loss is directly proportional to the area of burn injury. If burn area is 10% to 15% of the total body surface area (TBSA) the consequent fluid loss may cause circulatory shock. If the area is larger than 25% of TBSA, infection and inflammation of adjacent tissues results in more volume loss.[23]

3.4.2 STRESS AND HYPERGLYCEMIA:-

Hyperglycemia due to stress is due to a highly complex interplay of counter-regulatory hormones. This includes epinephrine, norepinephrine, growth hormone, cortisol, and cytokines.[24,25] The cytokine production and hormonal derangements may be affected by underlying illness. There exists complex mechanism between hormones and inflammatory cells [26] and this complex environment ultimately leads to more liver glucose synthesis and ineffective action of insulin . More liver production of carbohydrates by gluconeogenesis, plays major role in hyperglycaemia.[27,28]. The increased glucagon is the primary mediator of gluconeogenesis, although it occurs with epinephrine[29] and cortisol[30] which also contribute. Tumour necrosis

factor-α (TNFα) also promotes gluconeogenesis by stimulating the glucagon hormone .[31]

The resistance of insulin is seen as inability to suppress the glucose release from liver. In the periphery, insulin resistance occurs by two major pathways. The decreased insulin-mediated uptake of glucose occurs because of reduced signalling for receptor[32]and downregulation of glucose transporter (GLUT)-4.[33] super added with this , decreased clearance of glucose occurs since glycogen synthesis is affected in skeletal muscle.[34] Both increased cortisol[35]and increased epinephrine[36] reduce insulin actions . Cytokines such asTNFα[37] and intrleukin 1[38] inhibit post receptor signalling of insulin. The consequences of illness results from the reduced glucose uptake. Furthermore, hyperglycaemia increases the cytokine, inflammatory, and oxidative stress response, which sets up a vicious cycle whereby raised sugar further increases sugar.[39-41]There is a insulin resistance state which promotes a catabolic state, where lipid lysis takes place . The excess fatty acid further causes more insulin resistance. This in turn causes more glucotoxicity. Glucotoxicity, lipotoxicity, and inflammation occurs, in which are the key components in the exaggerated global insulin-resistance syndrome associated with acute form of illness. It also causes endothelial dysfunction, which has relation with insulin resistance. Hyper insulin state further increases hormone production and

impairs fibrinolysis. There is also glucose uptake due to GLUT 1 upregulation. It is glucose transporter involved in non insulin mediated glucose uptake .Though non oxidative metabolism is impaired oxidative metabolism is up regulated. In addition therapeutic interventions like steroids, enteral and parentral nutrition, fluids, catecholamine infusions can worsen hyperglycamia. Thus there are no studies comparing the outcome of diabetes and stress hyperglycemia and also to see whether the pathophysiology is same for both.

The following diagram and flow chart depicts the cause mechanisms in stress hyperglycemia

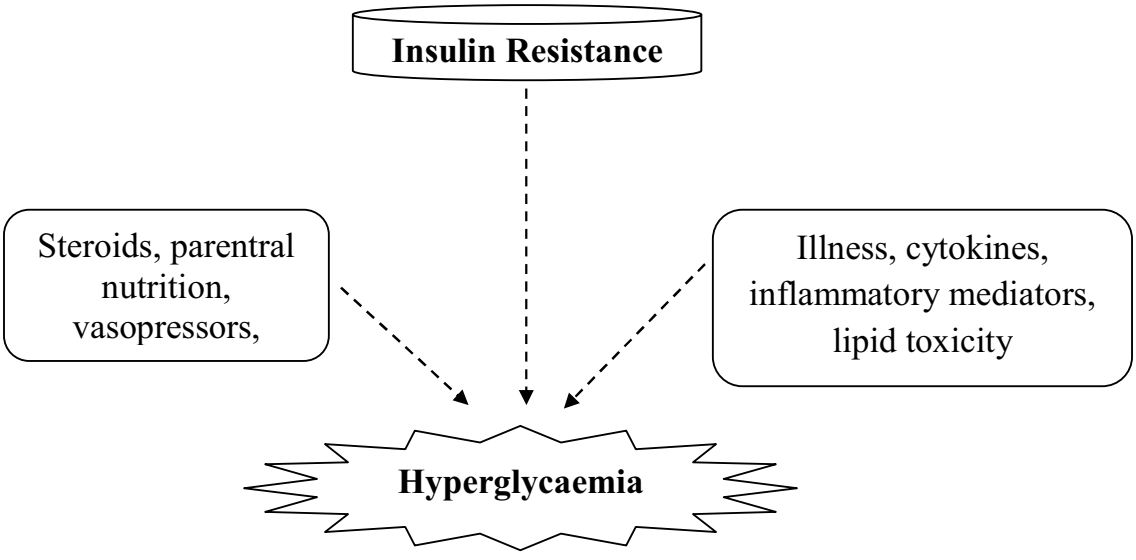


Figure - 4

GLUCOSE METABOLISM IN STRESS HYPERGLYCEMIA

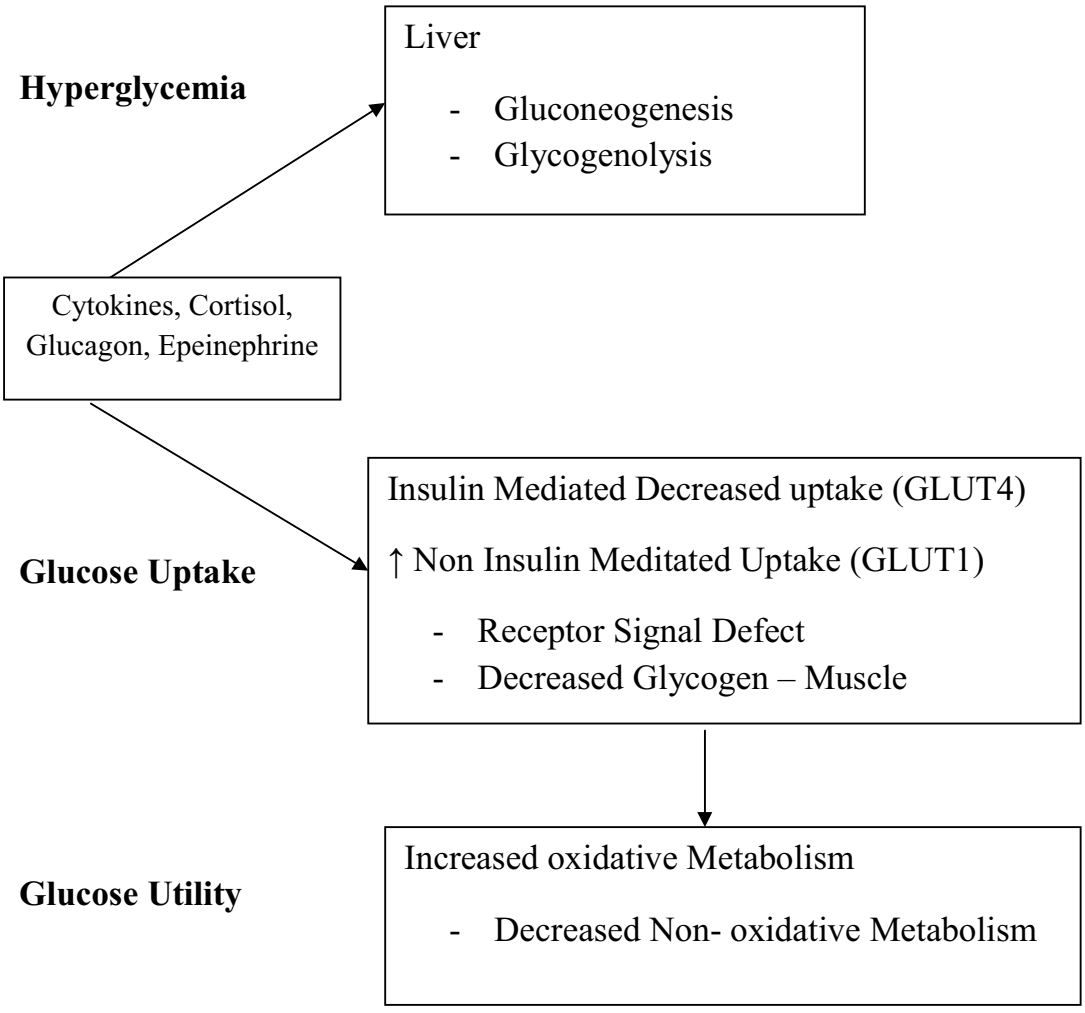


Figure – 5

3.4.3 BURNS AND HYPERGLYCEMIA :-

The post burns injury, hyperglycemic response occurs. It results in alterations in body metabolism. It causes energy expenditure, increased protein break down, and alteration in fat metabolism . There is relationship between insulin resistance and fat metabolism post burn trauma. Reduced response to insulin action is the prime factor , as more datas already show,and liver function derangement results in mortality and morbidity of injured patients. There is also link between first 48 hours hyperglycemia and mortality. Higher the glucose more is the graft loss. Fat metabolism is also altered post burn injury, and this may be related to the resistance to insulin which occurs following post burn injury.

Burn patients also have increase in lipid lysis, inadequate increases in the fat metabolism disproportionate to energy needs, and triglycerides deposits in distant sites, in other tissues , which includes skeletal muscle and liver.[42, 43,] Studies show there is different metabolic system following a burn injury .[45] The first phase which occurs after 48 hours of injury is called the “ebb phase” [45,46] This part of the phase appears to be influenced by the type of the trauma, release of hormones, which includes catecholamines, dopamine, glucagon, and cortisol.[47,48,49] In the acute stage of hyperglycemia insulin release is suppressed by epinephrine.

The amount of lipid release is variable after trauma. It may be increased, not changed, and decreased. The rise in lipids depends upon the nature of adipose tissue before and after injury. The cycling of free fatty acids is increased. This is due to catecholamines acting on hormone sensitive lipase to produce lipolysis and also raised lactate concentrations .

The second phase of burn injury after several weeks of injury, is called the "flow phase.[50, 51.] Plasma sugar comes down initially, but is on the higher range, and insulin level risesover period of time marking the state of insulin resistance. Though hormones play a role in insulin resistance, hormone levels decrease after the initial injury.[52,53]

Skeletal muscle is the major uptaker of 70–80% of whole body glucose by insulin. In burns decrease in muscle mass following burn injury could cause insulin resistance during flow phase . Lipid lysis is also increased. Increased lipid lysis is due to increase adrenergic activity. It is also related to subcutaneous fat. When more fat is lost increased lipolysis occurs. Though lipid lysis is elevated after burns, the concentration of lipids is fluctuating. Glycerol concentrations are elevated in burns.it signifies lipid lysis. Once released from adipocyte it cannot be reutilized for triglyceride synthesis. It is used as a measure of lipolysis post burn trauma.

Lipids are hydrophobic and bind to proteins in plasma. In burns patients there is reduced protein levels, this alters the ratio of binding of lipids to proteins in burns patients compared with healthy controls[54]. Intracelullar turnover of FFA depends on the breakdown of muscle and adipose tissue triglycerides. This again is reesterified into VLDL and incorporated into adipose tissue TG. In burns patients for each turnover, one glycerol and three FFA are released. The rate of release does not meet the bodies energy demands and in excess so reesterified as VLDL-TG.

In healthy adults, 65% of VLDL-TG is reesterified and released after four days of hyperglycemia. Thus the free fatty acid cycle is related to increased sugar after trauma yet the link not clear. The following flow chart depicts the events post trauma.

The following flow chart depicts the events post trauma – catecholamines and cytokines are increased which leads to more delivery of free fatty acid.

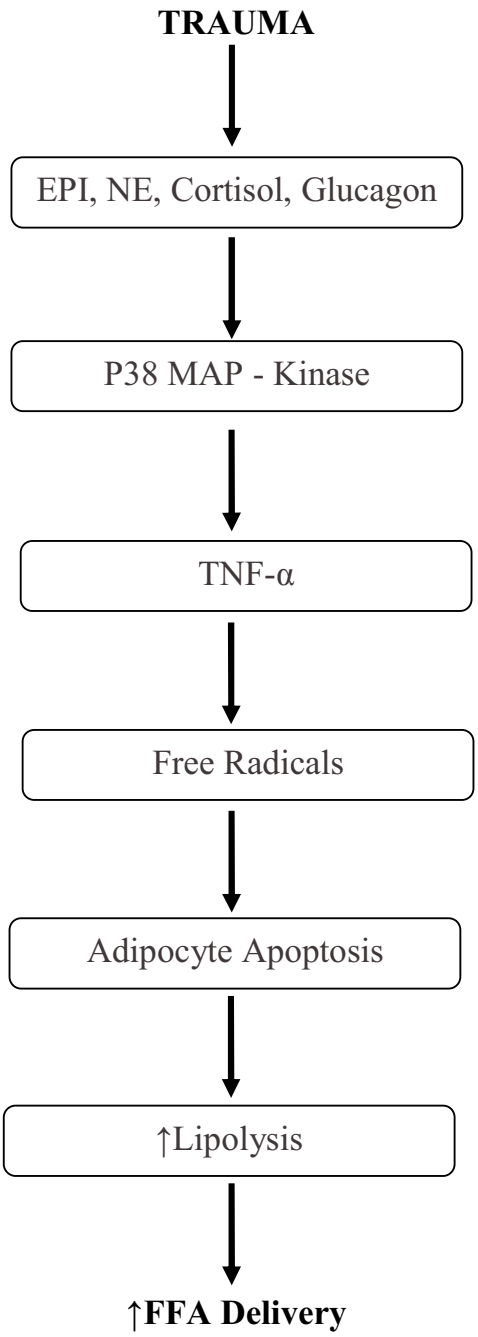


Figure - 6

3.5 INTERACTIONS BETWEEN GLUCOSE AND FAT METABOLISM

The metabolism of glucose occurs in liver and muscle tissue. Liver and muscle are also involved in the metabolism of fat. This overlapping is to be understood. The interaction in these tissues between glucose and fat helps to understand the physiology in resistance to insulin actions. Insulin resistance depends more on fatty tissue. The liver and muscle metabolism is as follows

3.5.1 HEPATIC METABOLISM:-

Liver is organ of metabolism .It plays a major role in glucose metabolism. It produces glucose in fasting state by gluconeogenesis and glycogenolysis. After eating promotes glycogen synthesis .Insulin resistance occurs when it fails to reduce blood sugar and lipid lysis. Raised sugar post injury is due to more glucose synthesis and glycogen break down that occur in the liver, which in turn occurs due to release in catecholamines via various methods[55] This occurs with stimulation of cAMP which in turn stimulate pyruvate kinase which inhibits glycolysis and thereby increase glucose production.

Catecholamines activate p38 a mitogen-activated protein kinase, causing the release of tumour necrosis factor-α (TNF-α) and interleukin-1

(IL-1), and stimulate nuclear factor-κB (NF-κB) via IKK [56] Due to the catecholamines, saturated plasma free fatty acid is elevated, which activates NF-κB via toll-like receptor-4 [57.] NF-κB can cause cell damage and liver tissue damage severe injury which is documented. NF-κB produce IL-6, which can cause increased liver glucose and decreased effect of insulin .[58,59] There are multiple factors involved in increasing the hepatic metabolic rate. During second phase of injury, more glucose is produced. There is more protein break down, so amino acids are used for gluconeogenesis. This hyperglycemia is not suppressed by insulin.

The hepatic glucose output also is not suppressed. This increased glucose during this time is responsible for post burn trauma hyperglycemia. There is also increased delivery of free fatty acids to the liver. This free fatty acid can be reesterified or stored for energy within hepatocytes as triglyceride. Hepatomegaly can occur because of increased free fatty acid and TG storage in liver. Triglyceride in liver increases mortality. It is also responsible for the insulin resistance. Thus both the TG in liver and insulin resistance are the mechanism involved in post trauma hyperglycemia. The liver actions are shown in following chart:

The Liver Metabolism is depicted in the following chart.

LIVER

Gluconeogenesis

Glycogenolysis

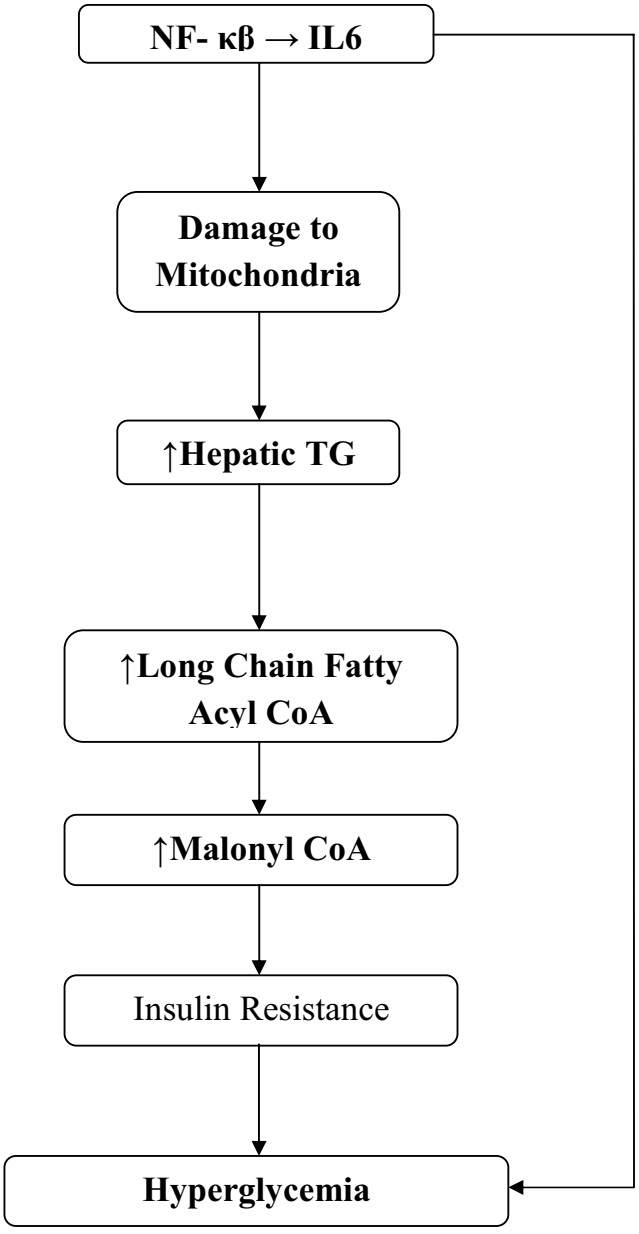


Figure - 7

3.5.2 MYOCYTE METABOLISM:-

Muscle is responsible for resistance to insulin in diabetes mellitus Glucose transportation occurs inside the cell via GLUT 4 transporter. This moves to the surface of cell after binding of insulin to insulin receptor. This intiates signal cascade which involves insulin signal receptor phosphorylation by tyrosine, association of phosphatidylinositol kinase 3 with IRS-1, and mobilization of GLUT-4 stores. In diabetes this phosphorylation is decreased. The same mechanism occurs following post burn. The comparison is shown in Table 1 & 2.

During starvation the phosphorylation by tyrosine is decreased because of no insulin . The resistance to insulin is found to be due to different site of storage of triglycerides within the myocytes. There is increased muscle protein lipase and increased free fatty acid delivery. This increase intramyocellular TG and decrease insulin signalling and deceased glucose uptake. Studies show interactions between intracellular lipids which incudes intramyocellular TG, diacylglycerol (DAG), and fatty acyl-CoA, and insulin sensitivity.[60] The metabolic products of fat are thought to influence the insulin signalling or increase the protein kinase C(PKC) which inactivates IR and IR-1.

The insulin sensitivity is related to fat. Malonyl co A dehydrogenase splits down malonyl co A and oxidates fat . Palmitate oxidation also increases insulin sensitivity. The intracellular lipids accumulation is due to two cardinal factors, the release of fatty acids and β oxidation of fat . Beta oxidation of fat is increased but not able to meet the energy demands. Mitochondria is affected in multiple tissues by burns. IN Mitochondria oxidative capacity of pyruvate and palmitate is reduced. mitochondrial gene expression is also reduced. The mitochondrial dysfunction causes skeletal muscle apoptosis. The reason for mitochondrial damage is not clear in burns. It partly occurs secondary to reactive oxygen species (ROS).

After burns cardiac muscle has large influx of calcium due to mitochondrial nitric oxide synthase. The nitric oxide damage the mitochondrial membrane by blocking the cytochrome c activity. The calcium flux also increases phospholipase A2. This in turn produce free fatty acids that alters the membrane of mitochondria and activity of cytochrome c. Free fatty acids also increase reactive oxygen species. Thus increased fatty acids, along with dysfunction of mitochondria, result in more lipid deposits intracellularly which in turn reduces the action of insulin

The Muscle Metabolism is depicted in the following chart.

MUSCLE

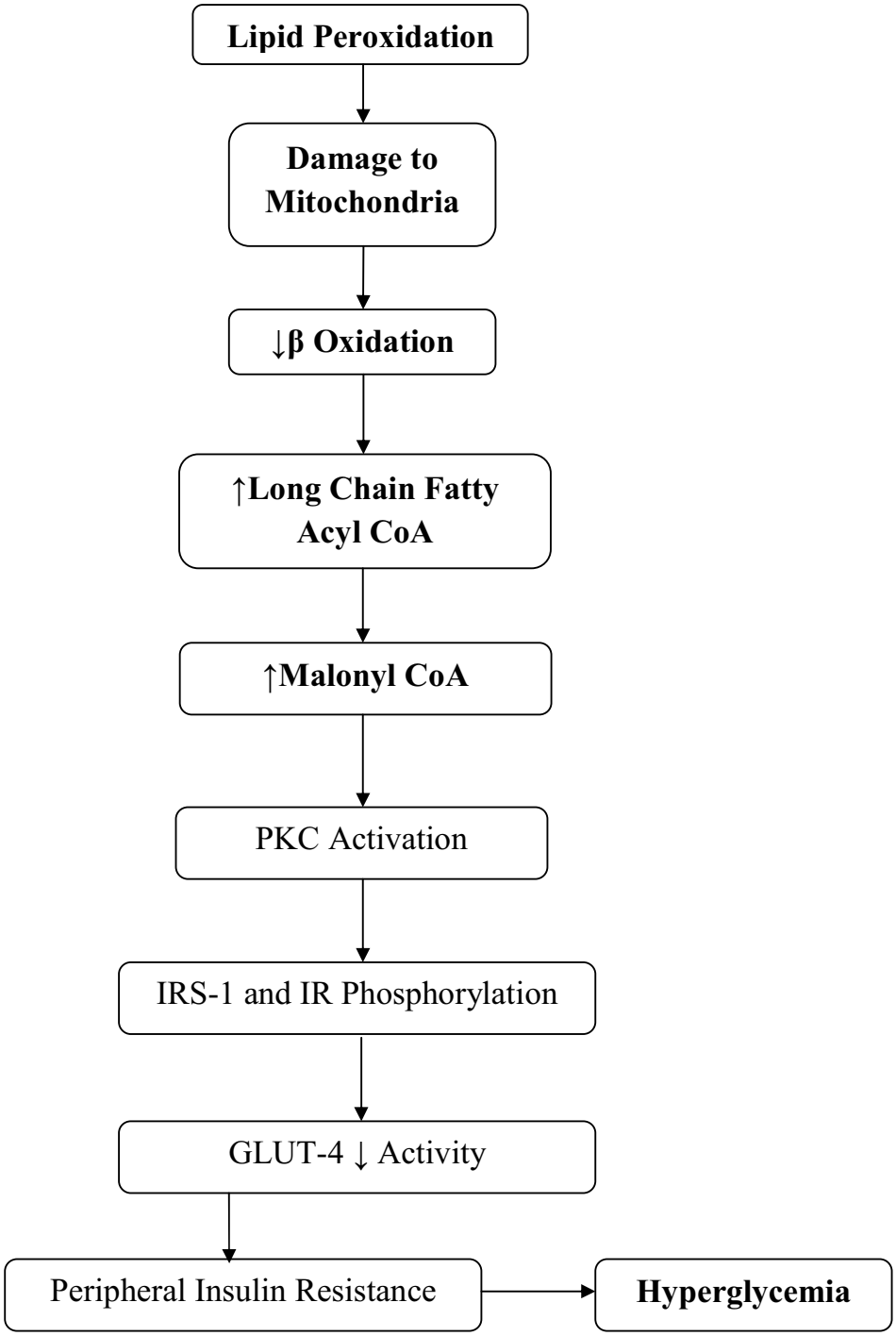


Figure - 8

Table - 1

Comparison of Cell Signalling in Diabetes and Stress

Cell Signal	**Diabetes Mellitus**	**Post Stress**
Insulin receptor phosphorylation	reduced	reduced
IRS-1 tyrosine phosphorylation	reduced	reduced
AKT/PkB association with IRS-1	reduced	reduced
PKC Phosphorylation	raised	raised

The muscle cell signalling is same in both acute and chronic stress

Table - 2

Lipid levels in muscle in diabetes mellitus Vs Post Stress

Lipids	**Diabetes Mellitus**	**Post Stress**
Triglyceride	raised	raised
Diacylglyceride	raised	no change
Long chain fatty acyl-CoA	raised	nochange

The intracellular lipid concentrations are not the same in acute and chronic stress

3.6 MECHANISM OF ADVERSE OUTCOMES:

Stress hyperglycaemia which is acute, results in metabolic and hormonal dysregulation , than that occurs in chronic hyperglycaemic state, of diabetes mellitus. It is this dysfunction which needs treatment. For example, in the failure of all organs due to microvascular endothelial abnormality , intensive insulin therapy is needed to protect endothelium.[61] Chronic hyperglycemia also sets a cellular preconditioning which is protective in acute hyperglycemia due to stress. This occurs by downregulation of glucose transporters in chronic hyperglycemia .GLUT-1 and GLUT-3 are glucose transporters independent of insulin. They are upregulated in acute illness and so glucotoxicity occurs. Various oxidative stressors are also involved in upregulation of GLUT-1 in endothelial cells.

The acute variations in glucose concentration during period of stress is responsible for mortality than the mean glucose concentration. These fluctuations cause increased endothelial apoptosis, endothelial dysfunction, and oxidative stress. It is the oxidative injury in acute hyperglycaemia compared to chronic hyperglycemia that is responsible for the mechanism of adverse effects of stress hyperglycaemia. Also hypoglycaemia can increase the adverse effects. Hypoglycaemia per se can increase inflammation which is already more in acute state.

The following diagram shows the overall effect of hyperglycemia

EFFECTS OF HYPERGLYCEMIA

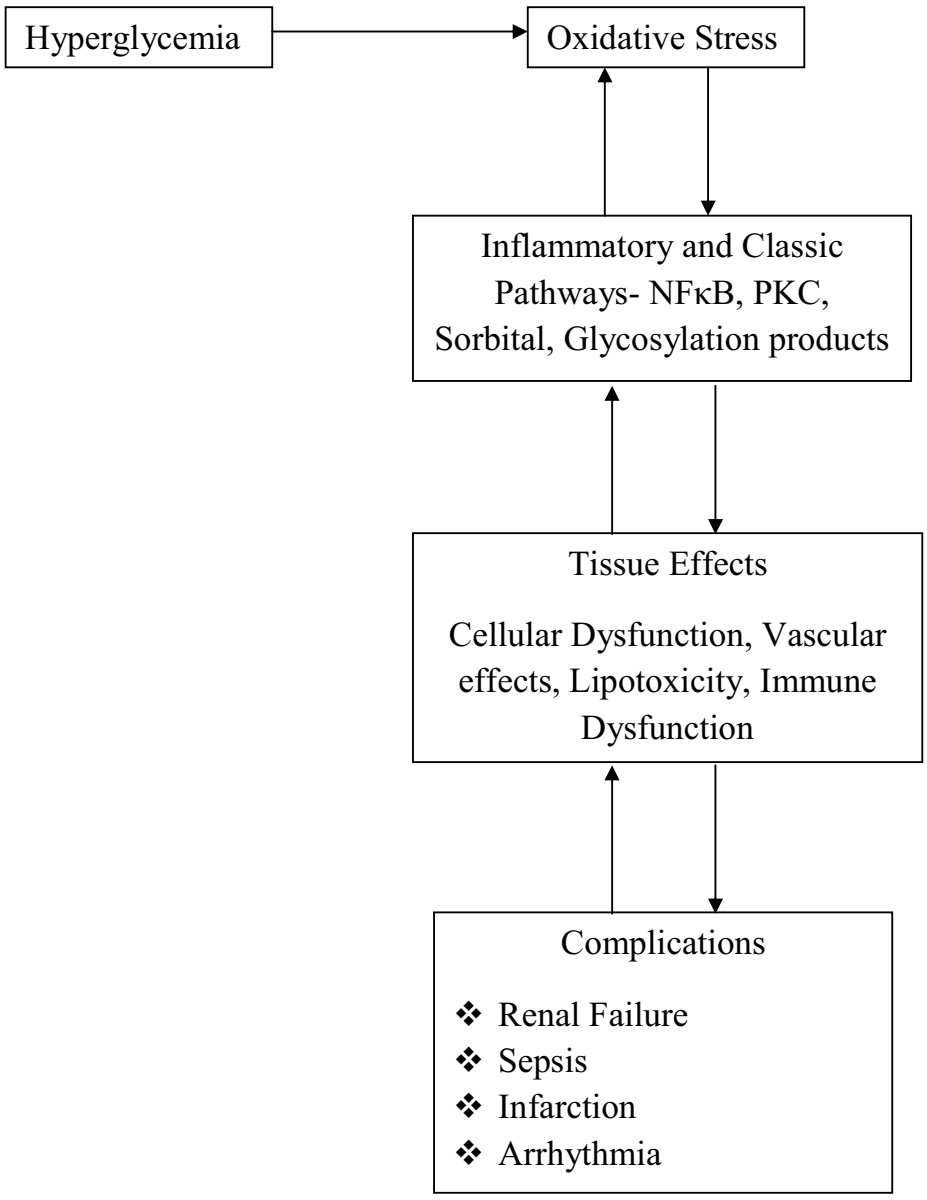


Figure - 9

3.6.1 HYPERGLYCEMIA AND CARDIAC SYSTEM:-

Immediate hyperglycemia produces multiple effects on the heart. It affects the preconditioning of the hypoperfused heart, which is protective for the decreased blood flow insult[62.] Additionally , the area of infarction is increased with hyperglycemia. There is also decreased cardiac blood flow . Cardiac myocyte death occurs through cell death or by exaggerating ischemic blood flow cellular injury[63,64.] The other effects of immediate hyperglycemia includes, changes in the blood pressure, elevation in catecholamine, abnormalities in the platelets, and electrophysiologic changes. Marfella et al, induced sudden rise in blood sugar (270 mg/dl or 15 mmol/l) over 2 h in healthy males and found that it elevated blood pressure, pulse rate , rise in catecholamine levels, and prolongation of QTc[65]. Some more studies have also shown an association between sudden rise in sugar and raised viscosity,hypertension [66,] and ANP levels. There is also increased mortality with hyperglycemia seen in myocardial infarction patients.

3.6.2 HYPERGLYCEMIA AND THROMBOSIS:-

More analysis have shown link between hyperglycemia and abnormalities in thrombosis. Hyperglycemia induces reduced fibrin lysis and plasminogen of tissue activity. Inhibition of plasminogen is increased.Thrombaxane biosynthesis is increased, with reduction in blood

sugar thrombaxane synthesis is decreased. Hyperglycemia causes increase of (IL)-6 levels elevates fibrinogen of plasma and fibrinogen mRNA.[67] In diabetes patients the activity of platelets is increased. which is seen as the attachment of platelets , on the extracellular matrix .[68] In a analytical study, 12 patients were subjected to hyperglycemic (250 mg/dl) and euglycemic (100 mg/dl) status.

Raised sugar causes activation of platelet and the expression of platelet P-selectin and lysosomal integral membrane protein (LIMP). Raised sugar also increased the plasma clotting von willebrand factor and urinary 11-dehydro-thromboxane B_2 (corresponds to thromboxane A_2 production). Howevere such effects not observed in normoglycemic patients. Thus increased platelet activity can occurs in stressful conditions.

3.6.3 HYPERGLYCEMIA AND INFLAMMATION:-

Vascular changes occur due to the involvement of inflammatory changes. Other studies suggest that increased IL-6, TNF-α, due to raised sugar produce acute inflammation. Antibody against TNF alpha will control the stimulation of IL-6 by glucose.. The inflammatory response is seen in adipose tissue, vascular smooth muscles and other tissue cell types. Hyperglycemia causes increase in IL-6, IL-18, TNF alpha. TNF-α causes enlargement of the necrotic area in left anterior desending artery

occlusion. In humans, the more rise in TNF-α levels more is the severity of cardiac dysfunction. It causes some injury to kidney due to hypoperfusion and to heart also leading to failure. The preconditioned staus however reduces the post ischaemic myocardial TNF-α production. IL-18 causes disruption of atherosclerotic plaques, which causes acute ischemic syndromes. Increase of these factors cause adverse vascular effects .The most common relation between inflammation and raised sugar level is due to pro inflammatory transcriptional factor, nuclear factor (NF)-κB by exposure to different cell types during 1–8 days of hyperglycemia[69,70]. Nuclear factor (NF)-Kb has its own adverse effects.

3.6.4 HYPERGLYCEMIA AND ENDOTHELIAL CELL DYSFUNCTION:-

Hyperglycemia and poor cardiac outcomes are due to effect of raised sugar levels on blood vessel endothelium. It plays a critical role in homeostasis.The bolod vessel lining maintains the blood vessel in a relaxed , thrombolytic, antioxidant, and anti aggregating . During stress, the vessel endothelium undergoes dysfunction, nonregulation, and failure. There is increased cell to cell attachment , new vessel cell formation , becomes more permeable , inflammatory and failure. Human in vivo studies using this data confirm that sudden rise in sugar seen in the hospital causes endothelial dysfunction [71,72] Hyperglycemia directly alters the

function of endothelium by inhibiting nitric oxide . This is again has been linked to increase in the cell to cell adhesion, disturbed new vessel formation, increased permeability of the cell, inflammatory changes and pro thrombotic state. The function of endothelium is evaluated by measuring vasodilatation effects , on atrey like brachial artery. Hyperglycemia also triggers the output of reactive oxygen species (ROS) or activates other mechanisms.

3.6.5 HYPERGLYCEMIA AND THE BRAIN:-

Sudden onset hyperglycemia seems to be linked with increased neuronal damage due to brain hypoperfusion.. The major portion of the brain that is more prone to injury from raised sugar is the ischemic penumbra. It surrounds the ischemic core. During stroke in progression the penumbra becomes areas of infracted brain tissue or recover as non infracted tissue. The elevated glucose concentrations is associated with increased lactate and tissue acidosis. Lactate has been seen to cause damage to the neurons, astrocytes, and endothelial cells of the brain.[73-75.] Lactate-choline ratio is seen, which predicts the outcome and also infarct size. More recently, some investigators demonstrated a positive link between hyperglycemia and production of lactate [76.] Through this mechanism, hyper glucose state causes infarction of brain due to ischemia . Hyperglycemia

also causes accumulation in the neocortex with glutamate. Increased glutamate levels are perdictors of ensuing damage that occurs to neurons.

It is also seen from studies, that hyperglycemia causes break down of DNA, blood-brain barrier alteration, rapid change of polarization in penumbral tissue which is severely hypoperfused, β-amyloid precursor protein elevation, and increase in superoxide levels in the neuronal tissue.[77-80] Hydroxyl free radicals are increased in hyperglycemia. They cause tissue damage. Elevated glucose levels also cause decreased nitric oxide, increase IL-6, m RNA, and decreased cerebral flow. Thus studies show the link between hyperglycemia and neuronal injury.

3.6.6 HYPERGLYCEMIA AND OXIDATIVE STRESS:-

Hyperglycemia also causes oxidative injury in the form, by generating more reactive oxygen species (ROS). The oxidative stress occurs when there is imbalance between production of ROS and its metabolism . Endothelial injury when occurs, produces super oxide anion than nitric oxide.This in turn causes production of secondary mediators , growth factors, transcriptional factors. Thus from direct tissue injury or hyperglycemia induced secondary factors oxidative stress cause tissue injury and death.

3.6.7 HYPERGLYCEMIA AND IMMUNITY:-

There is relationship between raised glucose levels and infective state. The major reason identified is defective phagocyte function . There has been funtional alterations in monocyte and neutrophil, including the adhering and chemotactic mechanism, phagocytic, bacterial killing, and respiratory chain . Granulocyte function improves with reduction in blood sugar .The adherence of granulocyte is improved.

There has been elevated levels of cytosolic calcium. Increased cellular calcium cause reduction in ATP level and defective phagocytic response.. There exists a link among polymorpho nuclear cellular calcium and fasting sugar level. They are inversely related to the phagocytosis of cells. Decrease in the glucose levels results in decreased intracellular calcium, increased energy content, and effective phagocytosis.

The microvascular side veffects of diabetes is due to changes in metabolic pathway. Most of these pathways contribute to immune dysfunction. Protein kinase c pathway mediates the neutrophil dysfunctionas a result of hyperglycemia . It is seen reduced super oxide formation and decreased activity of phospholipase D occur with hyperglycemia . Impaired antioxidant formation has link with leukocyte dysfunction. In neutrophils also reduced ROS occurs due to hyperglycemia .Hyperglycemia

also inhibits glucose 6 phosphate dehydrgenase. Aldolase reductase pathway is found to cause infections and there is a decrease in infections with aldolase reductase inhibitor. In acute hyperglycemia reduction in lymphocyte function is also observed. Tcell subsets CD4 and CD8 are also decreased.

Finally studies on the impact of hyperglycemia on the immune system constantly show that high glucose content causes immune suppression and immune dysfunction. Control of glucose or reduction of glucose improves the immune function.

3.7 MANAGEMENT:-

Currently there is no separate management for stress hyperglycemia and diabetes. However there may be variations in treating ICU and non ICU patients. Treatment modality are tried after results of Nice-Sugar study It says that glucose control should not be withheld in intensive care unit, but less intensive target should be achieved.[81,82.]

In the setting of non intensive care patients no data exist to guide the treating plan , but individual treatment according to glucose levels can be done.[83,84] There are no studies that have specially studied the best treatment for the management of stress induced hyperglycemia. It is reasonable to follow methods, for treating admitted patients with

hyperglycaemia, since acute hyperglycaemia is a short duration event, and a dynamic disorder which responds to changes in the disease course. The accepted method for control of sugar is insulin therapy, but in the surgical and medical ICU insulin infusions are preferred for treatment. There is stacking of insulin and hypoglycemia in edematous patients or hypo perfusion with subcutaneous insulin [85].

Subcutaneous dosing of insulin is ideal for most medical and surgical patients who are treated outside intensive care unit . in the outpatient treatment , analogues of insulin which produce less hypoglycaemia than do short and long acting human insulin were preferred, but such evidence was not found in any study.[86.] Results of other studies also show that subcutaneous basal bolus insulin was effective than the titrating units of insulin used for attaining a safe, effective sugar control. Patients treated with insulin infusion in intensive care unit later changed to subcutaneous insulin had better outcomes. The dose of insulin can be adjusted daily, but requires couple of days to reach control of sugar. Dose adjustment should be according to diet intake. .Diet management should also be done in hyperglycaemic patients. This strategy includes giving consistent foods rich in carbohydrate , or treating with insulin after food according to estimated levels of carbohydrate taken by patient during enteral feeding, close monitoring is necessary. Insulin can be withheld if it is necessary.

The quantity of glucose given by fluids or other modes is mostly not noticed. They may be restricted when necessary. There is no reliable evidence that tells reduction of glucose fluctuations, gives better outcomes. However, changes in insulin replacement can be done and also to keep adequate carbohydrate intake[87,88].

Patients with good sugar control require reduction of insulin accordingly. Experts don't practice with the oral drugs for most hospital inpatients because of its delayed onset of action and effects and the common side effect of hypoglycaemia in patients whose food intake is variable.. The adverse effects of drugs is also contraindication.

Multiple studies suggest the treatment of glucose insulin potassium (GIK) infusions for cardiac and neurological benefits [89,90]. Control of sugar is not the aim of such infusions. Though insulin has a important role directly in the infusion effects , other confounding factors also has effects in this therapy. The basis behind such intervention is mismatch between decreased glycolysis product in infracted tissue and increased fatty acids levels due to lipid lysis [91]. In hypoperfused heart ,ATP is reduced and elevated inorganic phosphate levels seen.. ATP is important because it maintains the cell membranes, myocardium and its contractility, and

protects from the adverse recations of fatty acids, which is found in underperfused myocardium[92,93,94]. Free fatty acids cause increased heart activity,more heart damage, and conduction disturbances in heart. Accordingly, studies in hearts of the rat , have demonstrated the usefulness of glucose insulin potassium therapy, in the glycolysis, ATP levels , and the levels of phosphate which is inorganic in the damaged tissue [95].

In other animal experiments also this treatment improved heart function and metabolic effects. As a treatment for patients in acute stress, this infusion is associated with decrease in all adverse effects and good improvement in survival. There is less data regarding usefulness of this therapy in strokes or cerebral ischemia only few studies have shown safety of GIK therapy in the acute stroke. Besides GIK therapy, insulin has its effect on complications. Insulin is helpful for endothelial cell function. Insulin is also vasodilator in arteries. The vasodilatory properties of insulin is due to nitric oxide. Endothelial cell cultures of arteries have demonstrated there is nitric oxide synthase action and raise in levels of nitric oxide levels. P-selection is expressed in high glucose levels. Insulin therapy also has inflammation reduction effects. It decreases C-reactive protein (CRP) levels in patients.

Microbiological studies have shown that insulin can reduce the free radical stress and cell death in myocardium. Insulin also reduces the ROS, transcription factor NF-κB, intercellular adhesion molecule (ICAM)-1, and the chemokine monocyte chemoattractant protein (MCP). It inhibits the production TNF alpha. Insulin also improves lung and heart function . There is evidence that insulin is helpful in protecting from the CNS, renal, and lungs ischemia . In severe burns, which produce catabolic state raised sugar produces more muscle catabolism, while insulin has a anabolic effect . Insulin treatment also acts as fibrinolytic therapy. Insulin infusion decreases the collagen-induced platelet aggregation. Thus in summary it is seen that insulin is highly beneficial in hospital hyperglycemia either directly or through its effects on lipolysis, glycolysis, etc.

3.8 LINK BETWEEN METABOLIC STRESS, INSULIN, HYPERGLYCEMIA AND OUTCOME:

Thus in summary as already shown by studies, increase in counterregularory hormones increase catabolic state, liver gluconeogenesis, and lipolysis. These in turn lead to increased sugar, free fatty acids, ketones, and lactate. Insulin action is blunted due to glucotoxicity which leads to further rise in sugar. The low insulin and raised sugar affects, the immune system and function, metabolic needs of the body,and produces

products which lead to more tissue damage and organ derangements . Thus the combination of high sugar and low insulin is responsible for adverse effects in stress induced hyperglycemia.

3.9 PREVENTION:

It is genrally not predictable or preventable to identify hospital related hyperglycemia. However, early suspicion and intervention is useful to prevent stress hyperglycemia . From studies it is found that preoperative glucotoxicity could also affect the treatment of postoperative control for sugar. Even intraoperative sugar control is very dangerous for certain procedures. The effectiveness of control of sugar depends on the accurate glucose monitoring.

In an ICU set up there are various confounding factors which could make the measurement inaccurate like anemia, hypotension, because they cause capillary readings inaccurate. It is highly challenging to measure glucose in increased severity of illness. Constant checking of sugar will help to control sugar levels.Measurement errors can occur. Oral glucose tolerance test or close check of sugar in hyperglycaemic patients is necessary. It is ideal to follow up patients after discharge to see for underlying diabetes and to manage appropriately.

3.9.1 FUTURE PERSPECTIVES:

Studies for comparing the outcome is whether due to diabetes or hyperglycaemia due to stres are needed. HbA1c to be checked to differentiate between long term diabetes or stress hyperglycemia, and to diagnose stress exacerbation in diabetics..

A comparative study between normal patients having hyperglycemia and with diabetics having elevation of sugar due to stress , is needed. The optimum target glucose range which is to be maintained in stress hyperglycaemic conditions is still undefined. Studies also say that various targets and their risk benefit ratio should be analysed.

Till data are available, it is difficult to improve and control sugar with insulin regimens and monitor glucose . It will be helpful for the risk stratification.

Stress hyperglycaemia is thus a heterogeneous entity with unique pathophysiological features and complications. It has its own adverse effects. Present concepts from various studies, tell us that hyperglycaemia should be treated irrespective of its cause.

However, from studies it is seen the duration of hyperglycaemia and comorbid factors, which varies with each patients or populations seeks more attention . Thus stress hyperglycemia needs to be identified early and treated, and follow up and monitoring of the patients will provide more data regarding the stress hyperglycemia and its effects. Thus this study is done to see stress hyperglycemia in burns patients and its outcome.

4. MATERIALS AND METHODS

4.1 Inclusion criteria:-

A total of 150 patients who were admitted in the burns department in kilpauk medical colllege were taken up for present study. Patients with moderate degree burns (15-30%) were selected for study. Patients were seen random blood sugar, three days fasting sugar and postprandial sugar and HbA1c. Patients were diagnosed as DM as per american diabetes association criteria.

ADA criteria is as follows:

a) Fasting plasma glucose>=126mg/dl

b) 2 Hour plasma glucose>=200mg/dl

c) Random blood sugar >=200mg/dl

d) HbA1c>=6.5%

4.2 Exclusion criteria:-

Since it is study of stress hyperglycemia, patients already known diabetics were excluded from study. Severe burns patients were also excluded since mortality was high. Females with history of gestational diabetes were also excluded. Patients age < 18 years and other co morbid conditions, and taking steroids were excluded.

4.3 METHODOLOGY:

Patients with random sugar more than 200mg/dl after admission were selected and 3 days fasting and PPBS was taken, if sugar was high, HbA1c was done to rule out diabetes from stress hyperglycemia. HbA1c was done by biorad method. If patient had diabetes, HbA1c would be higher than 6.5 (according to ADA criteria) and if it was less than 6.5 then patient is in stress hyperglcemia.HbA1c reflects the last three months sugar status so if it is high it could not be due to acute stress now.

4.4 STATISTICAL ANALYSIS:

The statistical analysis used in the study are chi-square test, for discrete variables, paired T test for continuous variables, ANOVA (analysis of variance) for more than two groups. All the tests were performed using SPSS (Statistical package for social sciences) version 17.

5. RESULTS AND OBSERVATIONS

General characteristics: In the present study a total of 150 patients were selected according to inclusion criteria, from the 700-800 patients admitted in the burns department with moderate degree burns in one year. Diabetes was ruled out from them by seeing HbA1c, and observations made subsequently. The total number of male and female in the study were 72 and 78. Among them newly detected diabetes were 17 in the study. Since it is study about stress hyperglycemia the diabetes were ruled out and the remaining 133 patients were taken to study. The total number of male and female with stress hyperglycemia were female 67 and male 66. Among them people were grouped into three age groups, those who are < 30 years, 30-45 years and more than 45 years. Their duration in hospital was divided into lesser duration of 15 days and more than 15 days.

The following observations were made subsequently:

5.1 SEX AND AGE GROUP:-

The number of male and female patients in each age group were first seen.

SEX	**AGE GROUP**			
	<30 years	**30-45years**	**>45 years**	**Total**
Female	40	21	6	**67**
%	59.7%	31.3%	9.0%	**100.0%**
Male	27	25	14	**66**
%	40.9%	37.9%	21.2%	**100.0%**
Total	**67**	**46**	**20**	**133**

CHART – 1

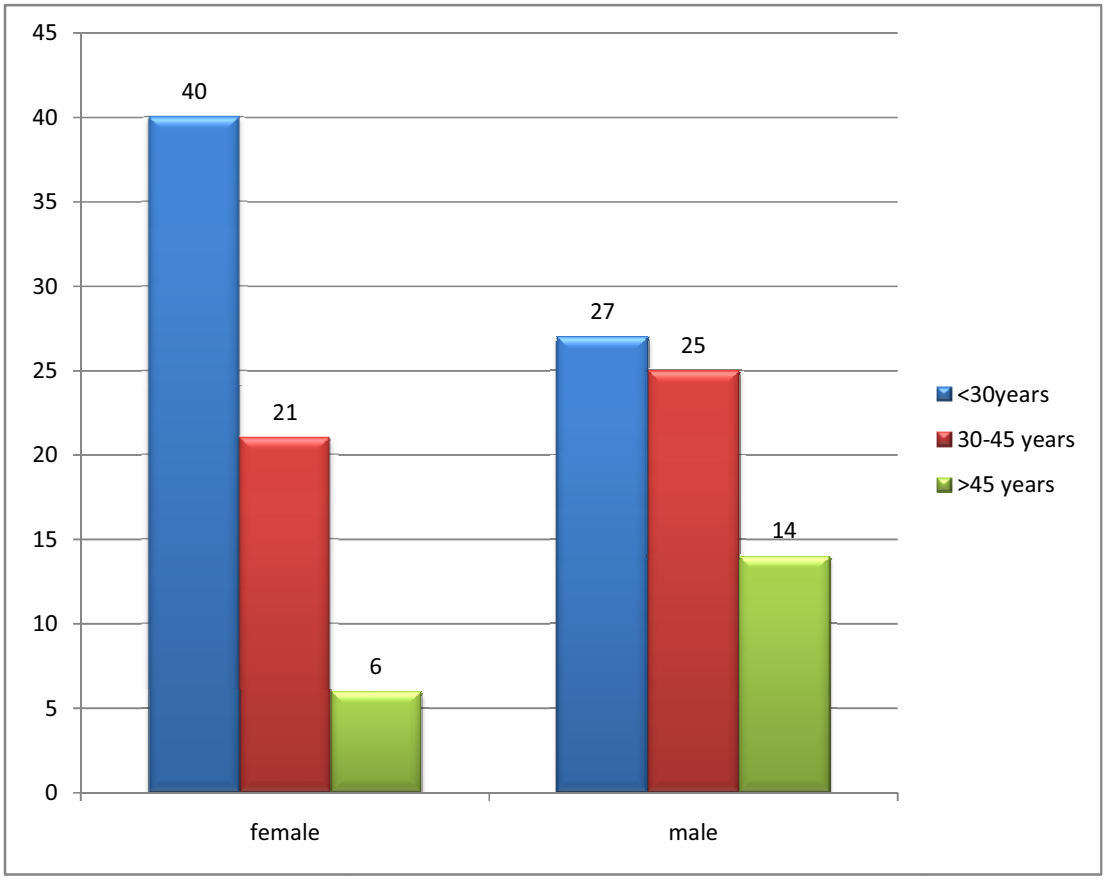


Figure – 10 Comparison between Sex and Age Groups

The study shows more number of patients in < 30 years age group in both males and females, patients in 30-45years of burns were almost equal between males and females. But in older age group men are more than in females. The chi square- 6.063 p value-0.048. There is statistical significance between the sex and age group in stress hyperglycemia.

5.2 SEX AND HOSPITAL DAYS:-

The sex and hospital days observed is as follows.

SEX	Hospital days		
	<15 days	**>15days**	**Total**
Female	23	44	**67**
%	34.3%	65.7%	**100.0%**
Male	12	54	**66**
%	18.2%	81.8%	**100.0%**
Total	**35**	**98**	**133**

In both the sex more number are found in longer duration of stay in >15days. In females 44/67 and in males 54/66 are found in longer duration of stay.

CHART – 2

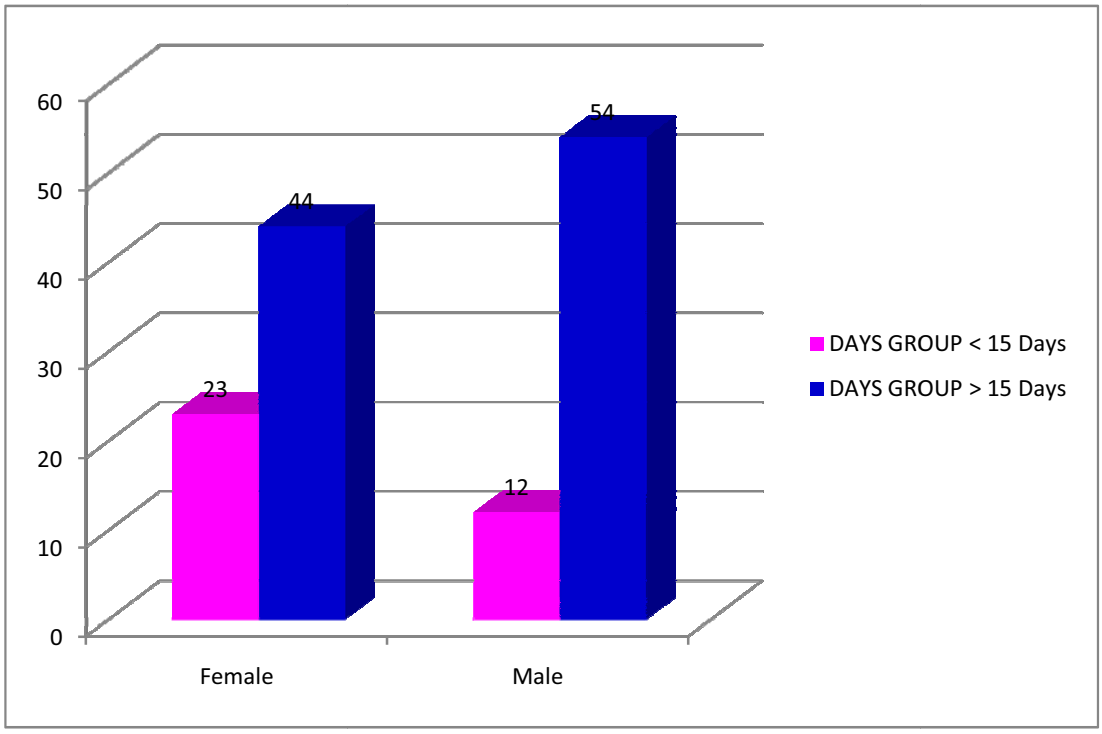


Figure – 11 Comparison between Sex and Hospital Days

The chi square and p value is 4.4070 and p=0.034. There is stastical significance between duration of days and the sex. From the study it is seen longer duration days are seen more in both males and females, but males are 54/67with 81.8%, so males with stress hyperglycemia have longer duration of stay.

5.3 SEX AND STRESS HYPERGLYCEMIA:-

Sex	Stress hyperglycemia		
	<250mg/dl	**>250mg/dl**	**Total**
Female	56	11	**67**
	83.6%	16.4%	**100.0%**
Male	49	17	**66**
	74.2%	25.8%	**100.0%**
Total	**105**	**28**	**133**

From the study it is seen the overall distribution of sex in stress hyerglycemia is 67/133 females and 66/133 males. The sugar range for stress hyperglycemia was grouped into lesser range of <250mg/dl and higher range >250mg/dl.

Chart - 3

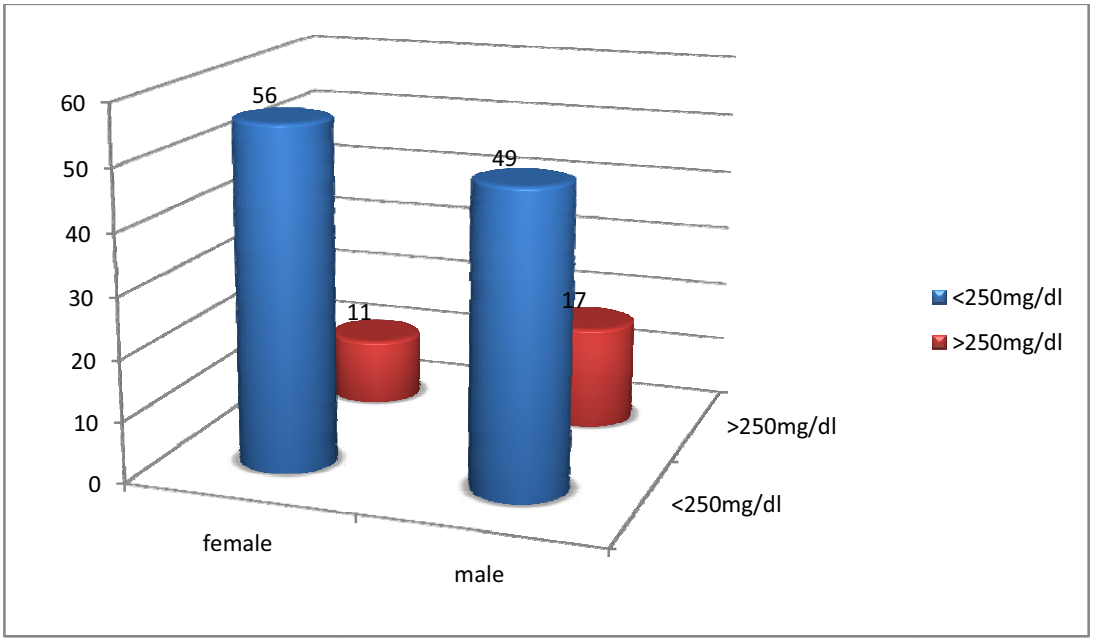


Figure – 12 Comparison between Sex and hyperglycemia

More number of females had sugar less than 250mg/dl which is around 56/67 ie is around 83.6% compared to males who were 49/66 which is around 74.2%.

The chi square and p value was not significant for sex and stress hyperglycemia.

5.4 STRESS HYPERGLYCEMIA AND AGE GROUP:-

STRESS HYPERGLYCEMIA	AGE GROUP			
	<30 years	**30-45 years**	**>45 years**	**Total**
<250mg/dl	53	37	15	105
%	50.5%	35.2%	14.3%	100.0%
>250mg/dl	14	9	5	28
%	50.0%	32.1%	17.9%	100.0%
Total	**67**	**46**	**20**	**133**

Comparison between stress hyperglycemia and age group showed more number in <250mg/dl sugar around 105/133 and around 28/133 in >250mg/dl.

Younger age group <30 years were around 53/105 and had sugar in the range of <250mg/dl.

CHART – 4

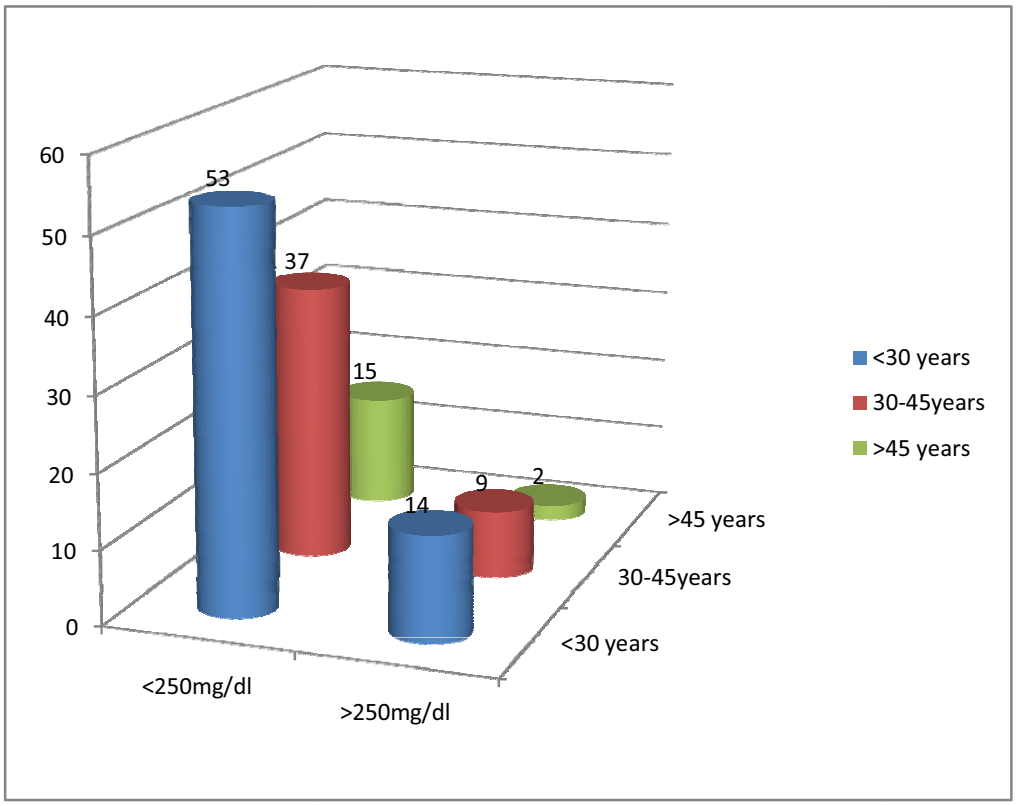


Figure –13 Comparison between Stress Hyperglycemia and Age Groups

The above figure shows that stress hyperglycemia is more in the younger age group around 50.5%. And sugar is also in the range <250mg/dl. The chi square is -0.250 and p value -0.883 which is not statistically significant.

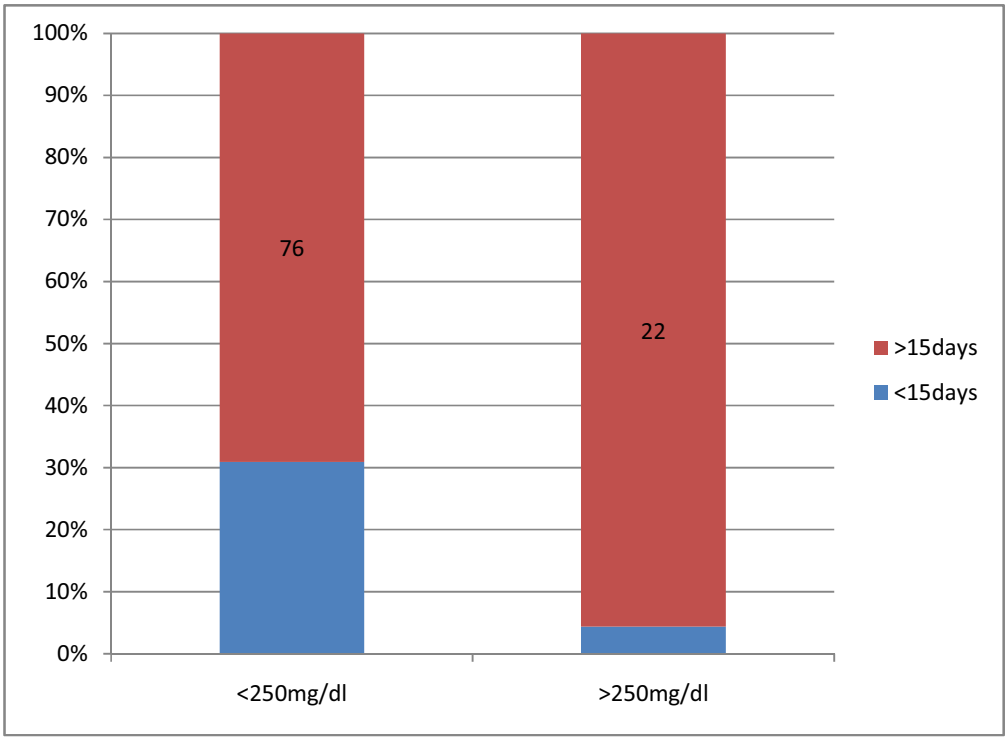
5.5. STRESS HYPERGLYCEMIA AND HOSPITAL DAYS:-

The sugar value was taken has two group those with sugar <250mg and those with sugar >250mg. The sugar value and hospital days observed in the study is as follows:

STRESS HYPERGLYCEMIA GROUP	**Hospital days**		
	< 15 Days	**> 15 Days**	**Total**
< 250 mg / dl	34	76	110
%	30.9%	69.1%	100.0%
> 250 mg / dl	1	22	23
%	4.3%	95.7%	100.0%
Total	**35**	**98**	**133**

Around 110 patients had sugar <250mg/dl and among them 76/110 were found in longer duration of stay. More than 250mg/dl sugar was found in 23 patient and among them 22 had longer duration of stay.

CHART – 5



Figure–14 Comparison between Stress Hyperglycemia and Hospital Days

The total patient with sugar value less than 250 were 110/133 , and among them 76 had longer duration of stay which accounts for 69.1% and more than 250 were totally 23/133 and among them 22 had longer duration of stay which is 95.7%. The chi square - 6.921 and p value - 0.009 which was statistically significant.

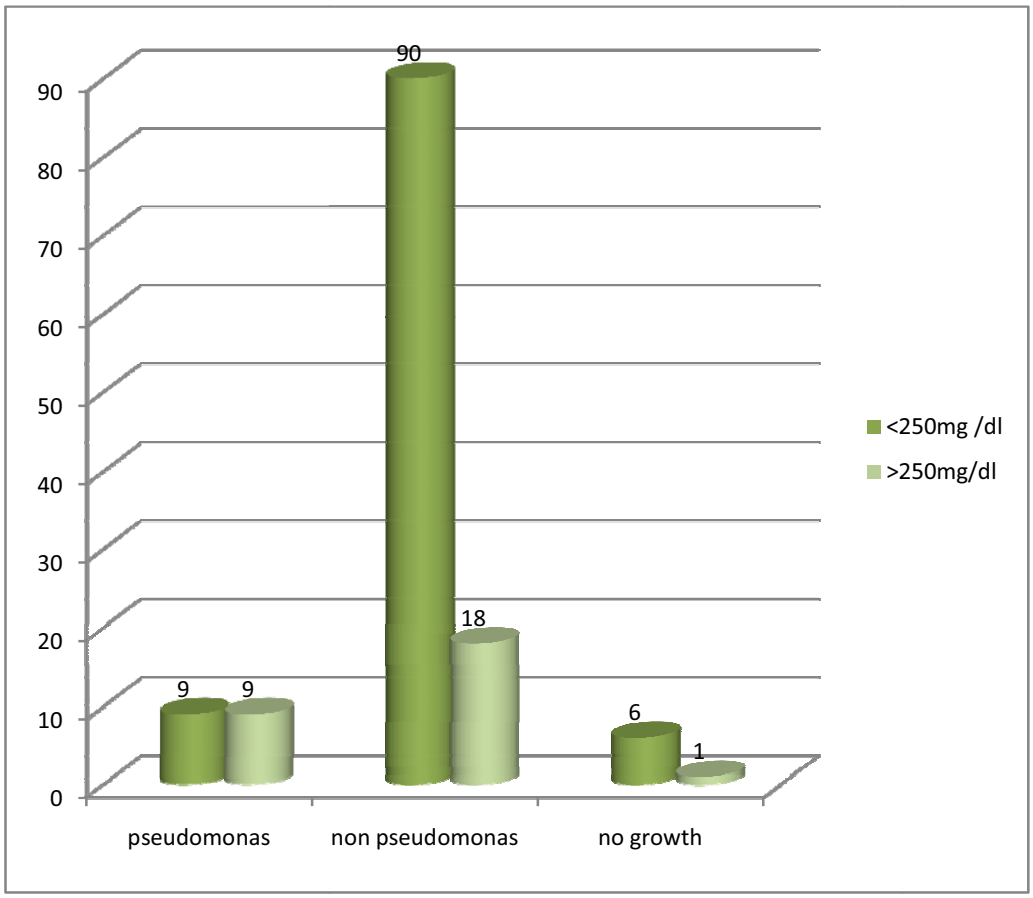
5.6 STRESS HYPERGLYCEMIA AND ORGANISM :-

The organism groups was divided into three. Pseudomonas, non pseudomonas, and no growth. Non pseudomonas organism included staphylococcus, proteus, klebsiella, E.coli. Acinetobacter, coccobacillus, and combined organisms.

ORGANISM GROUP	STRESS HYPERGLYCEMIA		
	< 250 mg/ dl	**> 250 mg/ dl**	**Total**
pseudomonas	9	9	**18**
%	50.0%	50.0%	**100.0%**
Non pseudomonas	90	18	**108**
%	83.3%	16.7%	**100.0%**
No growth	6	1	**7**
%	85.7%	14.3%	**100.0%**
Total	**105**	**28**	**133**

Non pseudomonas organisms were more around 108/133 which is 81.2%. No organism was found in 7/133. It is seen that non pseudomonas organism were also high in lesser sugar range 90/105 with sugar less than 250mg/dl.

CHART – 6



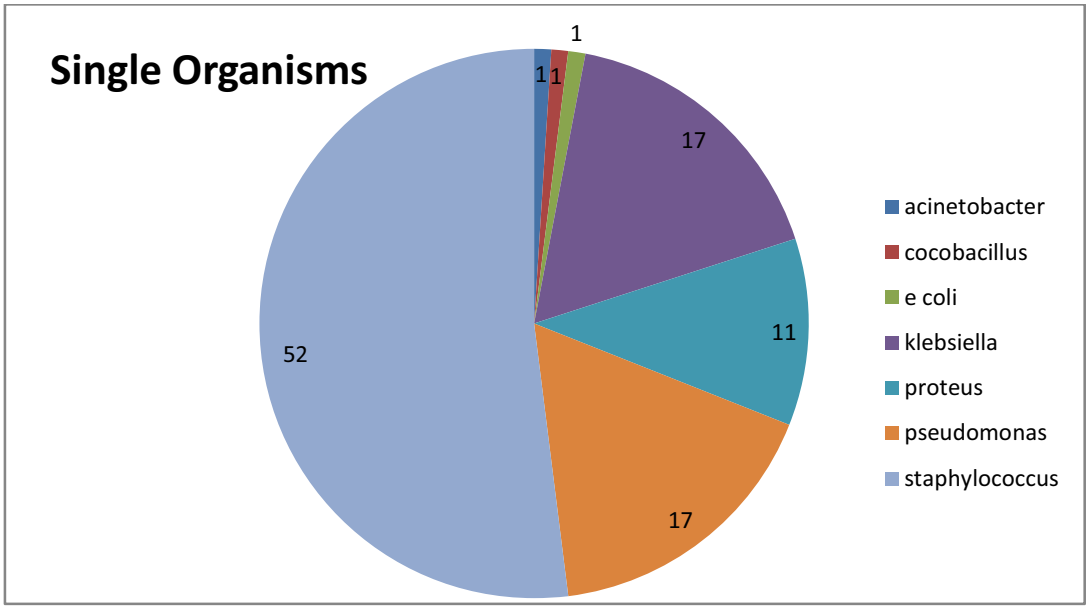
Figure–15 Comparison between Stress Hyperglycemia and Organism

The chi square – 10.518, p- .005 which is statistically significant. Non pseudomonas organism are more common in stress hyperglycemia

5.7 ORGANISMS IN STRESS HYPERGLYCEMIA:-

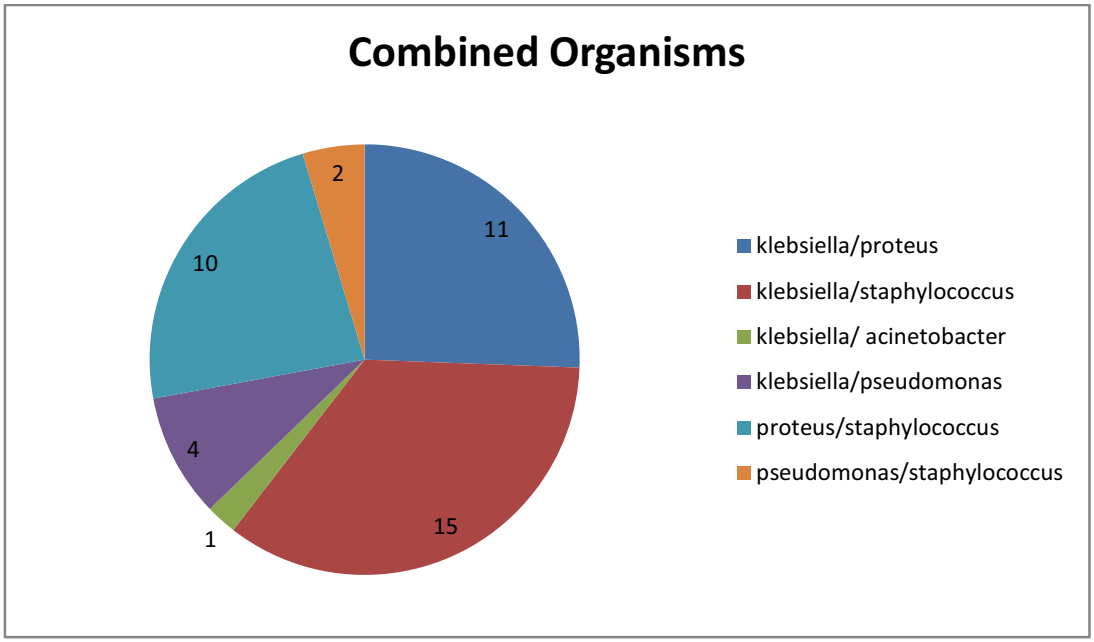
The total number of organism for the whole 150 patients were studied. It was seen there was only one acinetobacter, coccobacillus, and Ecoli infection. Klebsiella was found in 17, proteus-11, pseudomonas-17, staphylococcus-52. Combined infection klebsiella and pseudomonas-4, klebsiella and proteus-11, klebsiella and staphylococcus-15, klebsiella and acenatobacter-1. Proteus and staphylococcus-10, and pseudomonas and staphylococcus-2.

CHART – 7



Figure–16 Total Number of Single Organisms

The following chart tells the total no of combined organisms



Figure–17 Total Number of Combined Organisms

The combined organisms infection was more with klebsiella and staphylococcus.

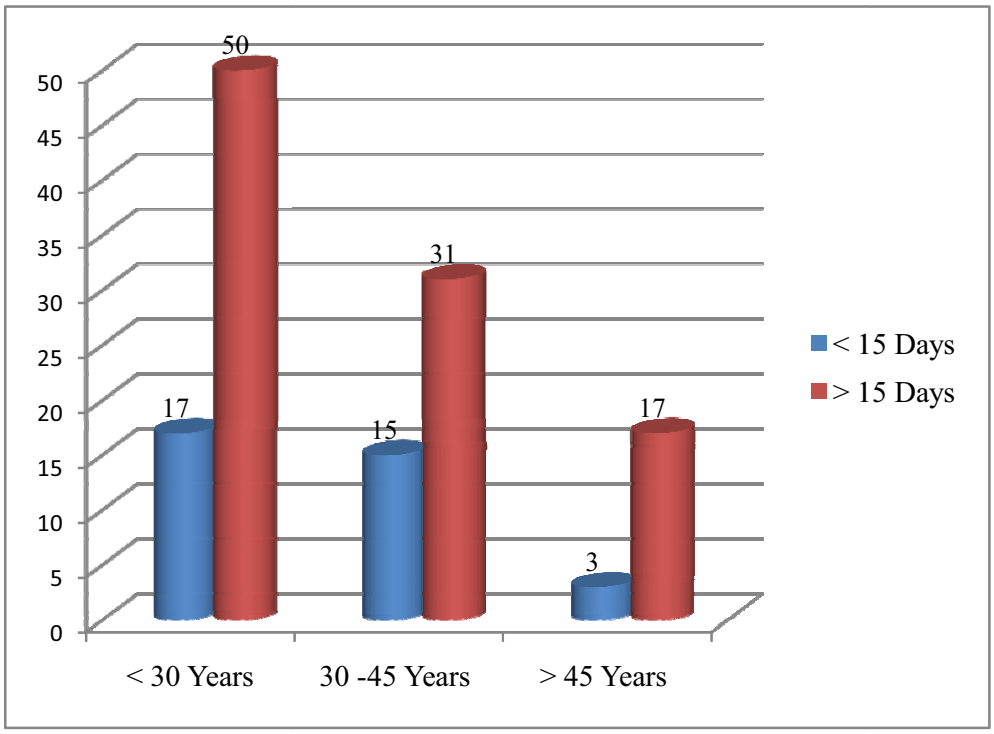
5.8 AGE AND HOSPITAL DAYS:-

DAYS	AGE GROUP			
Years	**< 30 Years**	**30 -45 Years**	**> 45 Years**	**Total**
< 15 Days	17	15	3	**35**
%	48.6%	42.9%	8.6%	**100.0%**
> 15 Days	50	31	17	**98**
%	51.0%	31.6%	17.3%	**100.0%**
Total	**67**	**46**	**20**	**133**

The analysis between the hospital days and age group showed more number in longer duration of stay 98/133 as compared to lesser duration which was 35/133.

The longer duration of stay was found higher among younger age groups which is less than 30 years of age around50/67 which is around 74.62%.

CHART – 8



Figure–18 Comparison between Age and Hospital Days

The above table and diagram shows the observation between the age and hospital days. It is seen that in all age group, more number are having longer duration of stay, but younger age group<30 years are around 50 and have longer duration of stay. There was no statistical significance.

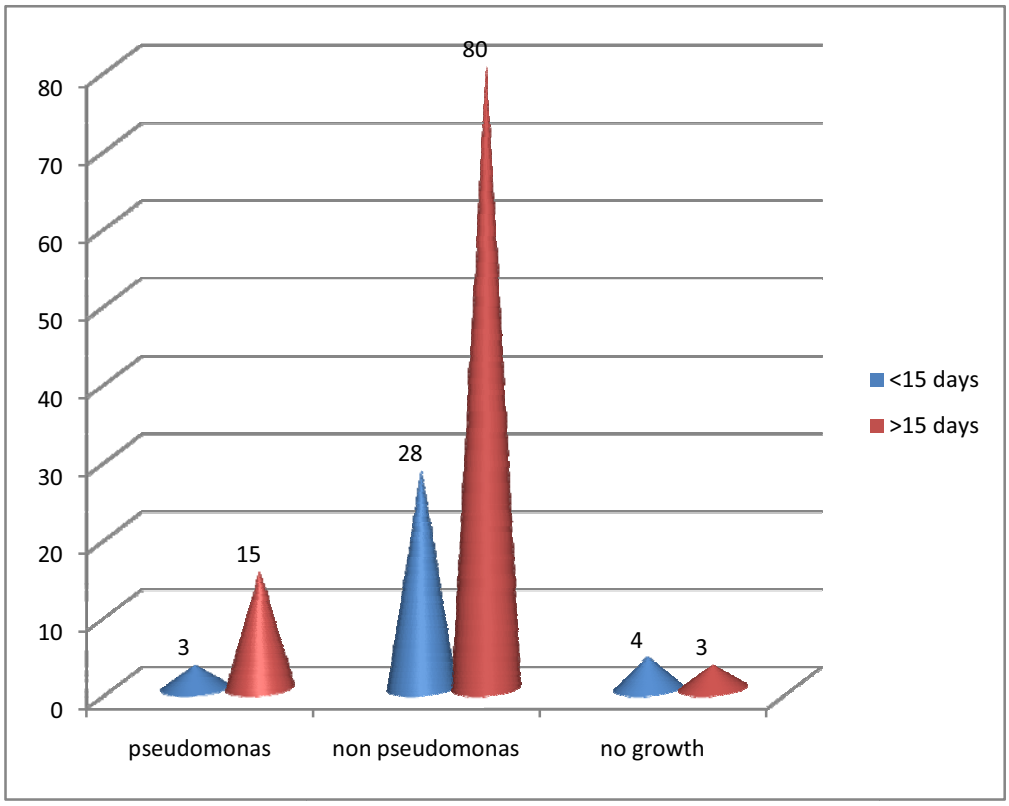
5.9. ORGANISM AND HOSPITAL DAYS:-

As already seen organisms observed in the study were grouped into three pseudomonas, non pseudomonas which includes other organisms like (staphylo coccus, proteus ,klebsiella etc and combined organisms) and no growth. The comparison between organisms and duration of stay is as follows.

ORGANISM GROUP	**Hospital days**		
	< 15 Days	**> 15 Days**	**Total**
Pseudomonas	3	15	**18**
%	16.7%	83.3%	**100.0%**
Non- Pseudomonas	28	80	**108**
%	25.9%	74.1%	**100.0%**
No Growth	4	3	**7**
%	57.1%	42.9%	**100.0%**
Total	**35**	**98**	**133**

Non pseudomonas organism 74.1% had longer duration of stay. It was not statistically significant. No growth of any organism was found in three patients who had longer duration of stay.

CHART – 9



Figure–19 Comparison between Organisms and Hospital Days

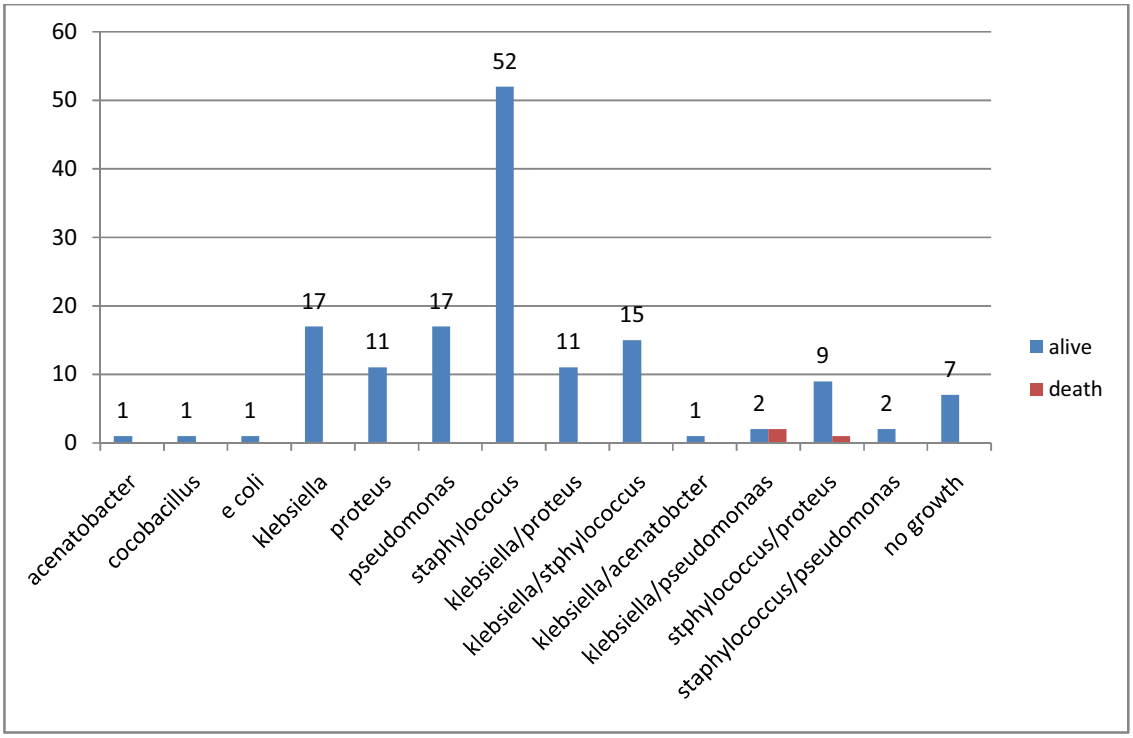
From the study it is seen organisms which include staphylococcus, klebsiella, proteus, acinetobacter, and combined organisms where more among stress hyperglycaemic patients and they had longer duration of stay. Among them staphylococcus was higher, found in 52 patients and among combined organism staphylococcus and klebsiella was high found in 15 patients.

5.10 ORGANISM AND MORTALITY:-

From the study only three mortality was found. The three was in diabetic group. Analysis was done for the total 150 patients. The mortality was see in combined organism group. It was seen in pseudomonas and klebsiella group and staphylococcus and pseudomonas group.

Pseudomonas and klebsiella was found among two and staphylococcus and pseudomonas was found in one patients who died. All patient were diabetic.

CHART – 10



Figure–20 Comparison between Organisms and Mortality

The above chart show that mortality was found among combined organism than single organism. Staphylococcus infection is seen in more number of patients 52/150.

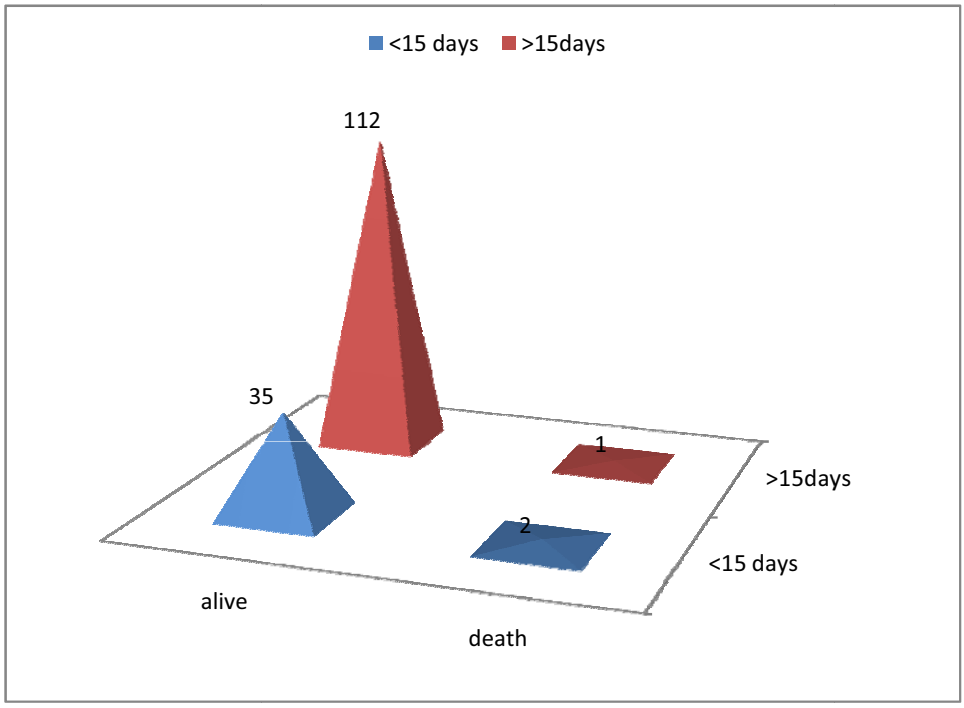
5.11. MORTALITY AND HOSPITAL DAYS:-

Since only 3 deaths and all three were diabetic, mortality and hospital days was seen for all the 150 patients. It was observed longer duration of stay was not associated with mortality.

OUT COME	**DAYS GROUP**		
	<15 days	**>15 days**	**Total**
Alive	35	112	**147**
%	23.8%	76.2%	**100.0%**
Death	2	1	**3**
%	66.7%	33.3%	**100.0%**
Total	**37**	**113**	**150**

Among the 3 who died 2/3 had shorter duration of stay and only one had longer duration of stay. It was not statistically significant.

CHART – 11



Figure–21 Comparison between Mortality and Hospital Days

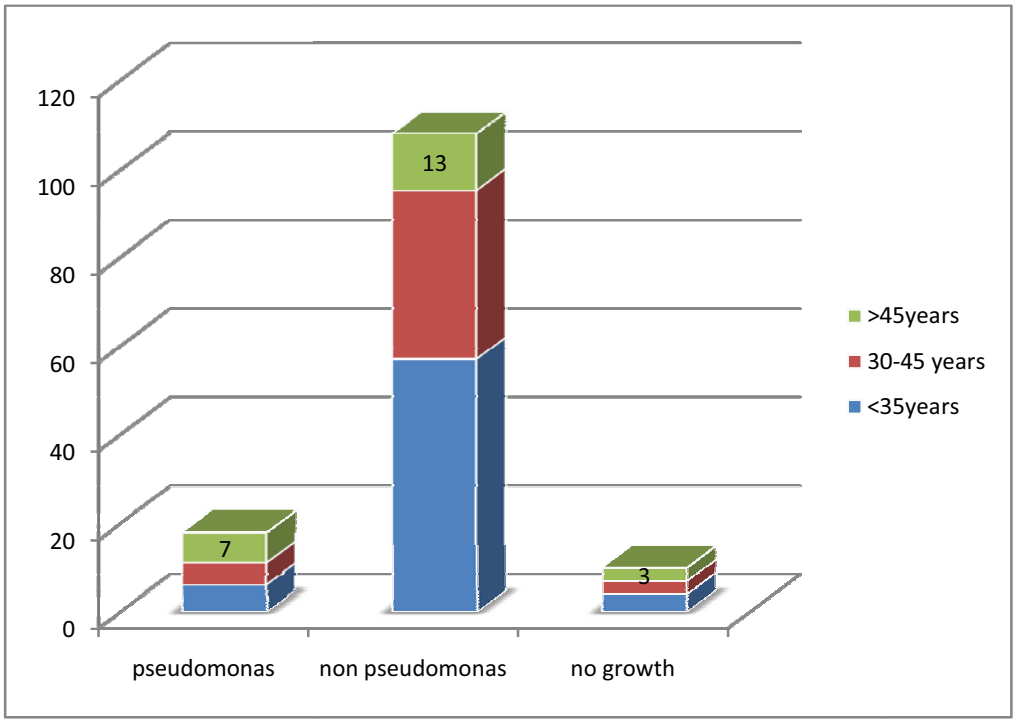
There were no death in stress hyperglycaemic patients in this study. Mortality was seen in 3 patients but all three patients were diabetic.

5.12 ORGANISMS AND AGE GROUP:-

ORGANISM GROUP	AGE GROUP			
	<30years	**30-45years**	**>45 years**	**Total**
pseudomonas	6	5	7	**18**
%	33.3%	27.8%	38.9%	**100.0%**
Non pseudomonas	57	38	13	**108**
%	52.8%	35.2%	12.0%	**100.0%**
No growth	4	3	0	**7**
%	57.1%	42.9%	.0%	**100.0%**
Total	**67**	**46**	**20**	**133**

As seen non pseudomonas organism were more and among them younger individuals had infection more with non pseudomonas organisms. Infection is always found in older age group. There was no older age without organism infection.

CHART – 12



Figure–22 Comparison between Organisms and Age Groups

From the study as already seen non pseudomonas organism were higher and their distribution was also higher in the younger age group. The chi square 10.125 and p value - 0.05 which is statistically significant. Younger individuals are having non pseudomonas infection more common.

5.1.1 CORRELATION:

Using bivariate correlation, correlations seen in the study. The average burns % observed was in the range of 23%in all the three age group. There was no significant correlation between height, weight, sugar, Hba1c, and age group in the study.

Descriptive Statistics

	Mean	Std. Deviation	N
AGE	33.28	13.646	133
PR	86.70	12.630	133
HEIGHT	158.00	4.616	133
WEIGHT	59.50	7.844	133
%BURNS	23.53	5.113	133
RBS	227.48	33.382	133
FBS-D1	166.29	32.504	133
FBS-D2	152.63	24.806	133
FBS-D3	141.64	24.261	132
HBA1C%	5.47	.574	133
DAYS	19.71	7.418	133

6. DISCUSSION

The analysis of stress hyperglycemia in 150 burns patients and its outcome was done in this study .They were analysed with respect to age, sex, percentage of burns, duration of stay, recovery or death, organism found and urine acetone. Since the treatment was varying for each patient and there was no protocol for treatment it could not be seen. Urine acetone was negative for all in the study. In this study out of total 150 patients 72 were males and 78 were females. Out of the 150 patients 17 were diagnosed as diabetes with HbA1c.

Among the 150 patients 133 were with stress hyperglycemia, which is around 88.6% in this study. This is comparable to previous studies, but incidence there was around 27% and 12% respectively [11 12] however in the previous studies the number of people involved was large which could attribute to lower incidence and also did not differentiate between diabetes and stress hyperglycemia. There are rarely any studies that distinguish between stress and diabetes induced hyperglycemia using HbA1c.

6.1 STRESS HYPERGLYCEMIA AND SEX:-

From the study it was seen females and males were more or less equal. Among the 133 patients 67 were female and 66 were male. Females were one number higher than males.

6.2 STRESS HYPERGLYCEMIA AND AGE GROUP:-

In the study patients were grouped into three age groups as already seen. Stress hyperglycaemic sugar was also seen for < 250 mg and > 250 mg. Age group <30 years were more in the study and they had sugar in the range of 200- 250mg. This shows stress hyperglycemia was more for younger age group as compared to older age. Inspite of co morbid conditions in older age, stress hyperglycemia has been found in younger age group in this study possibly due to more accidental burn injury seen in younger patients. There was however no statistical significance between stress hyperglycemia and age group.

6.3 STRESS HYPERGLYCEMIA AND HOSPITAL DAYS:-

From the study we see there are more number of patients with longer duration of stay among the stress hyperglycemia. From the figure-14, 69.1% with sugar < 250 are having longer duration of stay but, the number of people with more than > 250 sugar range are less, but among them 95.7% are having longer duration of stay. This shows stress hyperglycemia

increases the duration of stay probably due to tress hyperglycaemic effects on healing and other factors. The average duration of stay observed in the study is around 19.71 days. It is similar to previous study Silmara et al which shows longer duration of stay in hyperglycemia and mean days around 15.5 days.

6.4 STRESS HYPERGLYCEMIA AND ORGANISMS:-

Pseudomonas is most common organism among burns patients and so patients were taken as three groups for analytical purpose. Other organisms staphylococcus, proteus, klebsiella, acinetobacter, combined organism infection were taken as one group and no growth the other group. Non pseudomonas organisms as a group were high among the stress hyperglycemia. Thus in stress hyperglycemia non pseudomonas organisms are high, and this is statistically significant. Non pseudomonas infection people had sugar in the less than 250 mg range. It shows lesser sugar is also associated with more organisms.

The total number of organism found in study shows more was seen with staphylococcus around 52. Among combined organism more number was seen with klebsiella and staphylococcus around 15. The organism was seen for all the 150 patients. This shows from study common organism in high sugar is staphylococcus.

6.5 STRESS HYPERGYCEMIA AND MORTALITY:-

In this study group only 3 patients had died, and they were also diabetic newly detected. So burns with diabetes which is a more septic condition, would also have been the cause for death. Among stress hyperglycaemic patients there was no death seen in this study. This is because in the study more younger age had stress hyperglycemia, than older age group. Mortality increases with age, so younger age group were able to withstand the adverse effects.

From the study we see that females were more compared to males and among them more females were in the younger age group. In the older age group more males were found .young females are more prone to burns than males, could be a reason for more young females.

The duration of stay with respect to sex was higher among males around 81.8% as shown in figure-11, compared to females. The organism commonly found includes mixed organisms ie non pseudomonas organism which from the study was found to be higher in younger age group <30 years which is around 51%. In the study it is also found that older age group were not without organism. No growth was zero in the older age group which tells us that older age are more prone to infection.

6.6 AGE AND HOSPITAL DAYS:-

From the study there were more patients with longer duration of stay and this was also high among younger individuals. In the study using bivariate correlation, it was observed there was no significant correlation between variables pulse rate, height, weight, age, percentage of burns, random blood sugar, fasting blood sugar, HbA1c and day's .The average age was around 33. The average duration of stay 19.71 days. The average percentage of burns observed was 23%. The average range of HbA1c was around 5.47. Random blood sugar was in the range of 227 and fasting blood sugars in the range of 140-160.

7. CONCLUSION

- The stress hyperglycaemic patients observed in this study are 133/150 which is around 88.6%.
- Newly detected diabetes from the study are 17 with 11.3% incidence.
- The sugar range for stress hyperglycemia was between 200- 250 mg.
- The stress hyperglycaemic patients have longer duration of stay.
- Males with stress hyperglycemia had longer duration of stay.
- The Stress hyperglycaemia is higher among younger age group which is less than 30 years.
- Stress hyperglycemia is more among young females compared to males. 52% females had stress hyperglycemia.
- Stress hyperglycemia showed non pseudomonas organisms more.
- Non pseudomonas organism more in younger age group.
- Non pseudomonas organisms in stress hyperglycemia was found in the sugar range <250 mg / dl.
- Non pseudomonas organisms showed longer duration of stay.
- The common single organism found in the study is staphylococcus.
- The common combined organism found in the study is klebsiella and staphylococcus
- Mortality was found in diabetic patients with combined organism infection.
- No Mortality in stress hyperglycemia.

8. LIMITATIONS OF STUDY

1. The number patients taken for study should be more.

2. Long term follow up of patient with stress hyperglycemia to be seen to find out how many of them develop diabetes.

3. The effect of modality of treatment for stress hyperglycemia to be studied.

9. RECOMMENDATIONS

- Study to be done comparing outcomes in stress hyperglycemia and diabetes patients.
- Stress hyperglycemia in all stressful conditions to be compared to see which causes more hyperglycemia.
- HbA1c in stress hyperglycemia and diabetes patients can be studied to see any cut of value is present.

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ABBREVIATIONS

FFA	-	Free fatty acid
TNF α	-	Tumour necrosis factor alpha
IL-1	-	Interleukin 1
VLDL	-	Very low density lipoprotein
TG	-	Triglyceride
LIMP	-	Lysosomal integrated membrane protein
NF κB	-	Nuclear factor kappa B
ANP	-	Atrial natriuretic peptide
DAG	-	Diacylglycerol
PKC	-	Protein kinase C
IKK	-	Inhibitor of kappa B kinase enzyme

PROFORMA FOR STUDY ON STRESS

HYPERGLYCEMIA IN MODERATE DEGREE BURNS

S.No : Date :

Name : Age :

Sex : IP No:

D.O.Admission D.O.Discharge

Degree of burns

Body Surface Area Burnt

Area	% TBSA	Depth
Head & Neck		
Anterior Trunk		
Posterior Trunk		
Upper Limbs		
Lower Limbs		
Gluteal region		
Perineum		
TOTAL		

Past h/o:

Personal h/o:

Family h/o:

General examination:

Height : Weight :

CVS :

RS :

P/A :

CNS :

BP : PR :

INVESTIGATIONS :

Blood sugar (R) :

Fasting blood sugar :

Day 1	Day 1	Day 3

Post prandial blood sugar :

Urine Acetone :

HbA1c :

Blood urea :

Serum creatinine :

Serum electrolytes :

Hb :

Others :

DIAGNOSIS :

1. Burns
   - Degree
   - Percentage
2. Stress Hyperglycemia
3. Diabetes Mellitus

OUT COME :

1. Recovery/Death
2. No.of days hospital stay
3. Infections – Type – Organism
4. Ketones

MASTER CHART

S.No	NAME	AGE	SEX	PR	HEIGHT	WEIGHT	%BURNS	RBS	FBS-D1	FBS-D2	FBS-D3	HBA1C%	ORGANISM	DAYS	DM/SH	MORTALITY
1	lalli	19	female	78	154	48	30	202	127	129	132	5.1	aceinatobacter	27	SH	Alive
2	mariammal	28	female	92	158	53	30	300	197	189	191	6.4	coccobacilus	19	SH	Alive
3	jayaprada	30	female	78	161	55	30	221	153	148	151	6	ecoli	39	SH	Alive
4	kamaludeen	45	male	86	154	61	28	225	155	162	152	5.2	kleb/proteus	11	SH	Alive
5	shanti	30	female	97	149	55	30	203	128	131	136	5.6	kleb/proteus	17	SH	Alive
6	elumalai	35	male	70	157	62	17	202	126	129	132	5.2	kleb/staph	10	SH	Alive
7	amudha	25	female	64	155	50	19	204	131	136	141	5.6	kleb/staph	15	SH	Alive
8	durairaj	30	male	76	154	60	17	203	128	143	139	5.4	kleb/staph	16	SH	Alive
9	rajendran	52	male	102	152	62	30	205	142	146	151	4.8	kleb/staph	18	SH	Alive
10	vinayagam	80	male	82	161	48	22	201	135	127	143	5.1	kleb/staph	18	SH	Alive
11	manjula	18	female	88	154	48	27	212	221	210	209	6.6	kleb/staph	25	SH	Alive
12	arul	25	male	88	163	64	19	216	139	127	130	5.2	kleb/staph	34	SH	Alive
13	prashanth	48	male	86	162	54	28	269	246	238	226	7.9	kleb/staph	34	SH	Alive
14	rani	25	female	88	152	57	30	240	142	139	147	5.6	kleb/pseudo	27	SH	Alive
15	chinamal	72	female	90	156	64	28	318	232	218	239	10.4	kleb/pseudo	34	DM	Alive
16	kattimuthu	28	female	78	162	74	22	240	135	138	146	5.4	klebsiellaa	28	SH	Alive
17	vanisree	25	female	66	160	72	26	202	132	139	146	5.4	kleb/pseudo	17	SH	Alive
18	yogalakshmi	27	female	94	160	55	20	208	142	137	128	4.5	klebsiella	11	SH	Alive
19	gandhimathi	30	female	90	157	51	27	240	192	186	168	6.3	klebsiella	12	SH	Alive
20	shanthi	19	female	72	154	48	22	275	202	212	219	6.4	klebsiella	12	SH	Alive
21	selvam	24	male	88	155	60	26	202	133	129	147	5.4	klebsiella	15	SH	Alive
22	babu	23	male	86	152	70	15	221	179	189	185	5.8	klebsiella	15	SH	Alive
23	datchyani	31	female	69	155	49	16	202	128	139	143	5.5	klebsiella	18	SH	Alive
24	sakthipriya	19	female	68	158	56	28	234	169	207	166	6.2	klebsiella	18	SH	Alive
25	peter	24	male	72	149	60	28	265	190	182	191	5.4	klebsiella	18	SH	Alive
26	priya	21	female	98	165	75	30	202	131	127	138	5.3	klebsiella	19	SH	Alive
27	sasikumar	37	male	60	163	61	30	289	202	165	197	6.1	klebsiella	22	SH	Alive
28	kamatchi	55	female	100	153	51	30	423	242	229	199	6.7	klebsiella	22	DM	Alive
29	dakshayani	29	female	80	153	64	22	212	146	133	129	5.8	klebsiella	24	SH	Alive
30	indira	47	female	98	160	60	26	236	174	169	158	6.9	klebsiella	27	DM	Alive

S.No	NAME	AGE	SEX	PR	HEIGHT	WEIGHT	%BURNS	RBS	FBS-D1	FBS-D2	FBS-D3	HBA1C%	ORGANISM	DAYS	DM/SH	MORTALITY
31	aravindan	18	male	64	159	70	30	301	243	220	198	5.7	klebsiella	27	SH	Alive
32	pitchandi	30	male	84	160	62	22	234	167	136	124	5.8	klebsiella	29	SH	Alive
33	alamelu	35	female	86	155	58	22	362	225	199	179	6.9	klebsiella	42	DM	Alive
34	jansi	28	female	90	161	50	22	202	168	156	146	5.7	kleb/proteus	26	SH	Alive
35	peter	24	male	90	157	50	27	225	104	112	94	4.4	no growth	10	SH	Alive
36	usha	23	female	99	149	50	18	202	127	132	135	5.8	no growth	12	SH	Alive
37	mariammal	40	female	94	163	70	19	206	136	131	129	5.5	no growth	12	SH	Alive
38	rabeka	20	female	90	156	50	23	218	137	136	158	5.2	no growth	14	SH	Alive
39	krishnan	35	male	76	164	54	17	240	138	145	132	5.9	no growth	15	SH	Alive
40	narasiman	45	male	120	162	64	30	280	162	150	147	5.3	no growth	16	SH	Alive
41	muthukumar	23	male	69	161	49	24	223	132	129	120	5	no growth	24	SH	Alive
42	palanivel	52	male	72	163	64	24	202	145	137	128	5.2	kleb/proteus	18	SH	Alive
43	rajesh	24	male	69	164	52	18	204	129	125	127	4.9	kleb/proteus	21	SH	Alive
44	murali	35	male	88	156	62	20	302	201	199	185	6.4	staph/proteus	29	SH	Alive
45	manickam	35	male	76	154	60	25	218	128	138	126	5.8	kleb/proteus	18	SH	Alive
46	amudha	25	female	89	163	75	20	215	127	139	126	5.4	kleb/proteus	24	SH	Alive
47	sujanamal	80	female	112	161	60	30	230	202	135	127	6.3	staph/ proteus	10	SH	Alive
48	meena	28	female	76	151	58	25	302	231	187	220	8.5	staph/proteus	33	DM	Alive
49	balaraman	28	male	77	158	70	17	208	126	134	138	4.7	proteus	10	SH	Alive
50	rani	25	female	87	172	71	27	202	142	136	129	4.7	proteus	11	SH	Alive
51	deivayanai	35	female	96	158	57	30	202	132	126	127	5.5	proteus	15	SH	Alive
52	selvam	28	male	82	153	56	22	205	135	126	129	4.6	proteus	17	SH	Alive
53	kulanjium	37	male	89	160	72	19	220	145	121	134	5.1	proteus	19	SH	Alive
54	umakanth	25	male	86	159	62	15	220	175	126	134	5.6	proteus	20	SH	Alive
55	jayaram	26	male	72	165	73	19	227	156	143	132	6.6	proteus	22	DM	Alive
56	dandapani	27	male	84	158	62	28	240	187	177	159	5.6	proteus	30	SH	Alive
57	selvi	24	female	78	156	52	30	287	156	136	129	5.7	proteus	30	SH	Alive
58	nallasivam	37	male	74	166	62	16	202	143	132	127	5.3	kleb/proteus	14	SH	Alive
59	siddharth	24	male	86	162	54	16	224	159	143	139	4.8	kleb/proteus	15	SH	Alive
60	ragendran	31	male	88	165	60	23	202	138	125	122	5.1	kleb/proteus	17	SH	Alive

S.No	NAME	AGE	SEX	PR	HEIGHT	WEIGHT	%BURNS	RBS	FBS-D1	FBS-D2	FBS-D3	HBA1C%	ORGANISM	DAYS	DM/SH	MORTALITY
61	madinabeg	36	female	98	152	68	30	342	252	227	219	7.3	kleb/proteus	11	DM	Death
62	ramadoss	37	male	90	156	50	18	273	245	200	180	8.2	kleb/pseudo	13	DM	Death
63	shantha	44	female	110	159	52	20	240	196	179	154	5.1	staph/pseudo	24	SH	Alive
64	duraisamy	50	male	74	160	63	26	278	201	187	162	5.5	staph/pseudo	25	SH	Alive
65	kaviya	44	female	69	158	60	27	202	154	143	132	4.9	pseudomonas	10	SH	Alive
66	suseela	65	female	100	158	50	25	204	164	156	132	5.9	pseudomonas	10	SH	Alive
67	surya	29	female	98	154	60	15	202	133	127	126	5.8	pseudomonas	12	SH	Alive
68	mohan	27	male	78	156	63	18	205	126	134	122	6	pseudomonas	15	SH	Alive
69	datchyani	32	female	88	154	60	30	212	129	132	122	5.8	pseudomonas	16	SH	Alive
70	muniyamal	80	female	84	159	64	15	225	136	126	131	5.4	pseudomonas	16	SH	Alive
71	suganthi	33	female	94	161	60	27	225	172	185	112	6.2	pseudomonas	16	SH	Alive
72	kalavathy	19	female	70	154	48	18	253	112	128	143	4.9	pseudomonas	18	SH	Alive
73	jabasina	27	female	87	148	65	15	218	102	112	90	5.9	pseudomonas	20	SH	Alive
74	dayalan	60	male	84	160	60	15	390	185	166	159	5.9	pseudomonas	23	SH	Alive
75	boopathy	27	male	96	155	50	30	257	231	197	188	5.8	pseudomonas	27	SH	Alive
76	asthar	53	male	91	159	80	30	205	127	129	125	5.2	pseudomonas	28	SH	Alive
77	munusamy	47	male	98	158	70	30	213	162	164	202	6.2	pseudomonas	32	SH	Alive
78	padmapriya	38	female	83	158	52	25	260	152	146	102	5.5	pseudomonas	33	SH	Alive
79	ramsingh	50	male	98	157	62	30	252	198	152	108	5.9	pseudomonas	37	SH	Alive
80	roopadevi	30	female	92	157	60	24	282	164	158	149	7.4	pseudomonas	38	DM	Alive
81	malathy	25	female	86	154	44	21	356	225	168	156	5.4	pseudomonas	38	SH	Alive
82	ramachandran	42	male	79	162	62	22	225	169	158	`123	6	staphlococcus	9	SH	Alive
83	maruti	30	male	100	163	65	18	202	143	134	118	5.7	staph/proteus	22	SH	Alive
84	samandamurti	48	male	60	162	59	16	202	153	146	135	5.5	staph/proteus	22	SH	Alive
85	satishkumar	24	male	79	162	60	27	225	177	154	149	6.5	staph/proteus	23	DM	Alive
86	selvi	45	female	78	154	51	27	320	226	203	183	7.3	kleb/staph	36	DM	Alive
87	mariyammal	40	female	88	149	49	18	202	147	139	123	5.3	staphylococcus	12	SH	Alive
88	adhilakshmi	34	female	100	152	59	30	202	132	129	116	5.8	staphylococcus	11	SH	Alive
89	karthika	20	female	96	155	56	18	202	156	142	136	6	staphylococcus	16	SH	Alive
90	rajanbabu	58	male	100	155	72	22	225	158	156	146	5.3	staphylococcus	18	SH	Alive

S.No	NAME	AGE	SEX	PR	HEIGHT	WEIGHT	%BURNS	RBS	FBS-D1	FBS-D2	FBS-D3	HBA1C%	ORGANISM	DAYS	DM/SH	MORTALITY
91	sargunam	40	male	72	164	57	23	225	168	136	125	5.1	staphylococcus	19	SH	Alive
92	roja	20	female	76	164	70	18	214	206	168	156	5.7	staphylococcus	42	SH	Alive
93	baskar	29	male	99	158	64	17	202	187	173	164	5.5	kleb/staph	14	SH	Alive
94	alamelu	32	female	100	154	60	25	270	188	165	128	5.6	kleb/staph	16	SH	Alive
95	vellama	35	female	88	154	63	27	201	156	130	112	5.1	kleb/staph	22	SH	Alive
96	ramamurthy	18	male	78	170	72	19	202	145	130	123	5.9	kleb/staph	22	SH	Alive
97	samandham	30	male	98	162	60	25	202	157	147	129	5.5	kleb/staph	24	SH	Alive
98	samsun	32	male	70	155	60	24	265	198	170	138	5.9	kleb/staph	27	SH	Alive
99	gnanavel	32	male	96	157	62	27	202	178	156	122	4.5	staph/proteus	15	SH	Alive
100	nagaraj	41	male	81	159	49	30	202	156	138	123	4.7	staph/proteus	16	SH	Alive
101	mounammal	60	female	88	160	60	26	202	179	156	143	4.3	staphylococcus	10	SH	Alive
102	mangai	32	female	97	158	65	28	276	221	194	147	6.8	staph/proteus	22	DM	Death
103	chandran	42	male	101	152	55	20	202	167	156	143	4.9	staphylococcus	26	SH	Alive
104	savithri	42	female	74	156	42	15	208	182	146	122	6	proteus	34	SH	Alive
105	krishnaveni	60	female	76	152	62	26	287	245	197	185	7.4	proteus	45	DM	Alive
106	sasikala	25	female	120	150	53	19	202	148	137	122	5.6	kleb/proteus	12	SH	Alive
107	kavitha	20	female	110	156	46	30	208	156	143	129	4.9	staphylococcus	24	SH	Alive
108	soujanya	19	female	70	157	50	15	218	138	122	120	4.1	staphylococcus	11	SH	Alive
109	laxmi	30	female	75	168	54	25	202	176	165	142	5.1	staph/proteus	14	SH	Alive
110	santhakumar	20	male	89	157	55	18	202	163	156	122	4.4	staphylococus	9	SH	Alive
111	ravi	42	male	88	158	60	28	202	158	134	119	5.2	staphylococus	10	SH	Alive
112	revathy	19	female	77	156	62	20	220	196	156	132	5.2	staphylococus	12	SH	Alive
113	prashanth	23	male	110	155	60	24	256	188	164	132	5.1	staphylococus	12	SH	Alive
114	aruna	35	female	88	159	60	26	202	143	132	122	4.9	staphylococus	12	SH	Alive
115	hyedali	25	male	120	159	81	27	202	168	134	123	5.3	staphylococus	13	SH	Alive
116	vadivelu	42	male	83	165	64	25	202	163	154	134	5.5	staphylococus	13	SH	Alive
117	bamupriya	20	female	104	160	60	27	267	209	167	154	5.9	staphylococus	14	SH	Alive
118	lakshmi	30	female	64	158	67	23	202	167	134	122	4	staphylococus	14	SH	Alive
119	deepa	30	female	90	158	55	16	202	148	136	120	4.7	staphylococus	14	SH	Alive
120	dhandapani	30	male	82	162	58	30	209	189	165	145	4.9	staphylococus	15	SH	Alive

S.No	NAME	AGE	SEX	PR	HEIGHT	WEIGHT	%BURNS	RBS	FBS-D1	FBS-D2	FBS-D3	HBA1C%	ORGANISM	DAYS	DM/SH	MORTALITY
121	naniappan	40	male	100	163	70	15	252	182	165	152	6.2	staphylococus	15	SH	Alive
122	deshma	19	female	68	153	50	28	202	154	134	123	6	staphylococus	16	SH	Alive
123	manjula	29	female	98	154	64	22	220	143	132	124	5.4	staphylococus	16	SH	Alive
124	hariharan	24	male	78	162	55	20	262	185	143	134	6.4	staphylococus	17	SH	Alive
125	manjula	17	female	92	154	52	20	240	201	186	147	6	staphylococus	18	SH	Alive
126	shanmugam	50	male	116	155	48	18	202	178	163	146	4.8	staphylococus	19	SH	Alive
127	mala	18	female	88	164	48	30	202	164	154	134	4.8	staphylococus	19	SH	Alive
128	vadivelu	41	male	66	162	70	25	227	142	130	132	5.9	staphylococus	19	SH	Alive
129	balamurugan	24	male	84	158	63	26	332	208	198	156	6.3	staphylococus	19	SH	Alive
130	murugan	16	male	96	166	70	25	240	148	135	134	5.7	staphylococus	20	SH	Alive
131	muthu	29	male	84	168	68	30	218	186	177	156	4.9	staphylococus	21	SH	Alive
132	dhanalexmi	45	female	89	158	50	26	202	178	168	143	4.2	staphylococus	22	SH	Alive
133	gopi	29	male	84	159	57	20	218	209	187	142	5.3	staphylococus	22	SH	Alive
134	rabbin	24	male	94	151	52	17	240	189	149	120	5.1	staphylococus	22	SH	Alive
135	dharani	28	female	98	156	65	30	246	167	154	123	6	staphylococus	22	SH	Alive
136	yasodha	27	female	98	156	62	26	240	208	167	124	5.9	staphylococus	24	SH	Alive
137	unamalai	52	female	79	149	59	30	301	216	186	128	5	staphylococus	24	SH	Alive
138	priya	21	female	96	155	56	30	218	190	156	132	6	staphylococus	25	SH	Alive
139	shankar	58	male	84	154	60	19	223	201	156	167	6.8	staphylococus	26	DM	Alive
140	subaratina	26	female	88	157	56	30	271	223	180	178	5.5	staphylococus	26	SH	Alive
141	shailaja	22	female	84	155	70	20	217	220	178	165	5.2	staphylococus	27	SH	Alive
142	narasingh	20	male	100	168	60	15	231	225	185	170	6.6	staphylococus	28	DM	Alive
143	deivayanai	27	female	90	158	70	25	240	220	174	168	6.2	staphylococus	28	SH	Alive
144	krishanaya	65	male	102	158	64	24	240	220	170	160	5.8	staphylococus	28	SH	Alive
145	alagesan	37	male	88	159	60	28	316	253	220	168	6.6	staphylococus	29	DM	Alive
146	karthick	26	male	95	166	60	30	262	240	190	170	5.2	staphylococus	31	SH	Alive
147	jesinthmary	36	female	78	168	60	15	225	220	200	180	7	staphylococus	34	DM	Alive
148	selvaraj	60	male	98	157	72	27	245	220	190	180	5.5	staphylococus	34	SH	Alive
149	lakshmi	75	female	82	149	60	15	240	220	190	178	6	staphylococus	38	SH	Alive
150	sivagami	22	female	77	159	66	30	219	230	200	190	5.7	staphylococcus	12	SH	Alive

ETHICAL COMMITTEE
GOVT. KILPAUK MEDICAL COLLEGE, KILPAUK,
CHENNAI- 10.
Venue: PANAGAL HALL, KMC
Dt: 01.02.2011

CHAIRPERSON
Prof. Dr.V.KANAGASABAI, MD.,
Dean
Govt. Kilpauk Medical College, Chennai-10
Sub: Ethical Committee project work - approved – regarding.
Ref: Lr.No.3944/Audit/E1/09 Dt. 30.11.2010

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With above reference, the Institutional Ethical committee meeting for the following students was conducted at our Institution on 01.02.2011.

S.NO.	Name	Topic
1.	Dr.Navin Kumar, MS(Ortho), PG., Govt. Royapettah Hospital, Chennai.	1.To Identify a Safe Zone to approach proximal Humerus 2.To study Anatomical relations of Axillary nerve, its course & its Variations
2.	Dr.T.Satheesh Kumar, D.Ortho., PG., Govt. Royapettah Hospital, Chennai	Heriditary Multiple Exostosis
3.	Dr.J. Jeya Shambavi, MD(Pathology), PG., Govt. Kilpauk Medical College, Chennai-10	Clinicopathological Histomorphological and Immunohistochemical Study of Neuroendocrine Tumors of GIT
4.	Dr.L. R. Saranya. MD., (Paed.)PG., Govt. Kilpauk Medical College, Chennai-10	Cord Blood Zinc Level in Term-Small for Gestational Age Neonates
5.	Dr.A.Satheesh Kumar, MS(ENT), PG., Kilpauk Medical College, Chennai	Study on Cases of Chronic Suppurative Otitis Media in Tubo Tympanic Type Due to Sinusitis as Focal Sepsis
6.	R.Prathiban,(Msc.,Physiology), PG., Student, The TN. Dr.MGR Medical University,Chennai-32	Prevalence of Cardiac Dysautonomia in Type I Diabetes mellitus
7.	B. Manikandan, (Msc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Comparative Study of Left Ventricular Structure and Function in Obese and Non Obese Subjects
8.	G. Selvakumar, (MSc., Physiology), PG., Student, The TN Dr.M.G.R. Medical University, Chennai-32.	A Study of the Intraocular Pressure In Patients with Diabetic Normotensive, Diabetic Hypertensive and Normal Subjects

9.	R. Ragutji, (Msc.,Physiology), PG., The TN Dr.MGR Medical University, Chennai-32.	A Study of Pulmonary function in insulin dependent diabetes mellitus
10.	V.M. Jenila Vemy,(MscPhysiology), PG. The TN Dr.MGR Medical University, Chennai-32	Cardiovascular Autonomic Dysfunction in Chronic Kidney Disease
11.	Dr.G. Lakshmi, MD(O&G), PG., Govt. Kilpauk Medical College,Chennai-10	A Study of Association of Thyroid Disorders in Abnormal Uterine Bleeding
12.	Dr.R. Harini, MD(O&G), PG., Kilpauk Medical College, Chennai	Single Dose Antibacterial treatment for Asymptomatic Bacteriuria in Pregnancy
13.	Dr.E.Geetha, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of the incidence course of Pregnancy and Pregnancy outcome in Obstetric Cholestasis and to evaluate the efficiency of UDCA in relieving the Symptoms and Improving the Perinatal outcome in these Patients
14.	Dr.S. Nithya, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	Prospective Study of Prevalence of diabetes Mellitus, Thyroid Dysfunction and Hyperprolactinemia in Recurrent Pregnancy loss
15.	Dr.Mohideen Fathima, MD(O&G), PG., Govt. Kilpauk Medical College, Chennai-10	A Study of evaluation of multi system changes in Gestational hypertension / severe pre-eclamptic/eclampasia patients
16.	Dr.M.Padma Priya, MD(O&G), PG., Kilpauk Medical College, Chennai	Dyslipidemia as a Predictor of PIH
17.	Mrs.G. Savitha, (Msc.,Medical Bio Chemistry), TN Dr.M.G.R.Medical University, Chennai-32.	Association of subclinical hypothyroidism in metabolic syndrome patients
18.	Dr.K. Bharadhwaj, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Peripheral Vascular Disease in Type 2 Diabetes Mellitus
19.	Dr.B.Priya, MD(G.M.), PG	Study of Serum Bilirubin Concentration in Established Coronary Artery Disease
20.	Dr.R.Hema, MD(G.M.), PG.,	Study of Troponin I level in Supraventricular Tachycardia in Non Cad Patients
21.	Dr.P.Manoj Kumar, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study on Pulmonary Functions in Type 2 Diabetes Mellitus
22.	Dr.M.Dhanasekar, MD(G.M.), PG.,	Prognostic Risk Stratification of Acute Coronary Syndrome – Role of Highly Sencitive – Reactive Protien
23.	Dr.N. Karthik, MD(G.M.), PG., Govt.Kilpauk Medical College, Chennai-10	A Study of Comparison of QT Dispersion in Acute Myocardial Infraction Between Early Reperfusion and Late Reperfusion Therapy

24.	Dr.H. Anuradha, MD(G.M.), PG., Kilpauk Medical College, Ch-10	A Study of Stress Hyperglycemia in Moderate Degree Burns
25	Dr. V. Nandakumar, MD(G.M.), PG.,	A Prospective Study of Clinical Profile of Emphysematous Pyelonephritis in Type Two Diabetes Mellitus
26.	Dr.S.Sasikumar, MS(G.S.), PG., Govt. Royapettah Hospital, Chennai	A Study of Unusual Presentations of Appendicitis.
27.	Dr.S.R.Padmanabhan, MS(GS), PG., Govt. Royapettah Hospital, Chennai	A Comparative Study Between Autologous Platelet Rich Plasma and Saline Dressing for Diabetic Ulcer
28.	Dr.C.Rose, Scientist-G and Head, Biotechnology, Central Leather Institute, Chennai.	Wound healing efficacy of the chitosan – containing collagenous biomaterial. on burn wound
29.	E.K. Lavanya,B.Tech, Biotechnology, PG., Prathyusha Institute of Technology and Management, Tiruvallur.	Isolation and Characterization of Bacterial Pathogens from Eye Infection

We are glad to inform you that at the Ethical Committee meeting, the documents were discussed and the above short term projects are Ethically approved.

4/2/11

CHAIRPERSON

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Govt. Kilpauk Medical College,

Chennai-10.

To: The Individuals