"A STUDY ON RISK FACTORS AND PROGNOSIS OF DIABETIC KETOACIDOSIS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL CARE UNIT."

Dissertation submitted in partial fulfillment of the requirement for the award of the Degree of

DOCTOR OF MEDICINE

BRANCH I - GENERAL MEDICINE

APRIL 2016

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

CHENNAI, TAMILNADU

CERTIFICATE

This is to certify that the dissertation entitled **A STUDY ON RISK FACTORS AND PROGNOSIS OF DIABETIC KETOACIDOSIS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL CARE UNIT submitted** by Dr.SALINI.N.R to the faculty of General Medicine, The Tamil Nadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.D. degree, Branch I (General Medicine) is a bonafide research work carried out by her under my strict supervision and guidance during the academic year 2013 to 2016.

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DECLARATION

I, **Dr SALINI N R,** solemnly declare that, this dissertation **A STUDY ON RISK FACTORS AND PROGNOSIS OF DIABETIC KETOACIDOSIS IN PATIENTS ADMITTED IN INTENSIVE MEDICAL CARE UNIT** is a bonafide record of work done by me at the Department of General Medicine, Tirunelveli Medical College, under the guidance of Professor **Dr.S.S.NAZAR,M.D.,** Department of General Medicine, Tirunelveli Medical college, during the academic year 2013-2016. I also declare that this bonafide work or a part of this work was not submitted by me or any others for any award, degree, and diploma to any other University, Board either in India or abroad.

This dissertation is submitted to The Tamil Nadu Dr. M.G.R. Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Degree of Doctor of Medicine (M.D.), General Medicine Branch-I, examination to be held in April 2016.

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THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
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10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

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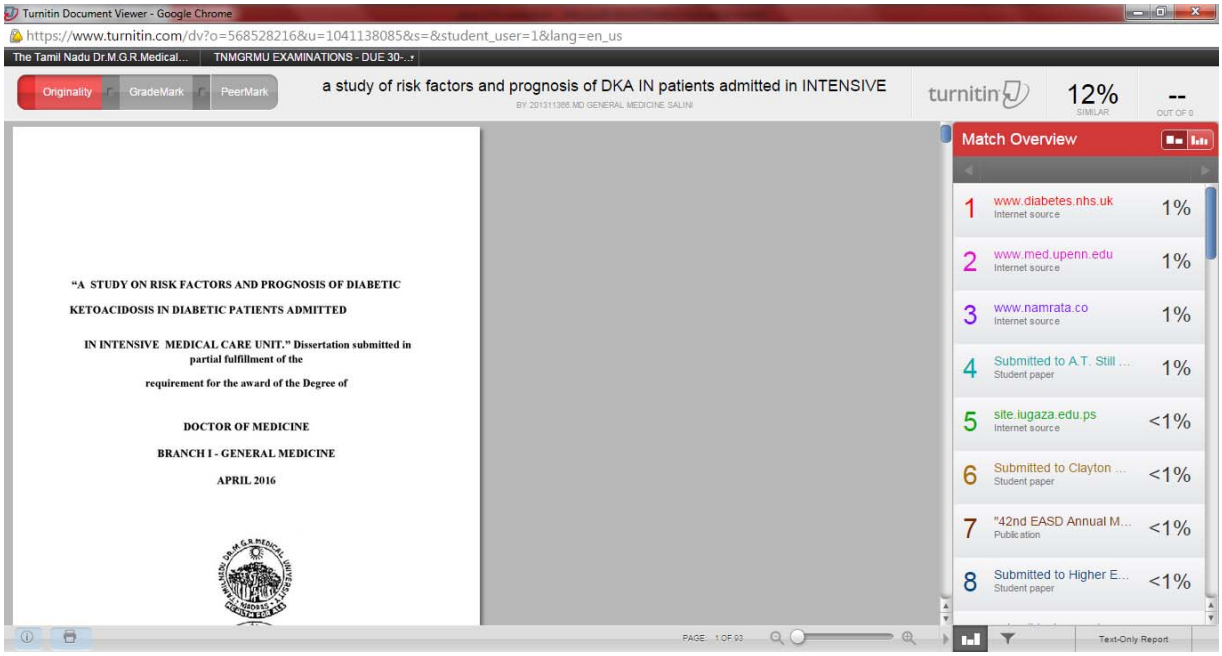
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INTRODUCTION

Diabetes mellitus is a metabolic disease; in which the body's inability to synthesis required amount of insulin causes elevated levels glucose in the blood. Diabetes mellitus is due to either the pancreas not producing enough insulin or due to the cells of the body not responding properly to the insulin produced

Hyperglycemia is due to decreased insulin secretion, decreased glucose utilization in peripheral tissues,or due to more glucose production. Hyperglycemia due to many causes but is most commonly due to type 1 or type 2 diabetes[1].

Diabetes mellitus is a metabolic disorder, characterized by hyperglycemia caused by absolute or relative deficiency of insulin. The worldwide prevalence of diabetes for all age groups was 2.8% in2000 and will be 4.4%2030. In the past two decade's worldwide prevalence of diabetes mellitus increased. As of 2014, an estimated 387 million people have diabetes worldwide with type 2 diabetes making up about 90% of the cases. This represents 8.3% of the adult population. The prevalence of diabetes with equal rates in both women and men. From 2012 to 2014, diabetes is estimated to have resulted in 1.5 to 4.9 million deaths each year.[2] Diabetes doubles a person's risk of death. The number of people with diabetes is expected to rise to 552 million by 2030.The prevalence of both types of diabetes varies around the world, and is due to differences in genetic and environmental factors.

Diabetes can affect more than 62 million Indians, which is more than 7% of adult population. The average age of onset of diabetes is 42 yrs. An estimate shows approximately 1 million Indians die due to diabetes every year. The prevalence was 2.1per cent in urban population and 1.5 per cent in the rural population[3] while in those above 40 year of age, the prevalence was 5 per cent in urban and 2.8 percent in rural areas. The International Diabetes Federation estimates that the total number of diabetic patients to be around 40.9 million in India and this will become 69.9million by the year 2030. Although the prevalence of both type1 and type 2diabetes increasing; the prevalence of type2DM is raising rapidly, because of obesity, sedentary life styles as country become more industrialized and increasing aging of population. The epidemic of diabetes is due to the rapid transition associated with change in dietary patterns and reduced physical activity as evident from more prevalence in urban population[4].

In a multifaceted country like ours, with various ethnicities, cultural and food patterns and above all with varied political outlooks on the aspects of health coupled with significant infrastructure limitations has effectively prevented us from having completely representative comprehensive surveys with regard to demography.

The disease and death registration systems are in a state of shambles. This has been a big bane of our health care system and has prevented us from taking

pre-emptive measures. With a health care system as frail as ours with numerous lacunae it becomes even more imperative to initiate cost-effective solution.

Aim of study

1. To assess risk factors of DKA admitted in intensive medical care unit
2. To assess prognosis of DKA patients in intensive medical care unit.
3. To assess duration of diabetes and the type of treatment patients had taken with outcomes of DKA

REVIEW OF LITERATURE

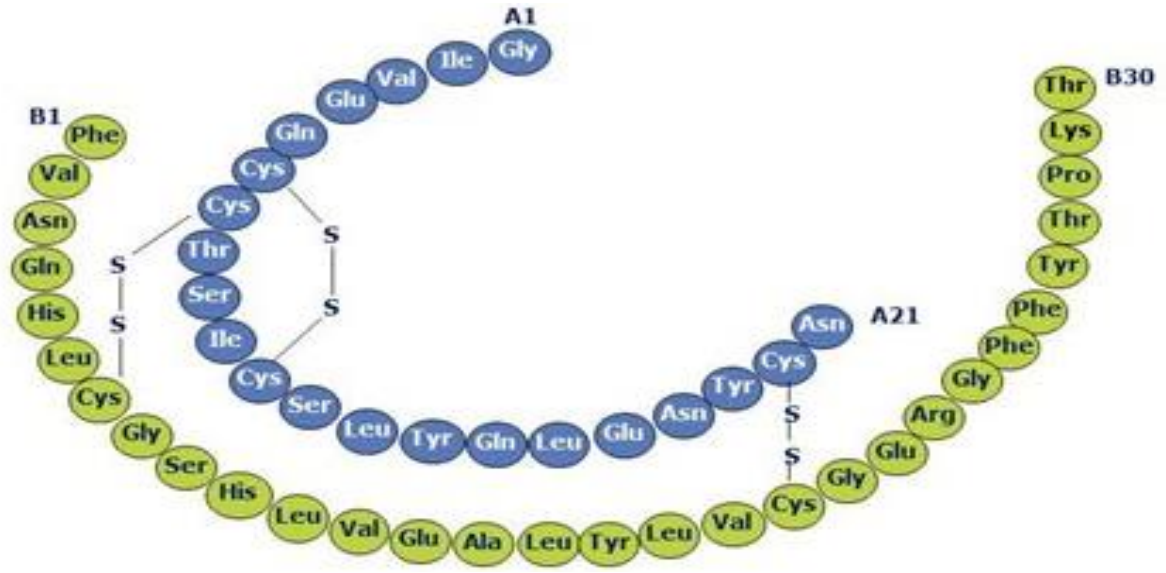
SYNTHESIS OF INSULIN

Insulin is secreted by islets of beta cells in pancreas. The islets are scattered throughout the pancreas, they are more found in the tail than in the body and head. Humans have 1 to 2 million islets 2% of the volume of the pancreatic gland consists of islets. Insulin is synthesized as a larger precursor polypeptide chain preproinsulin. It has 109 amino acids. It is rapidly converted to proinsulin in the endoplasmic reticulum by removal of leader sequence of 23 amino acids; which is transported into golgiapparatus where it is cleaved by a protease; an internal 31-residue fragment cleaved from proinsulin produces the C peptide and the A chain(with -21 amino acids) and B chain(with -30 amino acids) of insulin[5]. The mature insulin molecule and c-peptide are keeping together; both of them secreted from secretory granules in the beta cells.

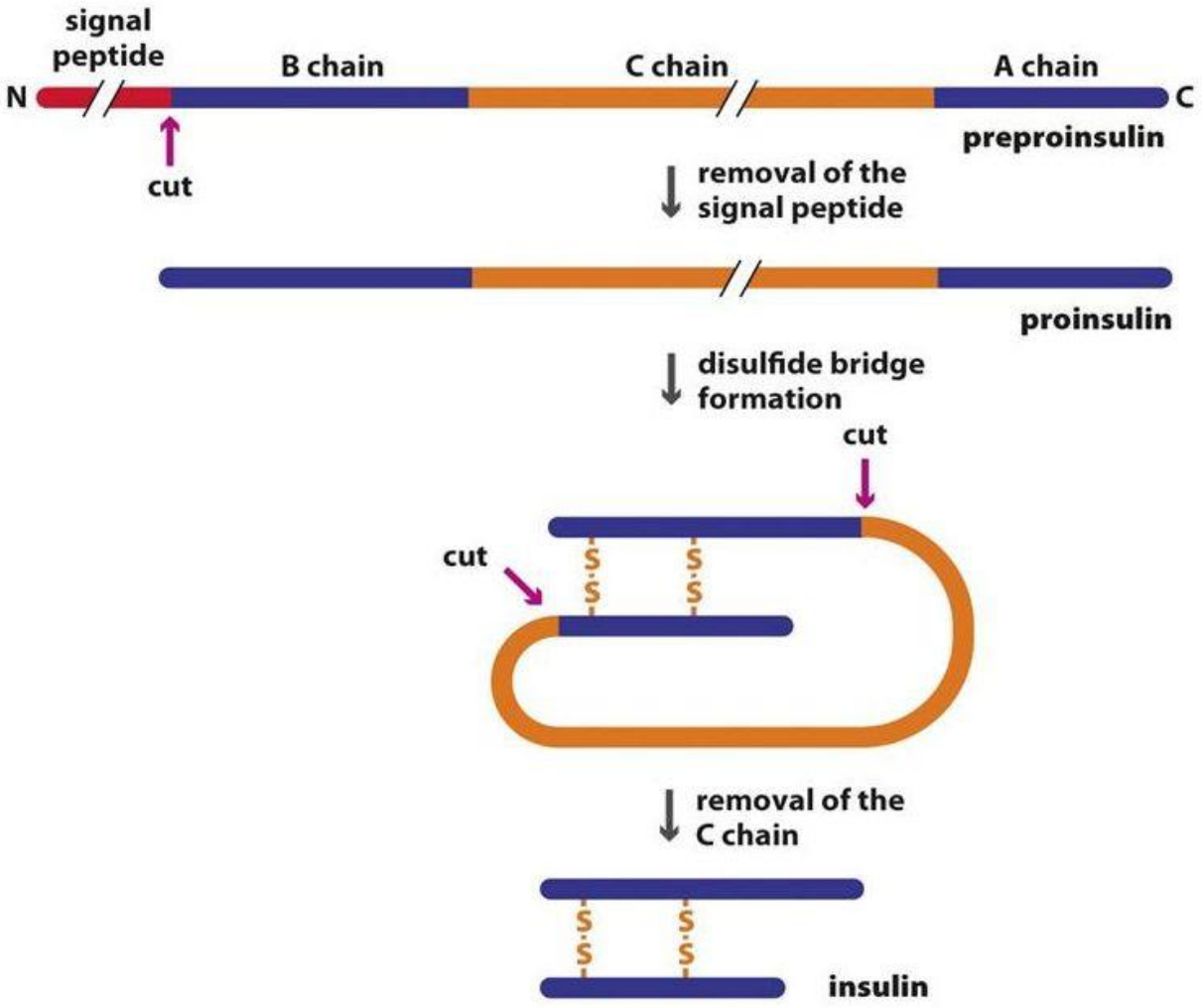
STRUCTURE OF INSULIN

Insulin is a protein hormone with 2 polypeptide chains. The A chain with 21 aminoacids and B chain with 30 aminoacids are joined together by a pair of disulphidebonds. Removal of sulphide bridges cause loss of activity.

Structure of insulin



SYNTHESIS OF INSULIN



SECRETION OF INSULIN

Glucose is an important regulator of insulin secretion from pancreatic beta cells. In the pancreatic βcells; ATP sensitive potassium (k+) channel plays an important role in glucose-stimulated insulin secretion. When Glucose levels reaches 70 mg/dl; insulin production starts by translation and processing of proteins. It is affected by aminoacid, ketone bodies, neurotransmitters, GIPs (Gastrointestinal peptide) etc. Insulin synthesis by Glucose stimulation; starts with

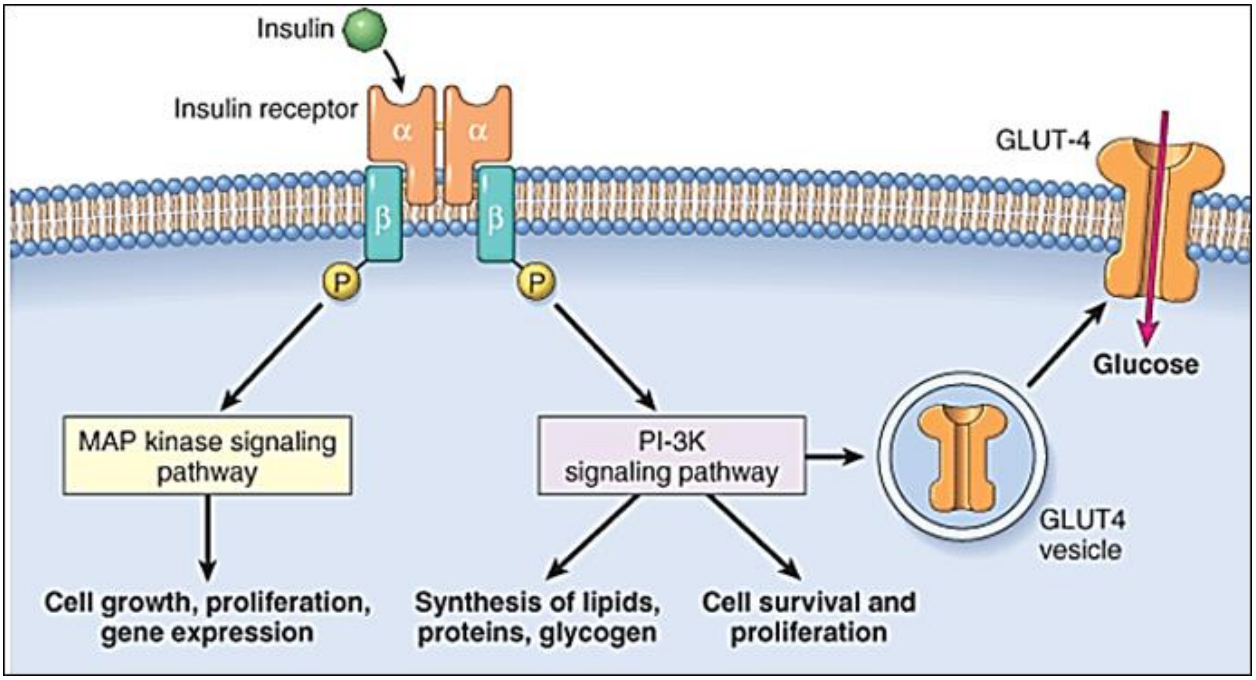
its transport into the beta cell by a facilitative glucose transporter GLUT2. Enzyme glucokinase play an important role for insulin secretion via glucose phosphorylation. Elevated blood glucose increases glucose metabolism in the β-cell and elevates ATP. This metabolic signal close KATP channels, causing beta cell membrane depolarization; activation of voltage dependent calcium channels, increased calcium entry, and insulin exocytosis.

ACTION OF INSULIN

Insulin metabolism takes place in the liver. The half-life of insulin in the circulation in humans is about 5 min. It binds to insulin receptors, and some are internalized. It is destroyed by proteases in the endosomes formed by the endocytotic process. The insulin receptor consists of four subunits held together by disulfide linkages.[6] Two alpha subunits that lie entirely outside the cell membrane and 2 betasubunits that penetrate through the membrane, protruding into the cell cytoplasm. The insulin binds with alpha subunits on the outside of the cell; because of the linkages with the beta subunits the portions of the beta subunits protruding into the cell become autophosphorylated.

Binding of insulin led to intrinsic tyrosine kinase activation; leads to receptor autophosphorylation and activation of intracellular- signaling molecules, insulin receptor substrates (IRS). Phosphorylation of cytoplasmic proteins and dephosphorylation reactions, promoted by IRS and adaptor proteins results various

metabolic effects like glycogen synthesis, protein synthesis, lipogenesis, and regulation of various genes in insulin-responsive cells.



ACTIONS OF INSULIN:

Immediate action consists of transport of glucose, amino acids, and potassium into insulin-sensitive cells. Intermediate action, within minutes include, inhibition of protein catabolism, stimulation of glycolytic enzymes and glycogen synthase, inhibition of phosphorylase and enzymes for gluconeogenesis.

Within hours it increase mRNAs for lipogenic and other enzymes.

EFFECT OF INSULIN ON VARIOUS TISSUES

General - Promotion of cell growth.

In Liver it reduce ketogenesis ,reduces glucose output due to decreased gluconeogenesis, promotes glycogen and protein synthesis, increased glycolysis and increased lipid synthesis.

ADIPOSE TISSUE

Insulin actions include glycerol phosphate synthesis, stimulation of triglyceride deposition**,** fatty acid synthesis, lipoprotein lipase enzyme activation**,** increased glucose entry into cells. Other actions includes hormone-sensitive lipase inhibition[7] and increased K+ uptake into cells.

MUSCLE

Insulin action includes reduces protein catabolism, increased potassium uptake, promote glucose entry into muscles, increased glycogen synthesis, decreased release of gluconeogenic amino acids**,** ketone uptake into tissues and increased protein synthesis in ribosomes[8].

Diabetes mellitus is a metabolic disorder characterized by chronic hyperglycemia along with abnormalities in carbohydrate, fat and protein metabolism due to deficiency in insulin secretion or action.

AETIOLOGICAL CLASSIFICATION OF DIABETES

TYPE1-DIABETES

Beta cell destruction leads to absolute insulin deficiency

1. Autoimmune
2. Idiopathic

TYPE2 DIABETES

1. Due to insulin resistance
2. Insulin secretory defects
3. Increased hepatic glucose production

SPECIAL TYPES

Genetic defect in beta cell dysfunction (MATURITY ONSET DIABETES OF YOUNG 1-6)

Genetic defects insulin action

1. Due to genetic disorders--like Klinefelter`s syndrome; Down's syndrome, Turner's syndrome; DIDMOAD; Friedreichs ataxia[9] , myotonic dystrophy
2. Endocrinedisorders (Acromegaly, Cushing'ssyndrome thyrotoxicosis glucagonoma; phaeochromocytoma)
3. Drug induced –steroids, diuretics -Thiazides
4. Exocrine pancreas[10] diseases like pancreatitis, neoplastic disease, cystic fibrosis, haemochromatosis, and pancreatectomy

5. Infections-virus like infection of rubella occurring via congenital transmission, mumps, coxsackievirus B

- Immune-mediated diabetes due to other causes.
- GESTATIONAL DIABETES.

AETIOPATHOGENESIS OF TYPEI DIABETES

TYPE1 DM Classified into type 1A, 1B

TYPE I A --- ISLETS autoantibody positive, associated with GAD (GLUTAMIC ACID DECARBOXYLASE) DR3,DR4

TYPE 1B ----autoantibody negative, genetics unknown

1.) GENETIC FACTORS

- Concordance of type1DM in identical twins---40%[11]
- Polymorphism HLA complex located on chromosome 6
- Most have HLA DR3, DR4Haplotype.
- Chromosome 11 mutation insulin gene located.
- Family history

2.) ENVIRONMENTAL FACTORS

Congenital rubella infection, entero viruses, rotavirus, parvovirus, coxsackie virus Toxin rodenticide, pentamidine

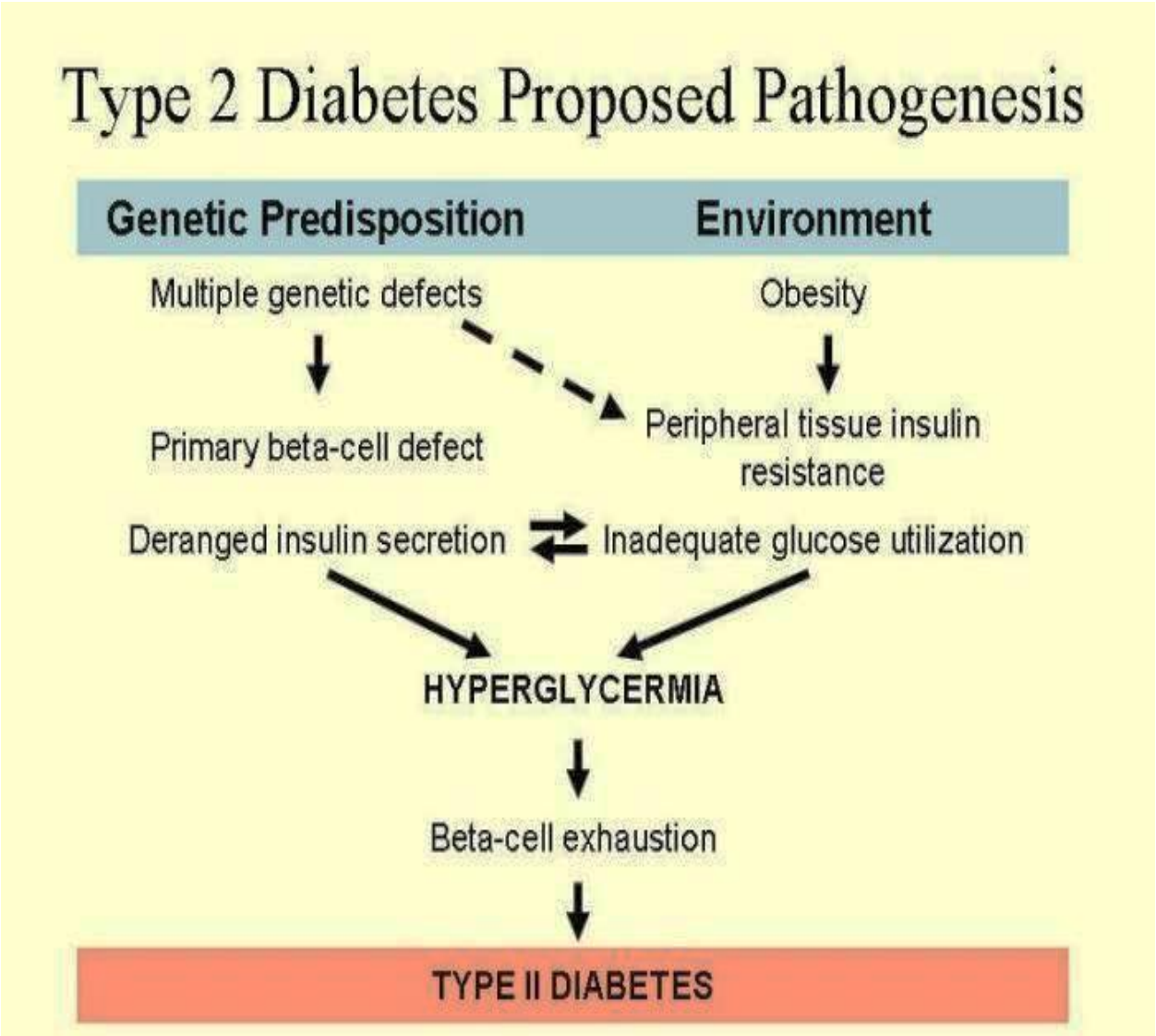
3.) Immunological factors-islet cell autoantibodies –different autoantibodies directed against islet cells includes

Eg: GAD 65 ANTIBODIES,IA-2,ICA-512,ZNT-8 ;present in 85% of new onset type1DM

PATHOPHYSIOLOGY

- ISLET CELL AUTOANTIBODIES
- ACTIVATED T LYMPHOCYTES
- RELEASE OF CYTOKINES-TNF∞,INTERFERON,IF 1

These factors leads to progressive destruction of beta cells leads to type 1 diabetes.


Type 2 Diabetes Proposed Pathogenesis
Genetic Predisposition
Environment
Multiple genetic defects
Obesity
Primary beta-cell defect
Peripheral tissue insulin resistance
Deranged insulin secretion
Inadequate glucose utilization
HYPERGLYCERMIA
Beta-cell exhaustion
TYPE II DIABETES

RISK FACTORS OF TYPE 2 DIABETES

Risk factors mainly includes genetic and environmental factors[12]

It includes genetic factors, family history, demographic characteristics, age, sex ethnicity, lifestyle and behavioral- risk factors etc. Others are physical inactivity, stress, diet, obesity BMI> 25, hypertension BP>140/90, HDL Cholesterol<35mg/dL, polycystic ovarian syndrome etc. Impaired glucose tolerance, HbA1C 5.7_6.4%, insulin resistance, pregnancy-related determinants (parity, diabetes in offspring, H/O OF Gestational Diabetes Mellitus)[13] are also included as risk factors.

PATHOPHYSIOLOGY

1) INSULIN RESISTANCE

Reduced biological response to either exogenously administered insulin or endogenously produced insulin; which is influenced by genetic factors and obesity. Insulin resistance occur in various tissues liver, muscle splanchnic system etc. In type2 DM both receptor and post receptor defects (insulin regulated phosphorylation) leads to insulin resistance. Post-binding defects are due to reduced production of insulin second messenger, decreased glucose transport into cell. In adipocytes which secrete various adipokines like leptin, free fatty acids, TNF, resistin, adiponectin, IL-6 these also influence insulin sensitivity. Adiponectin, insulin sensitizing peptide, decrease in obesity, cause increased

insulin resistance in liver. Free fatty acids reduce glucose uptake in skeletal muscle, increase glucose production in liver, and impairs beta cell function leads to insulin resistance. In muscles reduced receptor tyrosine kinase activity, reduced glucose transporters, reduced glycogen synthase play predominant role in resistance. Features of the insulin resistance syndrome includes hyperinsulinaemia type2diabetes, impaired glucose tolerance,[14] hypertension, low HDL cholesterol; elevated triglycerides, central obesity, micro albuminuria etc

2) IMPARIED INSULINSECRETION

Insulin production initially increases to compete insulin resistance and to maintain plasma glucose level normal. Secretory impairment initially mild, progressively involves glucose mediated insulin secretion. Increased glucose levels have glucotoxicity effect, inhibits insulin release from beta cells.

3) INCREASED HEPATIC GLUCOSE PRODUCTION

Insulin resistance in liver leads to hyperglycemia, decreased glycogen storage, more hepatic glucose production

4) INCRESED LIPIDSYNTHESIS

In adipocytes increased free fatty acid synthesis and lipolysis leads to lipotoxicity, which induces beta cell dysfunction.

DIABETES DIAGNOSING CRITERIA

Normal glucose tolerance	*Impaired Fasting Glucose or Impaired Glucose Tolerance*	*Diabetes* mellitus
Fasting Plasma Glucose<100 mg/dl	Fasting plasma glucose 100-125 mg/dl (IFG)	Fasting Plasma Glucose ≥126 mg/dl
2-hr PG <140 mg/Dl	2hour postprandial glucose 140 -199 mg/dl (IGT)	2 hour postprandial glucose ≥200 mg/dl
HbA1C <5.6%	5.7_6.4%	>6.5%[15]
FPG fasting plasma glucose; IFG, impaired fasting glucose; IGT, impaired glucose tolerance; PG, plasma glucose		

DIFFERENCE BETWEEN TYPE1 DIABETES AND TYPE2 DIABETES

	Type1	Type2
Typical age at onset years	<40	>50
Duration of symptoms	Weeks	Months to years
Body weight	Normal	Obese
Autoantibodies	Positive in 80%	Negative
Rapid death without treatment with insulin	Yes	No
Family history	Uncommon	Usually common
Ketonuria	Yes	Nil
Complications	No	More

COMPLICATIONS OF DIABETES

Diabetes has acute and chronic complications. Acute complications include diabetic ketoacidosis and hyperglycemic hyperosmolar state. Chronic complications of diabetes associated with more mortality of disease.it is classified into vascular and non-vascular complication. Chronic micro vascular

complications include diabetic neuropathy, nephropathy, and retinopathy. Macrovascular complications stroke, coronary artery heart disease, peripheral occlusive vascular disease[16].Other non-vascular complications include gastro paresis, infections, sexual dysfunction, and periodontal disease. Retinopathy includes proliferative or non proliferative, macular edema, cataract etc. Neuropathy includes peripheral neuropathy, motor weakness, autonomic neuropathy etc. Cause of death in diabetes 70% mortality due to coronary artery disease[17], 10% due to renal failure, 6% infections,10% cancer,1% DKA,5% others. Risk factors for more mortality in diabetes depends on duration of diabetes, micro albuminuria[18], onset, obesity, associated hypertension, dyslipidemia, HbA1C etc.

INFECTIONS

Diabetic patients are susceptible to common infections[19]. Inadequate glycemic control is a factor responsible for infections .Increase in blood glucose causes the growth and colonization of different organisms. Urinary infections, and soft tissue infections, lower respiratory infections occur in diabetes. Patients with diabetes bacteriuria common. Urinary tract infections and pyelonephritis are caused by *Escherichia coli*, and *Candida*. Diabetic patients are prone to postoperative wound infections.

Diabetes are prone to furunculosis, superficial candida infections, and vulvovaginitis, skinfolds and nasal infection. Acute complications of diabetes include Diabetic ketoacidosis and hyperglycemic hyperosmolar state.

INVESTIGATIONS OF DIABETES

Blood glucose

Urinalysis

- Analyses for glucose, ketones, albumin (both macro- and micro albuminuria)
- Macro albuminuria –urine albumin>300 mg/day.
- Urine albumin creatinine ratio 2.5-30 mg/mmol if microalbuminuria present.

Biochemistry include RBS, lipid profile and renal, liver and thyroid function.

- Glycemic control assessment **via** Glycated hemoglobin **(HbA1c)**

GLYCATED HAEMOGLOBIN (HBA1C)

Objective measurement of diabetic glycemic control over a period of weeks to months is given. In diabetes; the slow non enzymatic covalent attachment of glucose to hemoglobin raises the amount in the HbA1c fraction. Because erythrocytes got life span of 120 days; HbA1c gives glycemic control over 2-3

months. These fractions can be separated by chromatography.The rate of formation of HbA1cs depending on blood glucose concentration; for 1% increase in HbA1C indicate average rise of blood glucose value 36 mg/dl. ADA recommends twice per year measurement of for optimal glycemic control. Anemia, hemoglobinopathies, reticulocytosis, blood transfusion will interfere with result.[20]

MANAGEMENT OF DIABETES

It includes assessment of patients with history, clinical examination, and assessment of complications, investigation and medical management.

History includes durations of diabetes, life style issues assessment like exercise, stress, smoking, alcohol, and other comorbidities.

Clinical examination include body weight, body mass index, vitals and focus of infections etc. In diabetes target blood pressure should be less than 140/90mm hg[21].

Assess for dehydration– dry mouth, decreased tissue turgor if there is suspecting DKA.

Eye examination should be done yearly .Foot examination to assess peripheral pulses, sensation.

CNS examinations for patients with peripheral neuropathy, diabetic amyotrophy[22].

It includes sensory, motor system examinations.

Cardiac and renal evaluation yearly for detecting complications

Life style modification which includes primary and secondary prevention.

DIET control

Hypocaloric diet that is low-fat or low-carbohydrate, high protein

CARBOHYDRATE 45-50%

FAT-<30% SATURATED<7%

PROTEINS 20%

Reduced trans-fatty acids, diet with low glycemic index is recommended.

Exercise the ADA recommends 150 min/week of moderate exercise activity[23].Exercise benefits includes lowering plasma glucose and increasing insulin sensitivity. Lifestyle optimization and education are essential for all patients with diabetes. Lifestyle modification designed for weight loss, including exercise medical and surgical interventions approved for obesity, should be considered as primary approaches for therapeutic benefits in obese patients with diabetes, and for prevention of diabetes in high risk patients with prediabetes.

Avoidance smoking, addictions

TREATMENT OF ASSOSIATED CONDITIONS like-

Hypertension, dyslipidemia, obesity, heart disease.

MEDICAL MANAGEMENT OF TYPE 1 DM--Insulin administration

MEDICAL MANAGEMENT TYPE 2DM

Insulin, Oral anti diabetic drugs

Antidiabetic drugs

ORAL DRUGS

- **Biguanides**

Decrease hepatic glucose production, decrease gastrointestinal glucose absorption, and increase target cell insulin sensitivity

Example: Metformin

- **INSULIN SECRETAGOGUES (increase insulin secretion)**

1) Sulfonylureas

Examples: glyburide, glipizide, glimepiride, glibenclamide, tolbutamide

2) Meglitinide derivatives (Non sulfonylureas)

Eg. Repaglinide, nataglinide

3) Di peptidyl peptidase IV (DPP-4) inhibitors[24]

Eg. Saxagliptin, sitagliptin, vildagliptin

- Others included.
- Alpha-glucosidase inhibitors—Decrease GIT glucose absorption

  Eg. Acarbose, Meglitol

- Thiazolidinediones [25] (TZDs) –decrease insulin resistance, increased glucose utilization

  Eg ; Rosiglitazones, Pioglitazones

- Selective sodium-glucose transporter-2 (SGLT-2) inhibitors reduce glucose reabsorption

  Eg: Dapagliflozin

PARENTERAL DRUGS

- Amylin agonists[26] -slow gastric emptying, reduce glucagon eg. Pramlintide
- Glucagonlike peptide–1 (GLP-1) agonists(parenteral preparations)

  Eg. Exenatide, liraglutide

INSULIN PEPARATIONS

SHORT ACTING- Aspart, lispro, Regular, glulisine Duration of action 3-4 HOURS

Long acting --Detemir, glargine, NPH -Duration of action upto 24hrs

Combinations

1. 75/25 --75% protamine lispro, 25%lispro
2. 70/30 ---70% protamine,30% aspart
3. 50/50—50% protamine,50% lispro
4. 70/30 –70% NPH, 30% regular

DIABETIC TREATMENT GOALS

- Glycemic control - Pre-prandial capillary plasma glucose 70–130 mg/dl
- Peak postprandial capillary plasma glucose<180 mg/dl
- HB A1C should be less than7.0%
- Blood pressure_--- less than130/80
- **LIPID PROFILE**
    1. Triglycerides less than1.7 mmol/l (150 mg/dl)
    2. Low-density lipoprotein less than 100 mg/dl
    3. High-density lipoprotein--- 40 mg/dl

DIABETICKETOACIDOSIS

Diabetic ketoacidosis is the most common and serious acute complication of diabetes. DKA usually occurs patients with type 1 DM, it can also occur in patients with type 2 DM. Type 2 DM patients with prolonged duration are more prone to DKA. Its incidence is between 4 to 8.0 per 1000 diabetic patients.[27]

DKA mostly precipitated by stressful conditions, such as infections, trauma.

DKA consists ketonemia, hyperglycemia, acidemia.

Hormonal imbalance leads to hepatic gluconeogenesis and glycogenolysis result in hyperglycemia. Enhanced lipolysis leads to free fatty acids production that results increased ketone bodies, and metabolic acidosis.

Symptoms include nausea, vomiting, increased thirst, polyuria, abdominal pain, breathlessness, altered sensorium etc.

Physical findings

- Fruity smell of breath
- Tachycardia,Dehydration,Hypotension
- Tachypnea, Kussmauls respirations[28]- rapid deep breathing
- Abdominal tenderness
- Lethargy, somnolence, coma,seizure

PRECIPITATING FACTORS FOR DKA

1) Inadequate insulin,

   Insulin omission,

   Noncompliance to drugs,

   Insulin pump failure (20-40%),

2) Infection 30-40%

   Sepsis

   Pneumonia, urinary tract infection, gastroenteritis

3) Myocardial infarction[29], stroke 6%

4) Unrecognized symptoms of newly detected diabetes --8%

5) Drugs. _10%

   Steroids, glucagon

   Diuretics, sympathomimmetics

6) Alcohol

7) Hypervolemia

8) Non adherence to insulin treatment plans:

   Financial problems,

9) Psychological factors

10) Pregnancy

11) Unknown

PATHOPHYSIOLOGY

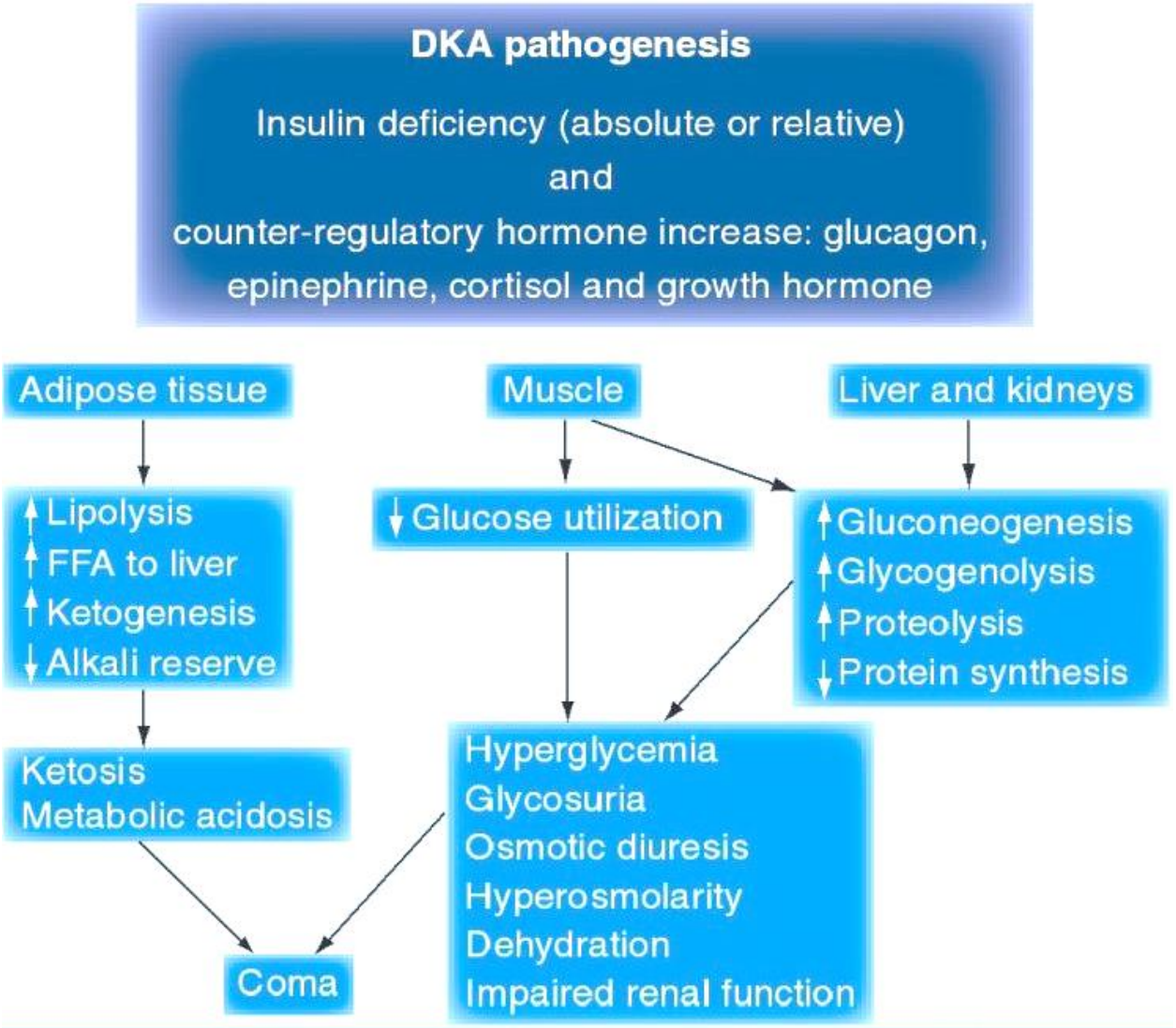
Normal metabolic homeostasis is maintained by balance between the anabolic action of insulin and catabolic effect of counter regulatory hormones.

Diabetic ketoacidosis is caused by insulin deficiency with elevated counterregulatory hormone (glucagon, catecholamine, cortisol, and growth hormone). Insulin deficiency can leads to increased glucose production via glycogenolysis, gluconeogenesis, and ketone body and adipocytes formation in the liver, increases formation of free fatty acids.

Defective insulin secretion and increased blood glucose levels decreases the hepatic fructose-2,6-bisphosphate, which changes the activity of phosphofructo kinase and fructose 1,6 bisphosphatase[30]. Hyper glucogonemia reduces the action of pyruvate kinase and insulin deficiency increases the activity of phosphoenolpyruvatecarboxykinase. These leads to glucose synthesis from substrate pyruvate and divert away from glycolysis. Insulin deficiency decreases glucose transport via reducing GLUT4 levels, which leads reduced glucose intake into skeletal muscle and which can reduces metabolism of glucose takes place inside cells.

Reduced insulin levels, and increased counter regulatory hormones, increase lipolysis and the release of free fatty acids.IN DKA, hyperglucagonemia

increase ketone body formation, through activation of the enzyme carnitine palmitoyltransferase I. This enzyme needed for fattyacid uptake into the mitochondria-where beta-oxidation and ketone bodies synthesis taken. Increased lactic acid synthesis lead to the acidosis. More free fatty acids lead to triglyceride and VLDL production.



DIAGNOSTIC CRITERIA FOR DKA

	Mild	**Moderate**	**severe**
Arterial PH	7.24 to 7.30	7.00 to < 7.24	< 7.00
Effective serum osmolality	Variable	Variable	Variable
Mental status	Alert	Alert/drowsy	Stupor/coma
Serum bicarbonate(meq/li)	15 to 18	10 to 15	< 10 mEq /l
Ketone (serum)	Positive	Positive	Positive
Urine ketone†	Positive	Positive	Positive
Anion gap (mEq/lit)	> 10	> 12 mEq /li	> 12 mEq /l

Anion Gap = (serum Na + K) – (Cl- + HCO3-)].

INVESTIGATIONS

1) Blood Glucose-250–600 mg/dl
2) Serum ketone level 7-10mmol/lit
3) Serum potassium levels; it can be normal, high or low.
4) In DKA if initial potassium values high, further treatment with insulin reduces potassium levels.
5) Serum magnesium- measurement it can be low or normal
6) Serum phosphate- normal or elevated
7) Serum sodium - low or normal
8) Serum chloride- Normal
9) Serum osmolality_ more than 320 mOsm per kg
10) Serum bicarbonate _< 15 mEq/L
11) RFT--urea, creatinine level; It can be increased due to dehydration and reduced renal perfusion[31]
12) Complete blood count for infections, sepsis.
13) ECG TO rule out MI or to find out electrolyte abnormalities.
14) HbA1C LEVELS
15) ARTERIAL BLOOD GAS

    Arterial blood gas measurement ---PH of arterial blood between 6.8 - 7.3;

    Arterial PCO2 values20–30mmHg;

Aniongap_usuallymorethan15mEq/L

16) **URINE TEST** for albumin, sugar, deposits and ketone should be done.
17) Chest radiography can be taken if pneumonia or chronic obstructive pulmonary disease suspected.
18) Serum amylase OR lipase can be done if suspected pancreatitis[32]
19) Liver function test should be done in patients with alcoholism; and suspected fatty liver disease.
20) Infection is suspected do urine and blood Cultures to identify organism.

DIFFERENTIALDIAGNOSIS

Hyperosmolar hyperglycemic state, Stress hyperglycemia

Gastroenteritis, Pancreatitis

Ketosis due to starvation, lactic acidosis, Hyperchloremic acidosis

Salicylate poisoning; uremic acidosis

High Aniongap metabolic acidosis present in alcoholic ketosis, ethylene glycol poisoning

HYPERGLYCEMIC HYPEROSMOLAR STATE

HHS consists of hyperglycemia, hyperosmolarity, severe dehydration and neurological manifestations like lethargy, coma [33] .It can be precipitated by infections, trauma, immunosuppressive agents, drugs like steroids, alcoholism stroke etc. It can be prolonged over weeks with presenting symptoms of polyuria, polydipsia, weight loss, confusion, and coma.

Investigations for patients with hyperosmolar hyperglycemic patients

INVESTIGATIONS	HHS
GLUCOSE	600-1200mg/dl
SODIUM	135-145meq/l
POTASSIUM,CHLORIDE,MAGNESIUM	Normal
PLASMA KETONES	+/_
S BICARBONATE	Normal
OSMOLARITY	330-380mosm/ml
ARTERIAL PH	7.3
ARTERIAL PCO2	Normal
ANION GAP	Normal
Creatinine	Increased

Management of HHS consists of fluids replacement, correction of precipitating factors, correction of osmolarity, low dose insulin and correction of electrolytes.

TREATMENT OF DKA

It includes adequate fluid replacement, insulin infusion, correction of hyperglycemia and electrolyte abnormalities.

Initial assessment to determine severity of acidosis include the following .If one or more factors present then consider severe DKA.

It includes

- Pulse high 100 or below 60 per minute
- Serum bicarbonate (HCO3) levels lessthan5mmol/l
- SPO2 below 92% on air
- Systolic BP below 90 mmHg
- PH OF arterial blood<7.1
- Blood ketones over 6 mmol/L
- Glasgow coma scale less than 12
- Anion gap above 16.

New principles in treatment of DKA

According to weight of patient, insulin infusion rate is calculated. Insulin dose adjustment; calculated via body weight allows insulin resistant states to be accommodated. . If blood ketone measurements are available the response of the

insulin regimen is determined by the rate of reduction of the ketones and can do dose adjustment if this is inadequate. During the first 6 hours, the bicarbonate level can be used for assessment but may be less reliable thereafter. It can be used only when glucose levels are relatively normal. Start a fixed rate insulin infusion only after initiation of fluid therapy[34].

Establish monitoring regime appropriate to patient; generally hourly blood glucose and ketone measurement; with at least 2 hourly potassium levels for the first six hours to be needed.

Initial management

- Assessment Airway, Breathing, Circulation.
- Then insert iv cannulae and start of iv fluids.
- Check vitals respiratory rate, blood pressure, pulse, temperature
- Respiratory rate; patient can have rapid deep breathing
- Temperature high in the presence of sepsis or infection
- Blood pressure it can be hypotension or normal
- Pulse tachycardia in sepsis, infection
- Oxygen saturation
- Insertion of nasogastric tube for airway protection
- Do clinical examination including system examination for identifying complications.

- Initial blood tests should be done as mentioned above, do monitoring of cardiac activity, SPO2 monitoring .Search for causes of DKA and treat causes.

FLUID REPLACEMENT

Immediate management

First one hours

Start intravenous fluids. Total water deficit for patients with DKA is approximately 7% to9% weight of the body[35]. Hypotension occurs due to a loss of morethan 10 percentage of body fluids. Initially, correction of dehydration using isotonic 0.9%saline can be given. The first liter can be given with in half hours and followed by iv fluids at a rate of 1L/hr; till the volume deficit is corrected. If systolic BP<90 mmHg give 500ml 0.9% normal saline over 10-15 min. then reassess ,if blood pressure remains <90mmHg seek senior review .Hypotonic(0.45%) saline solution considered for severe hypernatremia; serum sodium >155 mEq/L. If no improvement search other causes of hypotension treat it.

Systolic BP on admission >90 mmHg give0.9%normal saline 1lit over 60 min

THE NEXT step - REPLENISH TOTAL BODY WATER DEFICITS;

0.9% normal saline at a 150 to 500ml/hour rate is appropriate serum sodium is normal; Assess cardiac and renal status ,the degree of dehydration for

calculating rate of the fluid replacement Assessment of fluids replacement done by improvement in blood pressure, measurement of fluid balance, and clinical examination. IN a typical DKA patient complete fluid replacement take12 to24 hours.

INTRA-VENOUS INSULIN INFUSION

Initial bolus dose of regular insulin 0.1 unit/kg can be given; followed by rate of 0.1unit/kg/hour insulin infusion can be given.

Start continuous fixed rate via an infusion pump. 50units human soluble insulin diluted in 50ml with 0.9%sodium chloride solution give via infusion pump.

Regular insulin; 50units in 500 mL 0.9% saline, infused at a rate of 5 units per hour of insulin can be started.

Reduction in glucose level of 50 --75 mg/dl/hour is an adequate response[36]; Insulin can increase progressively by 50 to100% until an adequate glycemic response is obtained. Increase intravenous insulin by 50% if blood glucose change <50mg/dl/hr. Decrease insulin infusion by 50% if blood glucose decreases by >100 mg/dl/hr in any one hour.

Maintenance insulin infusion rates of 1 to2 unitsor higher and the anion gap has closed .When blood glucose reaches lessthan 250 mg/dl add 5%dextrose[37] to IV fluids. If the patient started to take oral feeds or sensorium becomes improved change insulin to subcutaneous route and the parenteral route can be stopped. Before stopping the intravenous insulin, give the first SC dose of insulin

injection. Rapid correction of glucose levels at a rate >100 mg/dl can cause osmotic encephalopathy.

Monitoring regime include generally hourly blood glucose measurement, ketone measurement, with at least 2 hourly potassium and other electrolytes for the first six hours. Potassium is often high but falls rapidly with insulin infusion. Regular monitoring is mandatory

MANAGEMENT 60 minutes to 12 hours

Give 0.9% sodium chloride 1000ml over first hour; followed by 0.9% sodium chloride 1Litres over next 2 hours.Repeat0.9% sodium chloride 1Litres over next 2 hours .Next 4 hours give 1000ml normal saline slowly. Repeat 1litres over next 4 hours. Then give 0.9% sodium chloride 1L1000ml over next 6 hours. Add potassium chloride to this solution if indicated

Adjust KCL Infusion

Plasma potassium(mmol/l)	Potassium needed(mmol per litre of infusion)
>5.5	Not needed
between3.5-- 5.5	40mmol/lit
<3.5	Additional potassium

Assessment of cardiac status at 12 hours should be done. Patients with heart disease/ renal failure, elderly, pregnant patients need monitoring for fluid

replacement. Fluids should be replaced under caution; monitored via the central venous pressure measurements.

Average loss of fluid and electrolytes in adult diabetic ketoacidosis

Water- 6litres; Sodium: 500 mmol ; Chloride-400 mmol Potassium: 350 mmol 3L extra cellular[38] can be replaced with saline; 3L intracellular replace with dextrose commence a fixed rate12 -24 hours.

Ketonemia and acidosis should have resolved. Adequate ketone reduction is 0.5mmol/L/hr. If it is not available; HCO3 should rise by 3mmol/litre/hour and blood glucose should fall by 3 mmol/litres/hour.

If patient is not taking oral feeds continue iv insulin infusion at a lower rate of 2-3u/hour and continue iv fluid replacement.

If ketoacidosis resolved patient taking oral feeds then change to subcutaneous insulin.

BICARBONATE REPLACEMENT IF PH>7 no bicarbonate needed, if it is<6.9 give 100mmol sodium bicarbonate in 400ml saline at a rate of 200ml/hour infusion.

ADDITIONAL PROCEDURES

Insertion of urinary catheter if reduction in urine output.

Insertion of Ryle's tube if patient persistently vomiting or having altered sensorium. Oxygen administration -SPO2<80MmHg.

Repeat chest radiography.

Maintain intake/output chart, minimum urine output should be 0.5ml/kg/hr.

Cardiac monitoring should be done; if DKA is severe.

Serious complications of DKA and its treatment

1) **HYPONATREMIA can be corrected by 3% hypertonic saline.**

2) **HYPOKALAEMIA AND HYPERKALAEMIA**

Hypokalemia and hyperkalemia are potentially life threatening conditions in the management of DKA[39]. Due to increased risk of acute prerenal failure associated with severe dehydration and it is therefore recommended that no potassium needed if the serum potassium level remains above 5.5 mmol/L. If the serum potassium level falls below 3.5mmol/l the potassium regimen needs review.

3) **HYPOGLYCAEMIA**

Severe hypoglycemia can cause cardiac arrhythmias, acute brain injury and death. Rapid blood glucose reduction can cause rebound ketoacidosis. Once the blood glucose falls to 14mmol/L give 10% dextrose.

4) **CEREBRAL EDEMA[40]**

Observed more in children than adults. Patients with cerebral edema can have symptoms of raised intra cranial pressure like sudden deterioration in mental status, headache, altered mental status, papilledema edema. Treatment includes IV mannitol, oral glycerol, head end elevation, hyperventilation .CT BRAIN can establish the diagnosis.

5) Metabolic acidosis and Lactic acidosis - Adequate volume replacement, and correction of bicarbonate, sepsis control will improve acidosis.
6) Acute renal failure
7) Aspirationpneumonitis
8) Pulmonaryedema
9) Adult respiratorydistress syndrome
10) disseminated intravascular coagulation, basilar artery thrombosis
11) Venous thrombosis and Arterial thrombosis
12) Pneumothorax, pneumomediastinum and subcutaneous emphysema
13) Rhabdomyolysis.

Prevention of DKA.

DKA occur due to lack of clinical communication[41].Insulin omission can be prevented by medical education, psychosocial assessment and treatment along with supervision of insulin administration. Educate patients with what are risk factors, symptoms of DKA so they can identify DKA of early onset.

MATERIALS AND METHODS

This study was an observational cross sectional study conducted in Tirunelveli Medical College, Department of Medicine, during the period from of August 2014 to August 2015. The aim was to study the symptomatology, risk factors and prognosis of Diabetic ketoacidosis. The necessary clearances from the concerned departments and the ethical committee was obtained prior to the start of the study.

All patients admitted to the Intensive medical Care Unit in whom a diagnosis of DKA was made based on clinical features, with diabetes and elevated random blood sugar, urine acetone positive were considered for the study. Patients were included in the study based on inclusion and exclusion criteria.

INCLUSION CRITERIA

1) Patients with type1, and type2 diabetes with high blood sugar, urine acetone positive

2) All type2 diabetic patients under stressful conditions like stroke, MI, sepsis

3) Patients with high blood sugar values on irregular medication

EXCLUSION CRITERIA

Terminally ill patients (more than one comorbidities).

Sepsis is identified by SIRS [42] criteria with pulse rate, temperature, respiratory rate. Blood culture and urine culture was done to identifying organisms.

An oral consent was taken from all patients for a detailed clinical history and examination and the required laboratory investigations. The details were collected from each patient was entered in the proforma. (Annexure-1).

The details of the patients regarding age, sex, presenting symptoms, risk factors like non-compliance, infections, other drugs were recorded. The vital signs like pulse and blood pressure, respiratory rate, temperature were recorded and Body mass index was calculated. All the patients were subjected to routine laboratory investigations like CBC, urine analysis, urine ketones, RBS, RFT, electrolytes. Arterial blood gas analysis was done for acidosis. Blood culture, urine culture was done for those patients with sepsis. Chest X- ray, ECG, USG abdomen was done according to patient symptoms. Continuous monitoring of RBS, electrolytes was done. Patient was treated with adequate IV fluids, and insulin infusion. Precipitating factors was identified and treated.

URINE ACETONE done by ROTHERA´S TEST[43]

PRINCIPLE - Nitroprusside in alkaline medium reacts with a ketone group to form a purple ring. It is given by acetone and acetoacetone, but not by Beta hydroxyl butyric acid. On boiling acetoacetate is converted to acetone and does not give this test positive.

PROCEDURE

Saturate 5 ml of urine with solid ammonium sulphate and add 0.5 ml of freshly prepared sodiumnitroprusside 5%. Mix well and add liquor ammonia from side of tube. A purple ring at the junction of liquids indicates the presence of ketone bodies.

ARTERIAL BLOOD GAS ANALYSIS

Procedure-Site-blood can be taken from arteries like radial artery/femoral artery and brachial artery. Pre heparinized syringes needed[44]-- Syringes flushed with 0.5ml of 1:1000 Heparin solution and emptied. More than 50% blood needed; ensure no air bubbles with in the syringe. Syringe should be transported as soon as to the laboratory for early interpretation via cold chain.

Principle of ABG Henderson---Hasselbalch equation[45]

$$\text{pH value}=6.1+\log\left[\frac{HCO3-}{0.03XPCO2}\right]$$

NORMAL VALUES OF ABG

pH values between 7.35 to 7.45;pCO2 between 35 to45 mmHg, pO2 between 72to104 mmHg, bicarbonate values between 22 to 30 mEq/l anion gap ranges from 8to16 mEq/l. ANION GAP= (Na+k)– (HCO3+ Chloride) mEq/l

INTERPRETATION

- pH Less than 7.35 acidosis; more than 7.45 alkalosis
- If pH and pCO2 decreases or pH and pCO2 increases in the same direction,it should be metabolic.
- If pH and pCO2 changes in the opposite direction it should be respiratory

METABOLIC ACIDOSIS **pCO**2=1.5x(HCO3)+8±2

For every 1 mmol/l decrease in HCO3 pCO2 falls by 1.25 mmHg

METABOLIC ALKALOSIS --for every 1 mmol/l rise in HCO3 pCO2 should be increased by 0.7 mmHg[46].In metabolic acidosis anion gap should be calculated

Statistical analysis

The information collected regarding all the selected cases were recorded in a Master Chart. Data analysis was done with the help of computer using Epidemiological Information Package (EPI 2010) developed by Centre for Disease Control, Atlanta, USA

OBSERVATION AND RESULTS

100 patients admitted with clinical features, raised blood sugar values, urine acetone positive were selected.

1) **SEX DISTRIBUTION(table1)**

Sex	**Cases**	
	No	**%**
Male	58	58
Female	42	42
Total	**100**	**100**

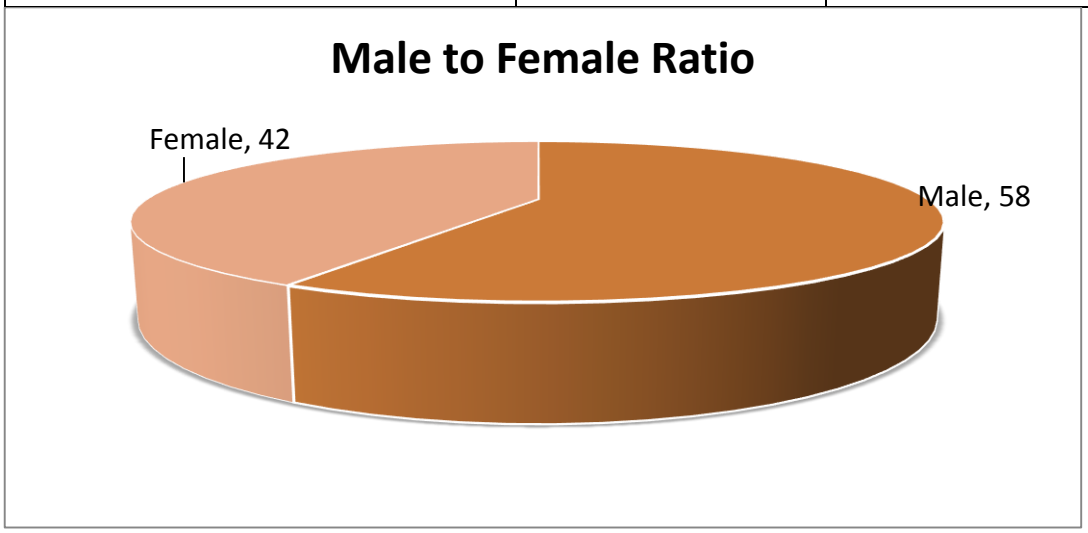


Figure 1 MALE TO FEMALE RATIO. Among 100 patients 58 males and 42 females, so males were commonly affected.

2) RATIO OF TYPE I TO TYPE2 DIABETES IN DKA(table2)

Among 100 patients 30 had type 1 diabetes, 70 had type2 diabetes. 9 patients were new onset diabetes. New onset type 2 were 8 patients, 1 patient had new onset type1.

TYPE OF DIABETES(INCLUDING NEW ONSET)	NO OF CASES	PERCENTAGE
TYPE 1	30	30%
TYPE2	70	70%
TOTAL	100	100

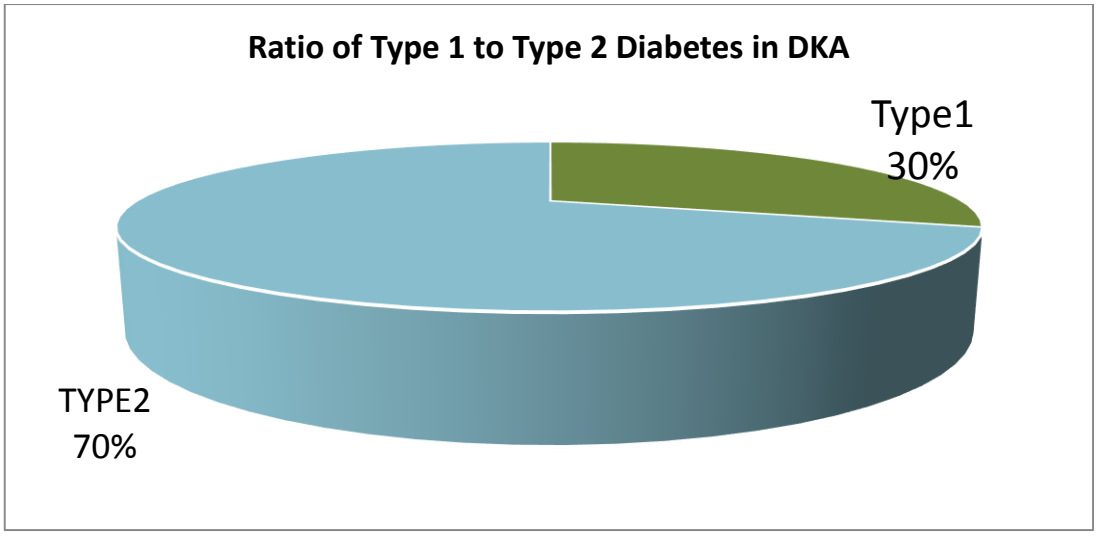
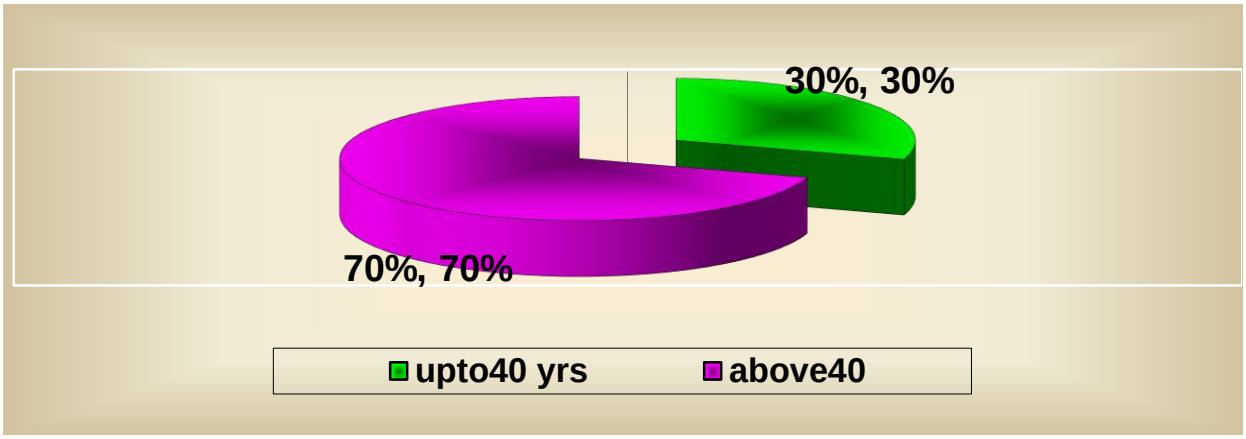


FIGURE 2 RATIO OF TYPE 1 TO TYPE2

3) AGE DISTRIBUTION(TABLE3)

Age	No	%
Upto 40 years	30	30
41 – 50 years	11	11
51 – 60 years	29	29
61 – 70 years	19	19
Above 70 years	11	11
Total	100	100
Younger group	30	30
Older group	70	70
Range	14-82yrs	
Mean	47.2 yrs	



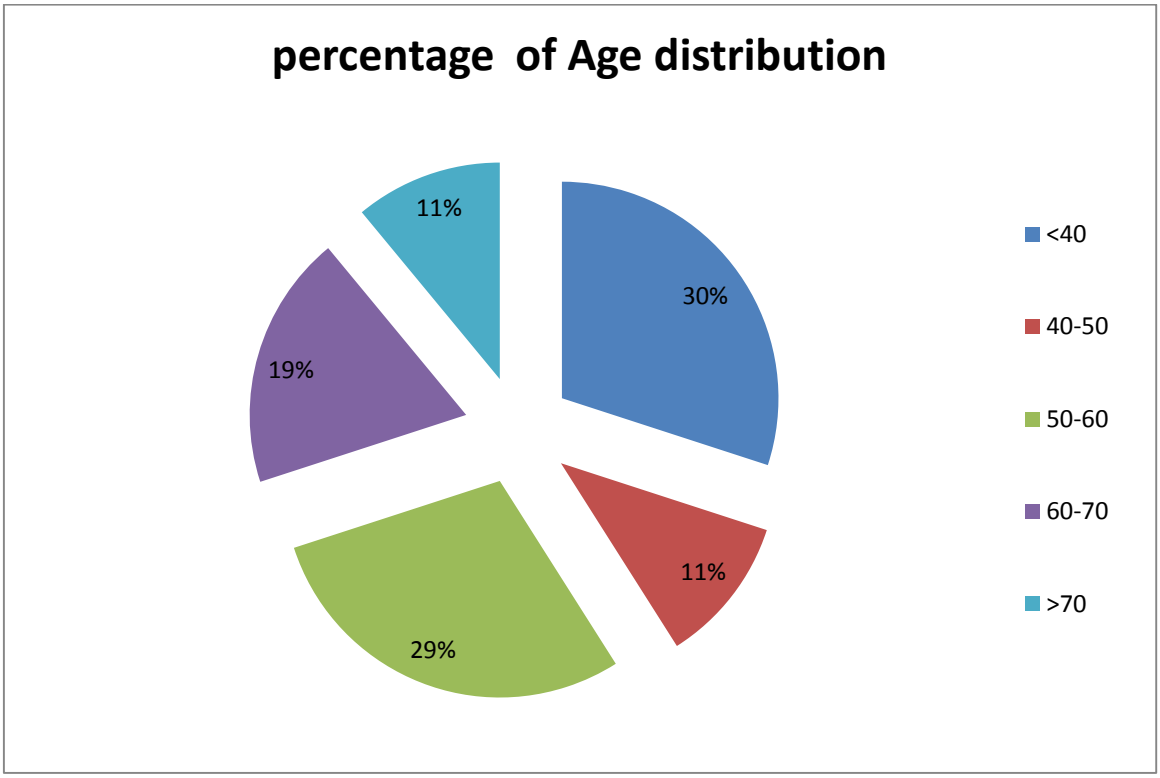


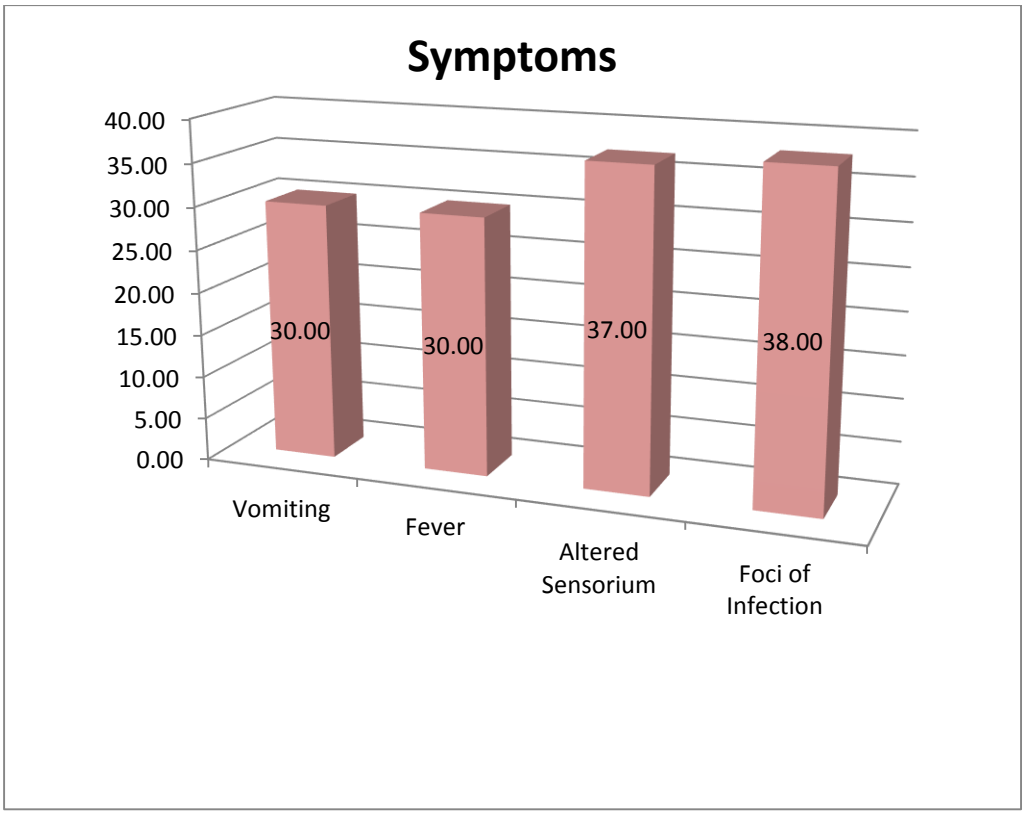
FIGURE 3) AGE DISTRIBUTION

In the study the youngest patient was a 14 year old male and the oldest patient was a 82 year old male.30 cases were younger than 40 years, mostly type 1 patients. 70 cases were above 40 years with type2 patients. The mean age group was 47.2 years. Among type 2 diabetes patients DKA mostly seen among age between 51-60 yrs.

4. PRESENTING SYMPTOMS(TABLE4)

symptoms	**NO OF CASES**
vomiting	30
fever	30
altered sensorium	37
foci of infection	38
total	**100***
*Some had multiple symptoms.	

figure4 –SYMPTOMS



Symptoms includes fever, altered sensorium[47], vomiting, foci of infections like, soft tissues infection, dysuria, cough, loose stools. Among that 38% had focus of infection like symptoms. 37 patients had altered sensorium, 30 patients had fever as symptom

5. TYPES OF FOCUS OF INFECTION(table5)

In this study 38% had symptoms like focus of infection. Among that 13% have urinary tract infection, 10% had soft tissue infections,7% had lower respiratory tract infections.8% had acute diarrheal illness.

Focus of infection	No of cases
Urinary tract infection	13
LRI	7
Soft tissue infections	10
Acute gastroenteritis/ADD	8
total	38

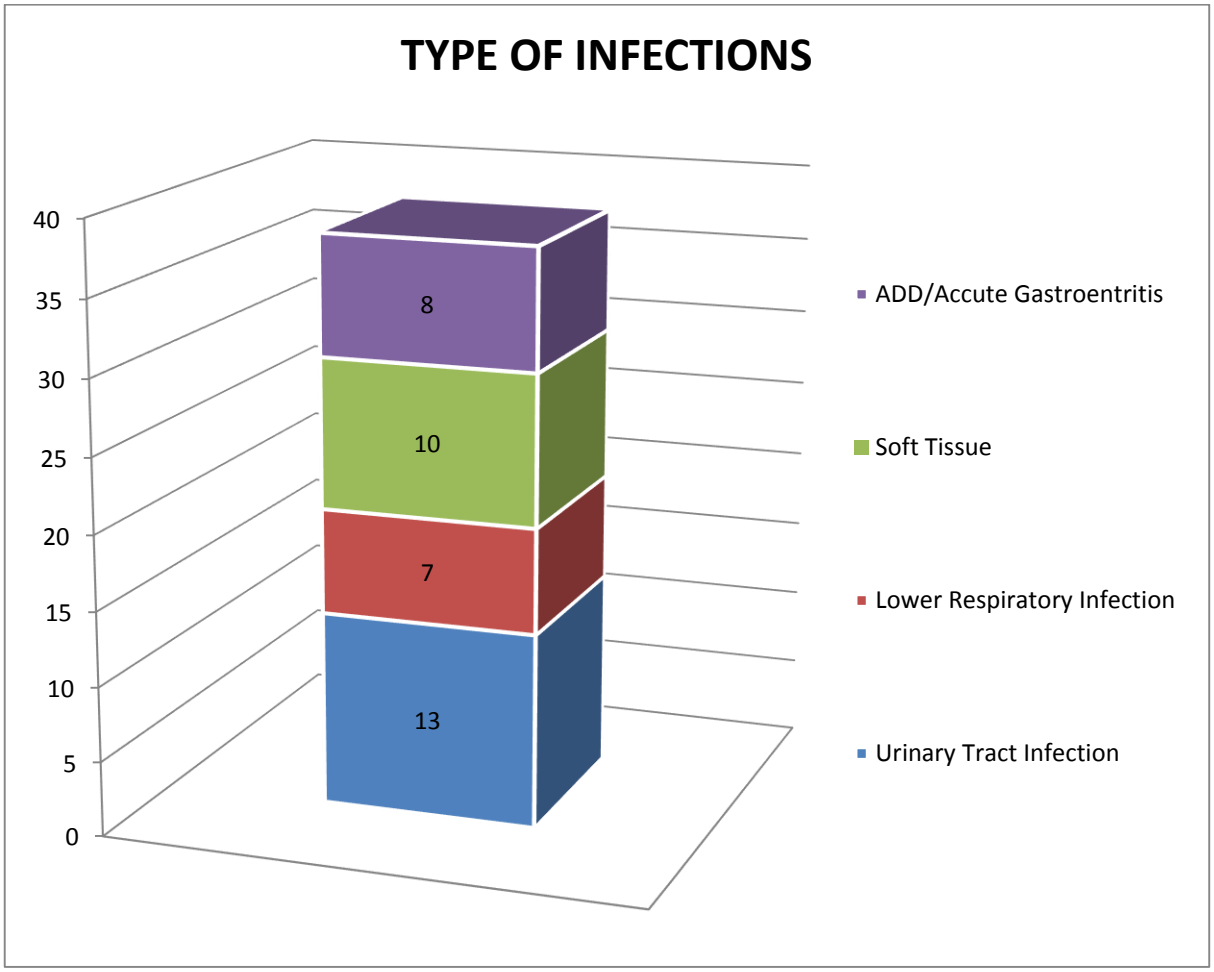


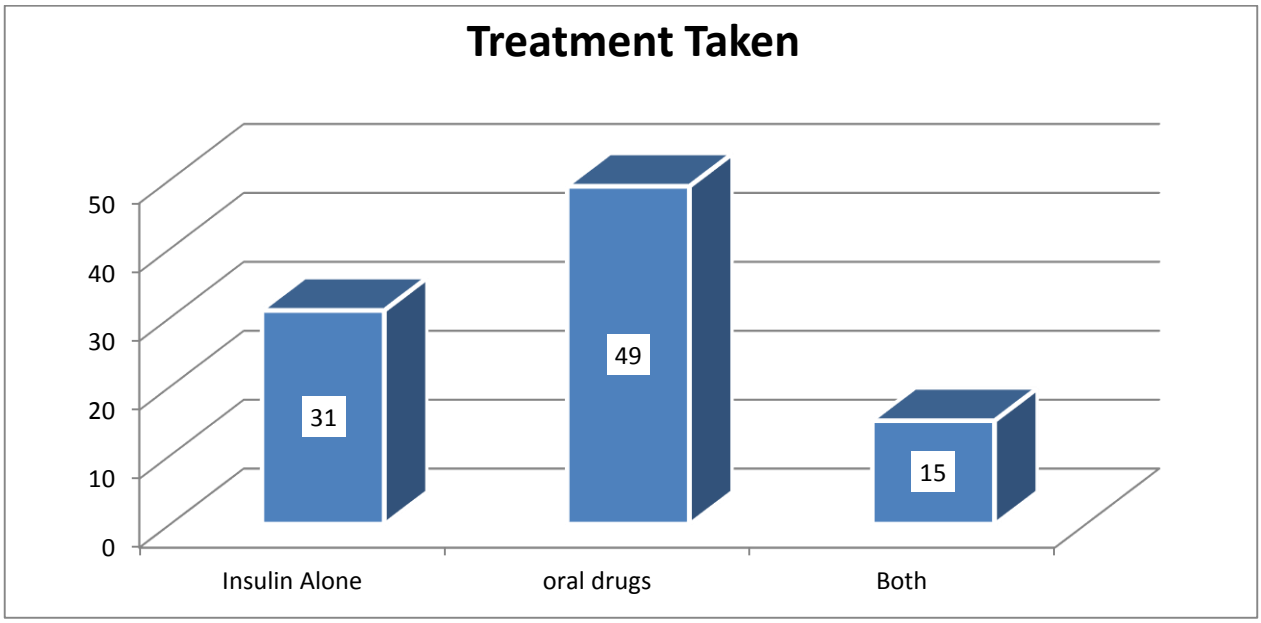
FIGURE 5- TYPE OF INFECTIONS

6. TREATMENT TAKEN (table6)

TYPE OF TREATMENT	NO OF PATIENTS
INSULIN	31
BOTH	15
Oral antidiabetic drugs	49
total	100*

*some patient were on insulin and oral drugs

FIGURE 6 TYPE OF TREATMENT



Among 70 patients with type2 diabetes 49 were taking oral anti diabetic drugs , both insulin and Oral drugs taken by 15patients . 30 type1 patients were insulin only. New onset diabetes treatment cannot assessed.

7. TREATMENT COMPLIANCE (table7)

Among diabetes patients most of them were not on treatment regularly. In this study 48% DKA patients were not taking treatment regularly. Diabetes of newonset included in compliance.

TREATMENT COMPLIANCE	NO OF PATIENTS
COMPLIANCE	52
NONCOMPLIANCE	48
TOTAL	100*

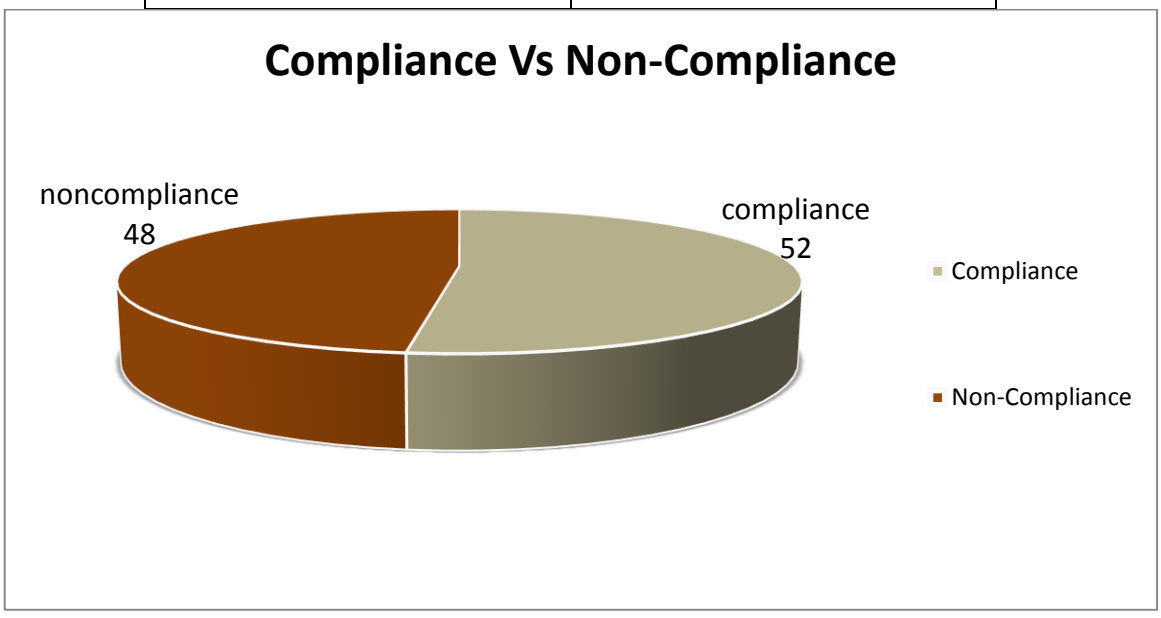


FIGURE 7 COMPLIANCE VS NONCOMPLIANCE

8. ASSOCIATED COMORBIDITIES (table8)

ASSOSIATED COMORBIDITIES	NO OF PATIENTS
coronary artery disease	12
chronic kidney disease	9
COPD	10
Stroke(CVA)	7
HYPERTENSION	2
total	40

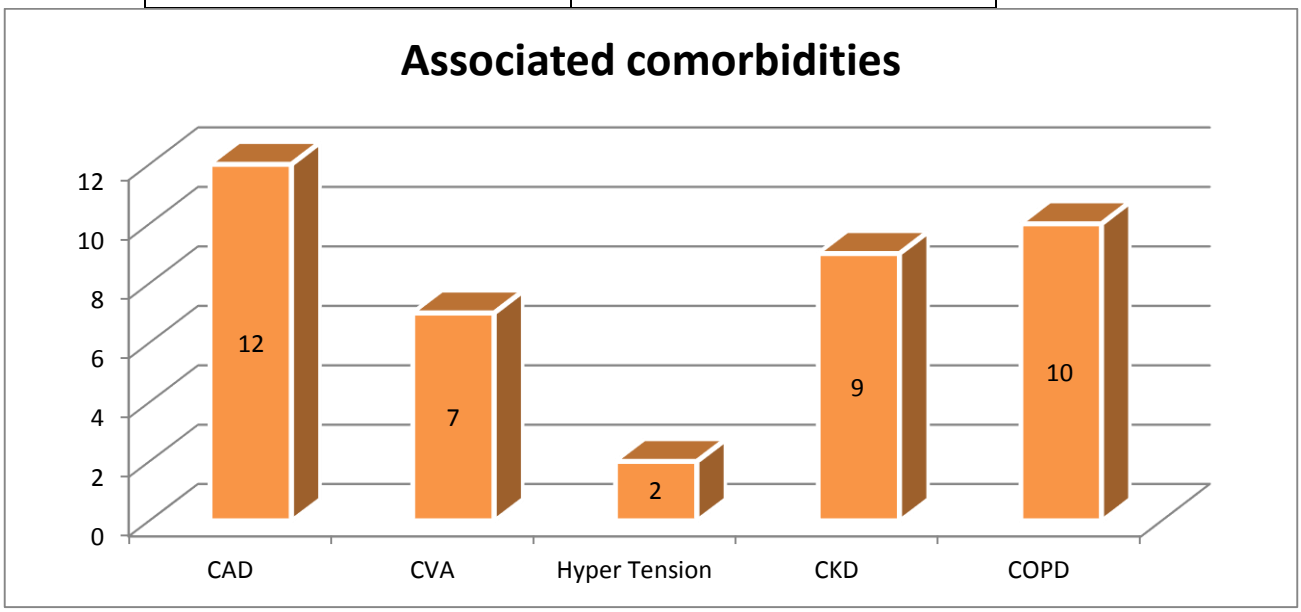


FIGURE8 ASSOSIATEDCOMORBIDITIES

CAD-12%,CVA7%,HYPERTENSION2%,CKD9%,COPD10%.Patients with more than one comorbidities not included.

9. DURATION OF DIABETES (table9)

Duration of Diabetes	NO OF CASES
<5YEARS	**58**
5-9 YEARS	**32**
≥10 Years	**10**
TOTAL	100

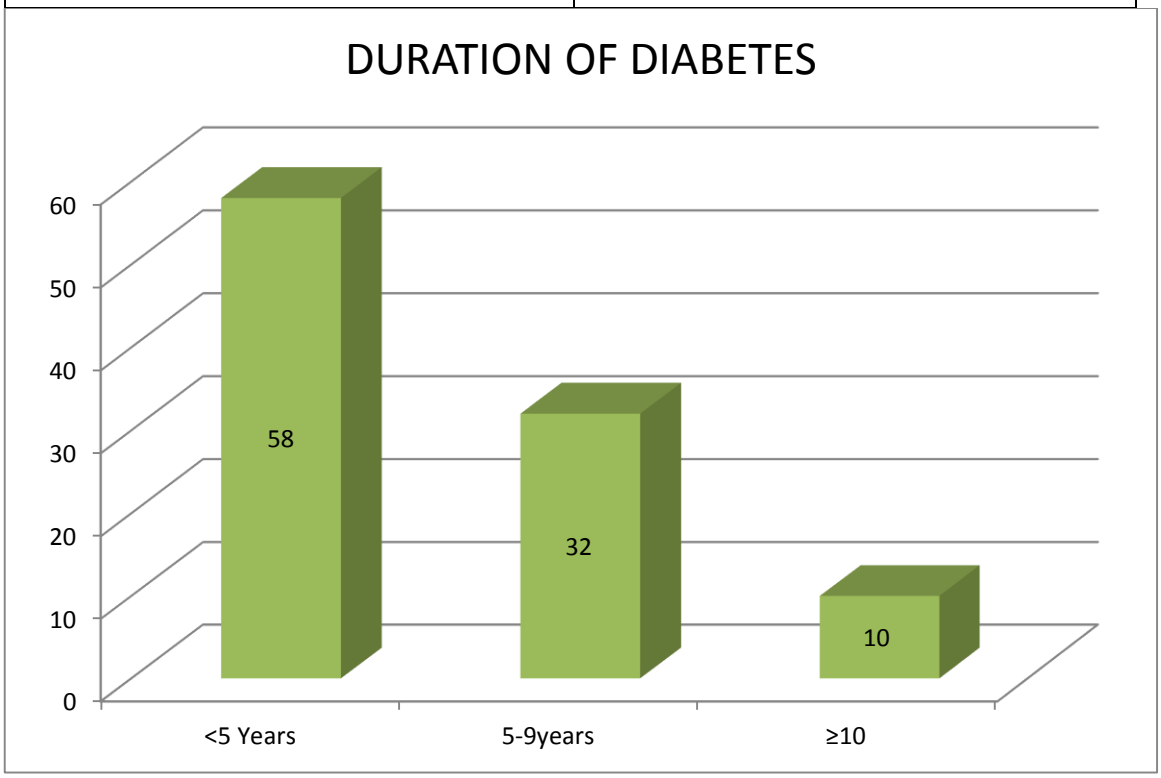


Figure 9 DURATION OF DIABETES

In this study 58% had diabetes of less than 5 years duration,32% had 5to9 years duration;10% had diabetes of more than 10 years . New patients were included in <5 yrs.

10. PRECIPITATING FACTORS OF DKA(table10)

Risk factors	no of cases
non compliance	48
infections	38
sepsis	14
steroids	10
myocardial infarction	5
stroke	3
alcoholic	8
	100*

*One patient had more than one precipitating factors

In this study DKA had multiple precipitating factors like noncompliance, infections, sepsis[48], MI, stroke, alcoholism. Among that 48% had noncompliance, 38 patients had infections, 14 had sepsis, and 10 patients were on steroids. 5 patients had acute stressful condition like MI, 3 had stroke. Overall precipitating

factors had p value <0.001; so each precipitating factors had significant association with DKA.

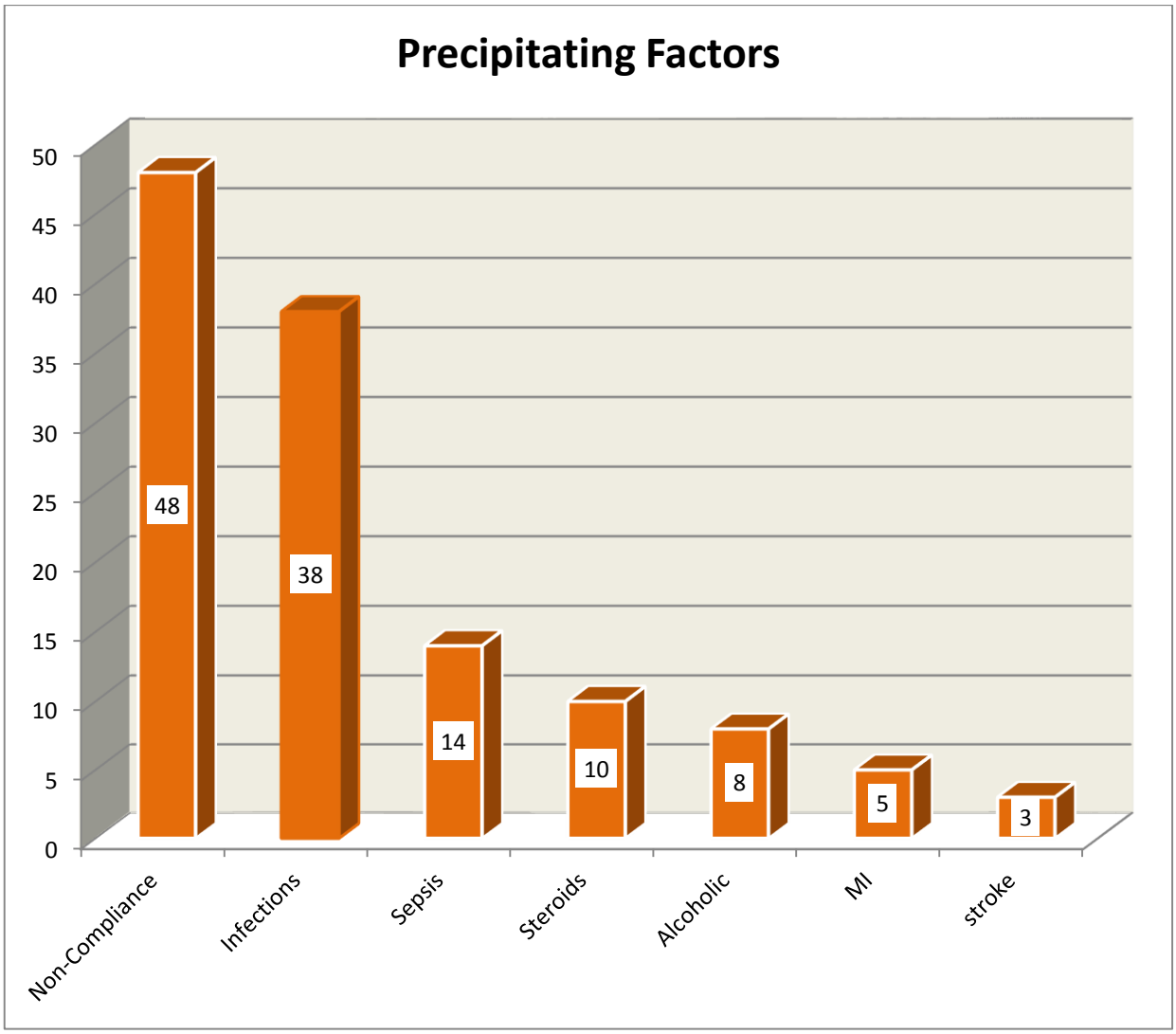


Figure 10 -PRECIPITATING FACTORS

11. BODY MASS INDEX(table11)

BMI	Cases	
<18	4	4%
18-25 (Normal)	66	66%
25-30 (Over-Weight)	25	25%
30-35 (Obese I)	5	5%
35-40 (Obese II)	0	0%
Total	100	100%
Range	16.7-32.1	
Mean	23.2	

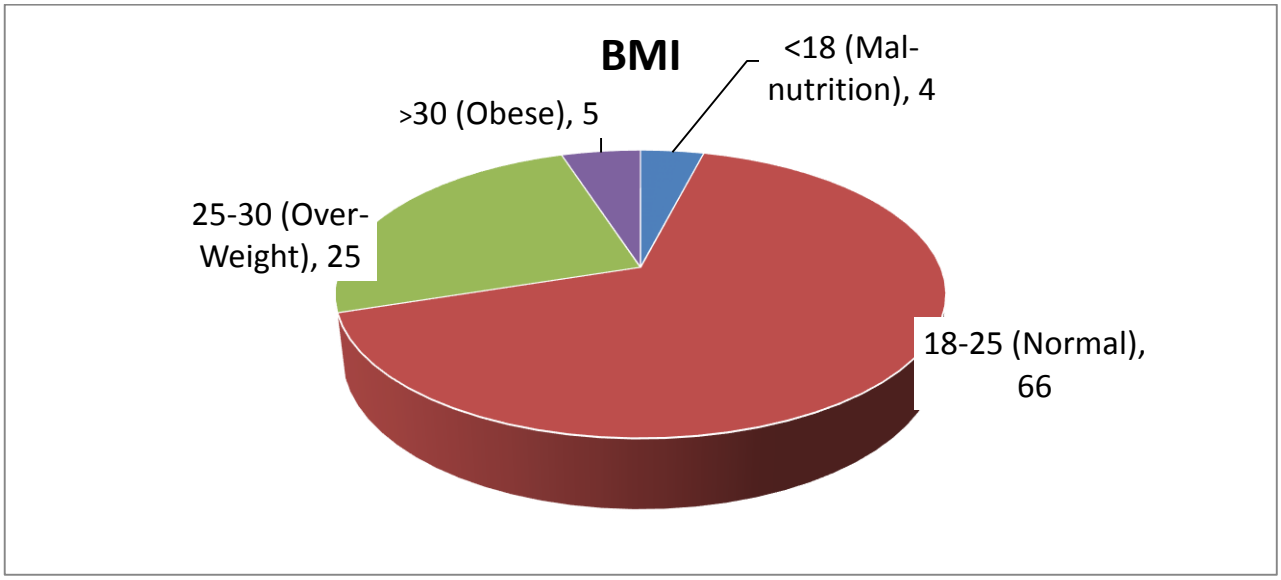


Figure 11 BMI

66% -normal BMI ,25% overweight category,5% obese I category. The mean body mass index around 23.2

12. VITALS(table12)

Pulse rate ranged from 68-108; mean 87.29. Respiratory rate ranged between 14-28 mean values 18.6.

	Range	**Mean**	**SD**
Pulse rate	68-108	87.29	7.46
RESPIRATORY RATE	14-28	18.6	2.89

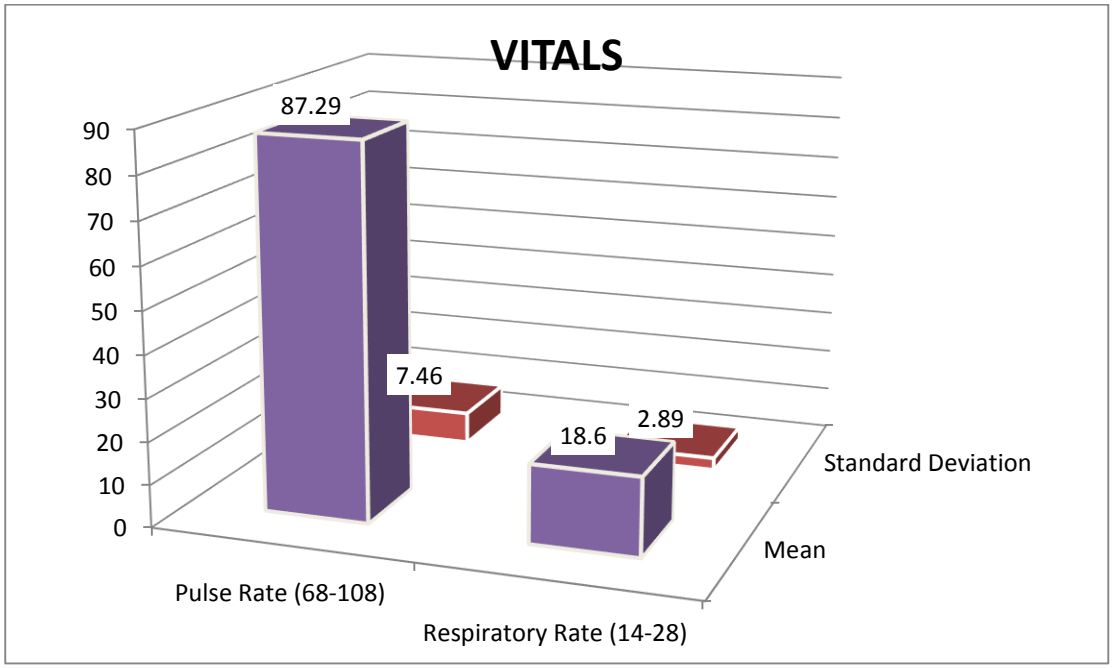


Figure12) VITALS

13. BLOOD SUGAR, UREA & CREATININE LEVELS(table13)

VARIABLES	Range	Mean	SD
Blood Sugar(mg/dl	380-659	523.22	62.19
Urea(mg/dl)	34-94	50.42	13.24
Creatinine(mg/dl)	0.8-3.9	1.612	0.56

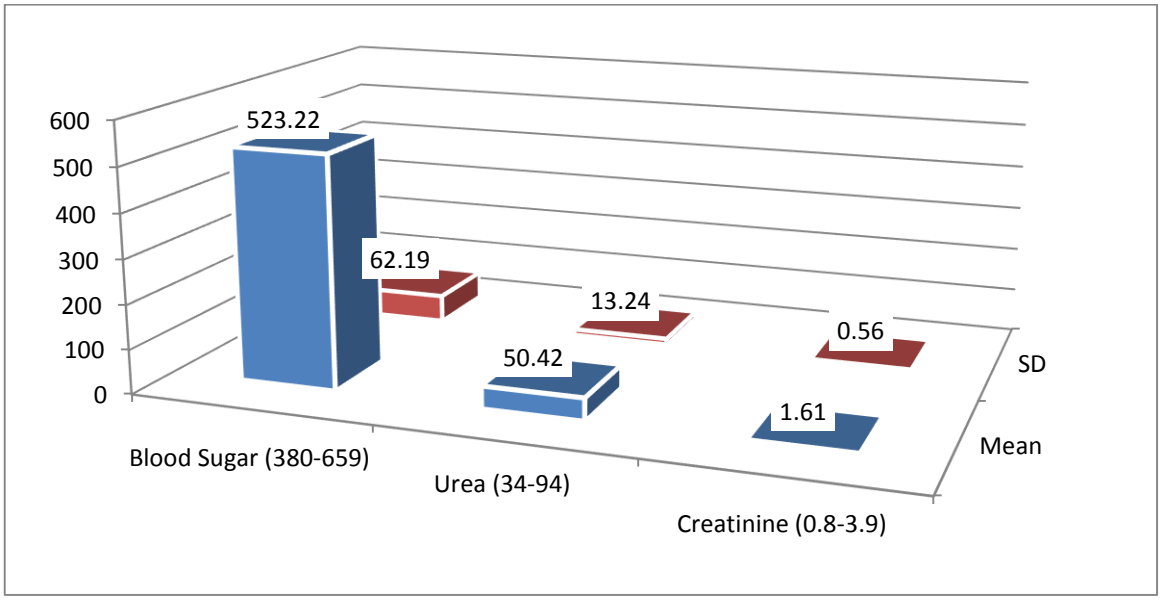


Figure 13 VARIABLES- BLOOD SUGAR, UREA, and CREATININE

The average blood sugar value was 523.22 mg% with a ranged between 380 mg %and 659 mg%. The mean urea values were 50.42 mg%. The average creatinine values were 1.612...Standard deviation (SD) was calculated.

14. ELECTROLYTE AND PH,BICARBONATE LEVELS(table14)

Variable	Range	Mean	SD
POTASSIUM (K)mEq/l	2.8-6.2	4.33	0.734
SODIUM(Na)mEq/l	124-136	133.24	2.86
pH	6.02-7.30	7.13	0.31

BICARBONATEmEq/l	6-17	12.56	2.69

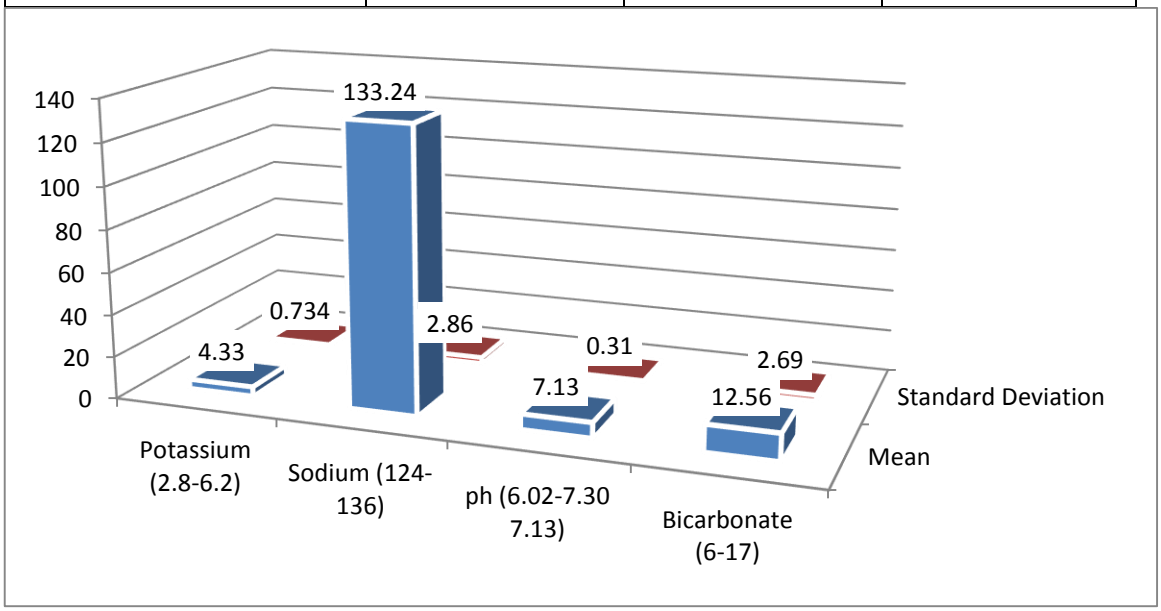


Figure 14 VARIABLES –POTASSIUM, SODIUM, BICARBONATE, PH

Average potassium value 4.33,ranged between 2.8to 6.2.Sodium levels ranges between 124to 136 meq/l. Average value of serum sodium 133.24..ABG ;PH Value ranged between 6.02 to7.30,mean pH 7.13.Bicarbonate ranged between6 to17;mean bicarbonate 12.56.

15. COMPLICATIONS(table15)

Complications	No of cases
Death	16
Prolonged hospital stay>2week	25

Sepsis	14
Electrolyte abnormalities	32
Respiratory failure	16
Severe acidosis	8
Altered sensorium with encephalopathy	14
Total	100*

*One patient can have more than one complications

DKA had many complications.IN this study among 100 patients most of patients had more than one complication.16 patients died due to various complications.

Morbidity assessed by prolonged hospital stay>2week duration with various complications.26 patients had prolonged hospital stay. Electrolyte disturbances [49] like hypokalemia, hyponatremia, hyperkalemia were occurred among 32 patients. Respiratory failure were seen in 16 patients.14 patients had developed altered sensorium, metabolic encephalopathy.14 patients had developed severe sepsis

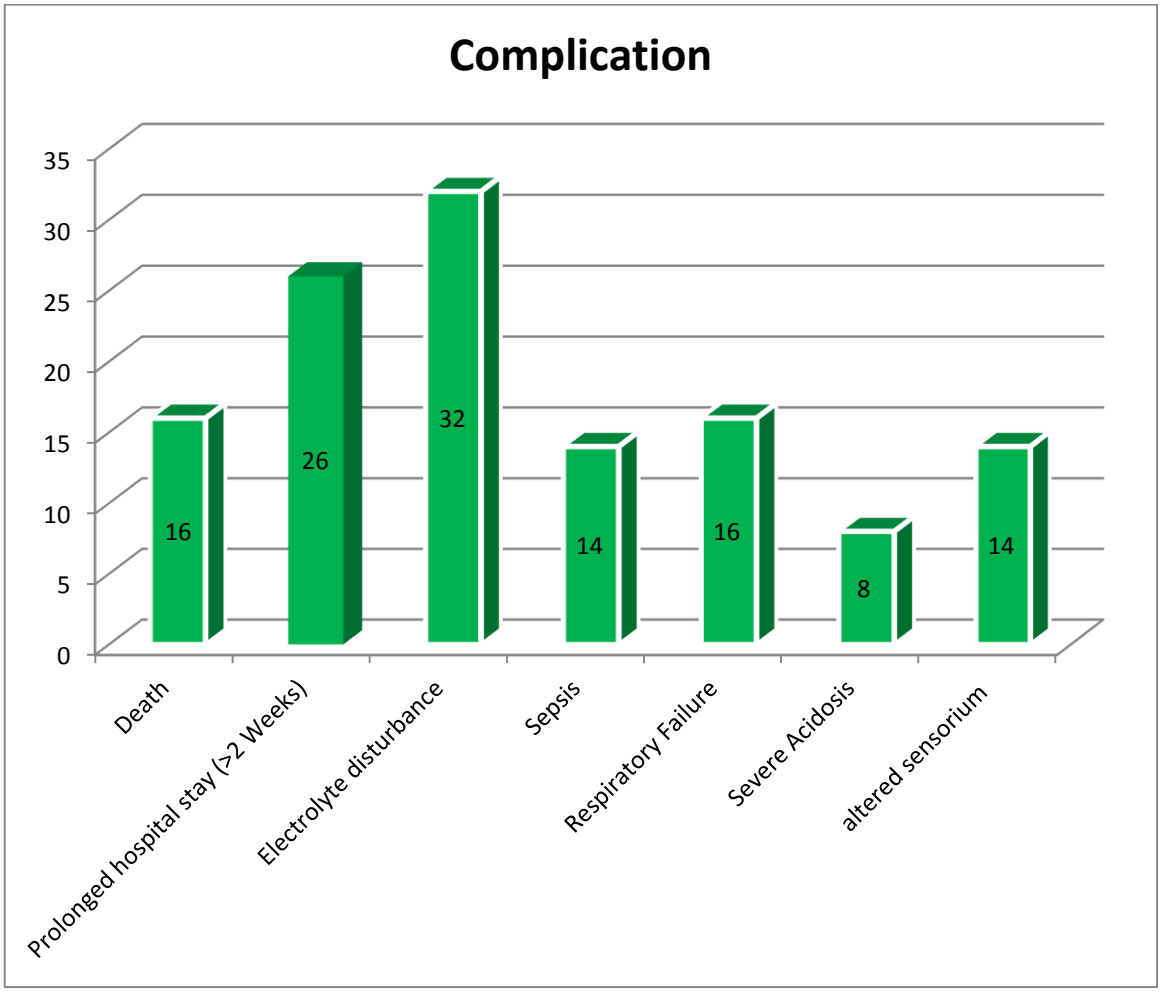


Figure 15 COMPLICATIONS

16. ELECTROLYTE ABNORMALITIES (table16).

Electrolyte	No of patients
hyperkalemia	4
Hyponatremia	11
hypokalemia	17

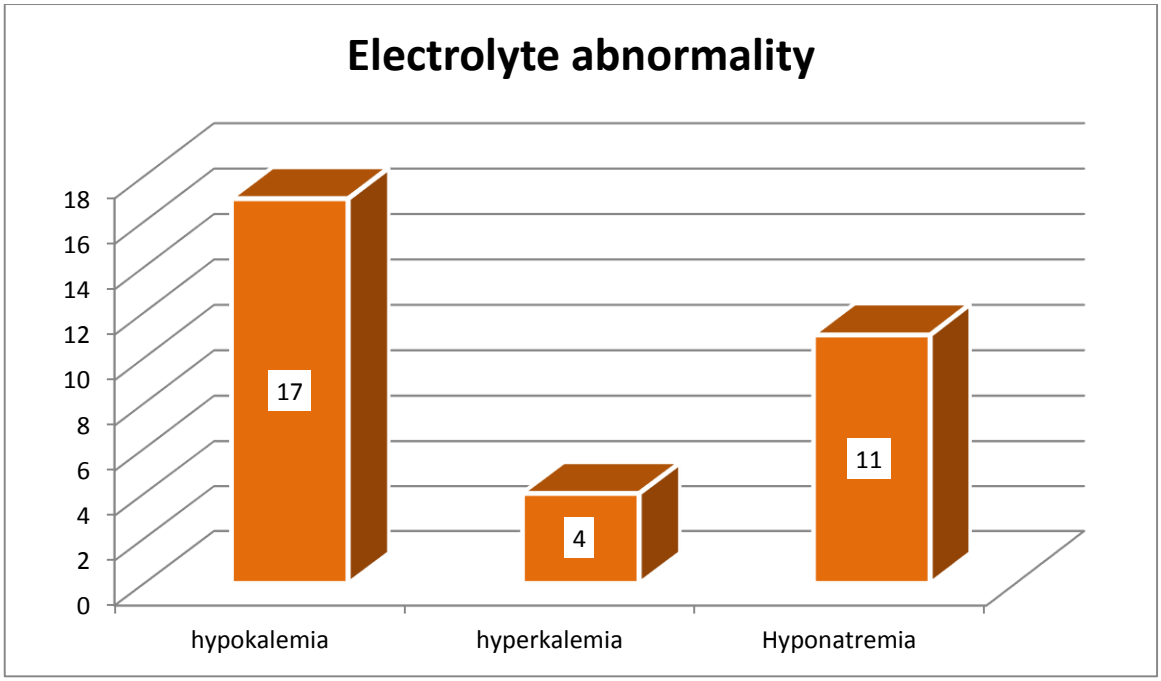


Figure 16 ELECTROLYTE ABNORMALITIES.

Among 100 patients,17 patients had developed hypokalemia,4had hyperkalemia,11 patients had developed hyponatremia.

17. RELATION BETWEEN TYPE OF TREATMENT TAKEN WITH MORTALITY AND MORBIDITY(table17)

Treatment type	**Death**	**<2 Weeks**	**>2week***	**total**
oralantidiabetic drugs	11	22	16	49
Insulin	1	24	6	31
Both	4	7	4	15
total	16	53	26	**100***

* some patient were on insulin and oral diabetic drugs ; some patients new onset DM

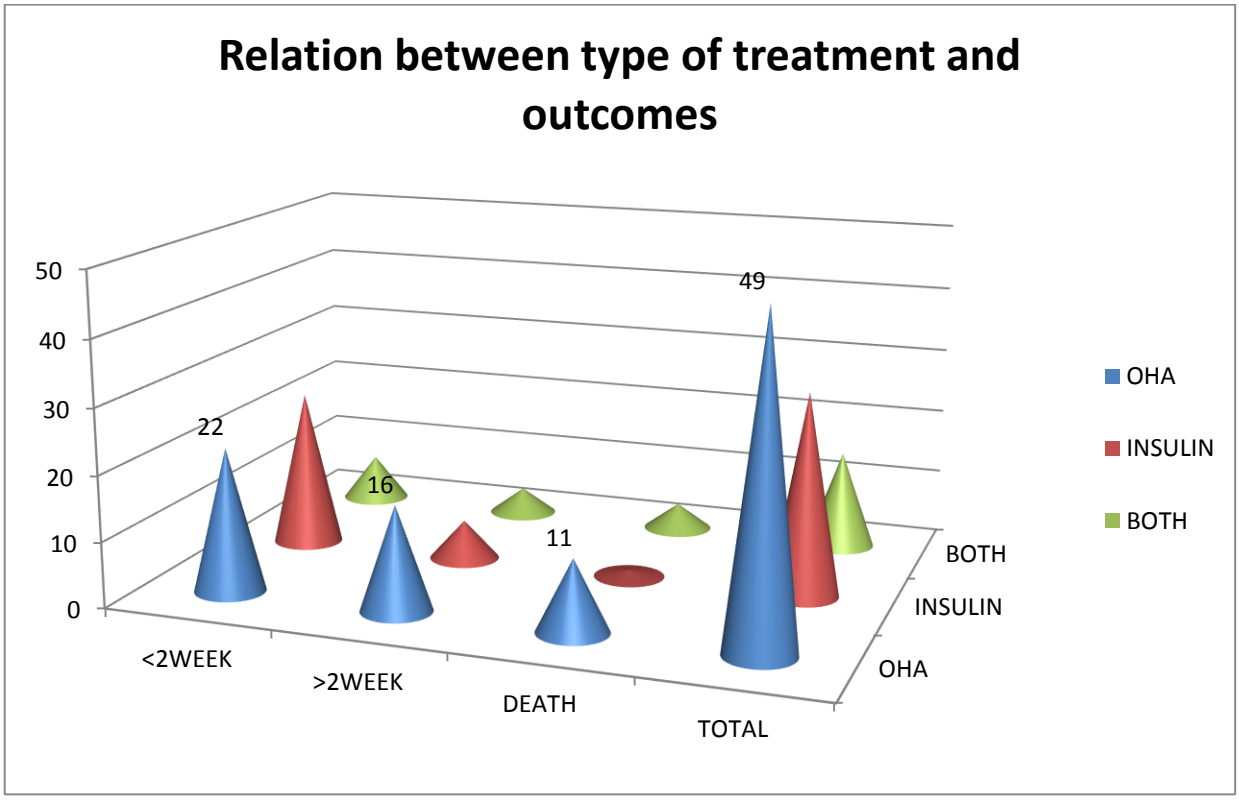


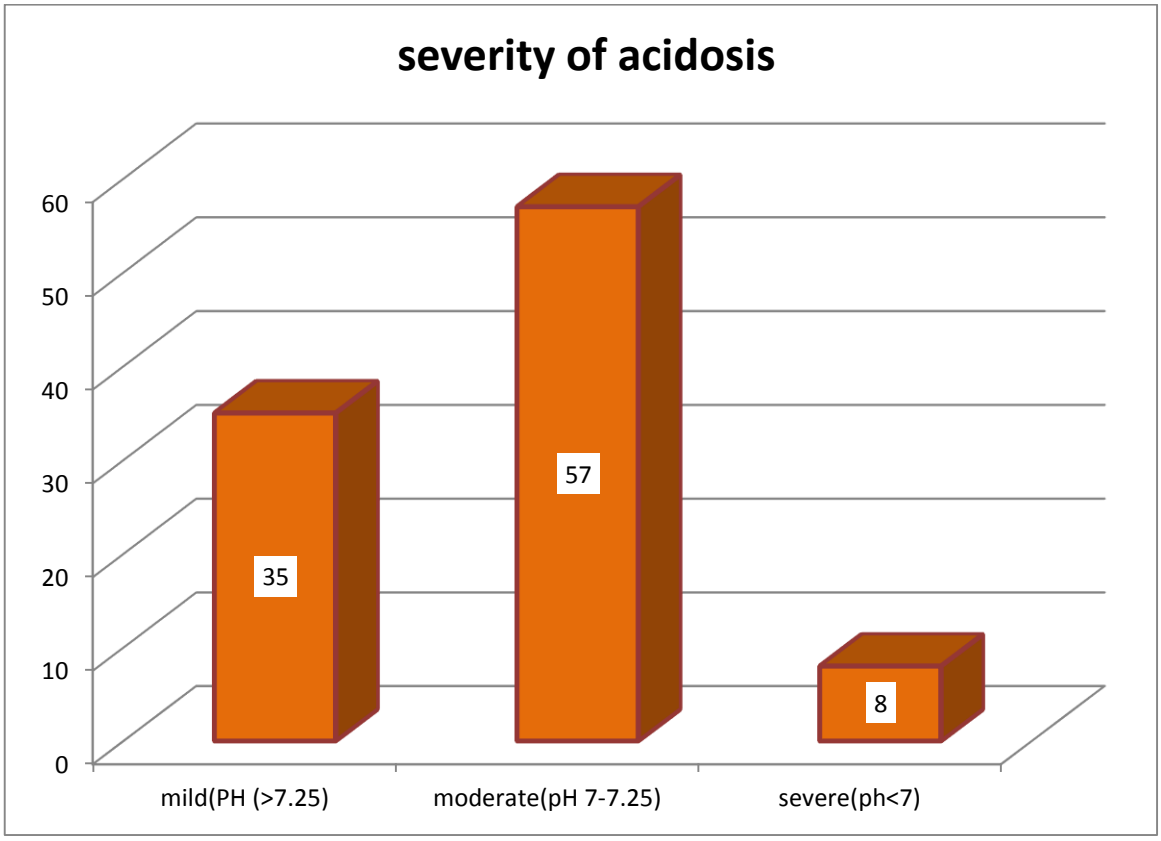
Figure 17 relation between type of treatment and outcomes

In this study patients with oral antidiabetic drugs had high mortality and morbidity (which was determined by prolonged hospital stay>2week and complications). Among total 16 deaths 11 patients were on oral diabetic drugs. Patients those were on both insulin and oral drugs 4deaths were observed. Patients with only insulin 1 death was observed.16 patients with OHA had prolonged hospital stay and complications. 4patients taken both had prolonged hospital stay.6 patients taken only insulin had prolonged hospital stay. Patients those taken oral hypoglycemic drugs had more mortality compared to insulin; p value was <0.001 statistically significant.

18. SEVERITY OF ACIDOSIS BASED PH VALUE; AND ITS ASSOCIATION WITH MORTALITY (table18).

type of acidosis	No of patients	Death	%
mild (pH>7.25	35	2	5.7%
moderate(pH 7 to7.25	57	10	17.5%
severe(pH<7)	8	4	50%
total	100		
Range 6.02-7.3			
Mean ----7.13			

Figure 18 -severity of acidosis



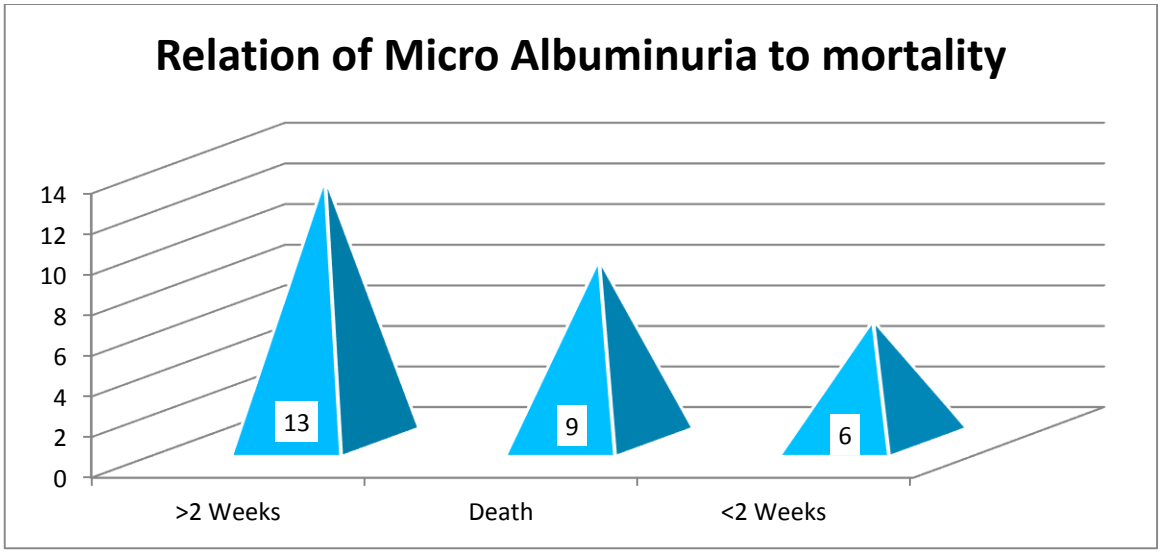
Based on PH value patients were classified into mild, moderate, severe acidosis .PH<7 with severe acidosis observed in 8 patients.57 patients had pH ranged 7 to7.25 grouped into moderate acidosis.PH value >7.25 with mild acidosis were 35 patients. PH value ranged from 6.02 to 7.30. Moderate to severe acidosis can cause more mortality and morbidity. 8 patients with severe acidosis, 4 death were observed. Patients with moderate acidosis 10 death were occurred. So there is a significant association between mortality with severity of acidosis, p value<0.001 was statistically significant.

19. RELATION OF MICROALBUMINURIA WITH MORTALITY AND MORBIDITY(table19)

Outcomes	NO OF PATIENTS
DEATH (16)	9
>2WEEK*(26)	13
<2WEEK	6
TOTAL	28

*>2 WEEK INDICATES MORBIDITY

Figure 19 relation of microalbuminuria with mortality and morbidity

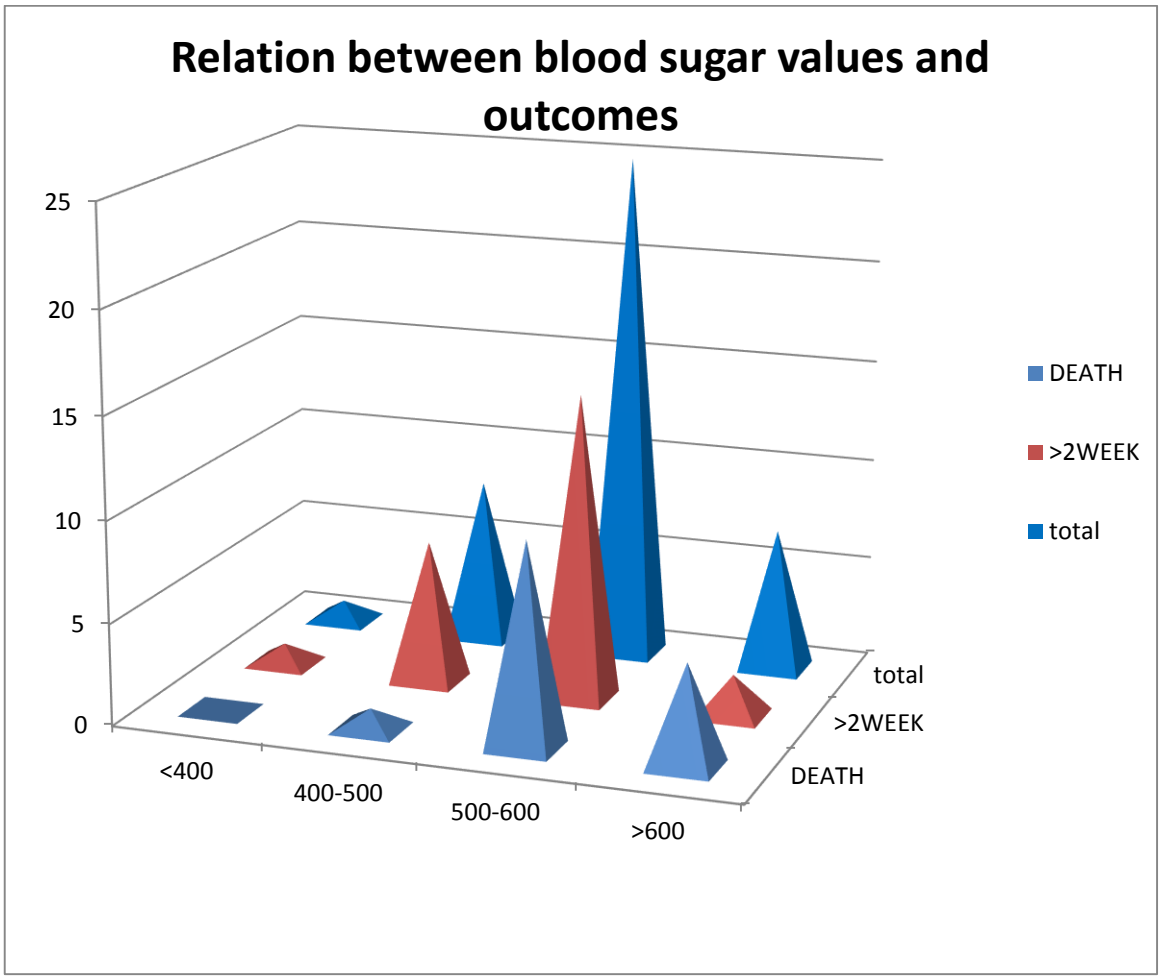


Microalbuminuria observed in association with morbidity and mortality.28 patients had microalbuminuria; among 16death 9Patients had microalbuminuria, patients with hospital stay>2 week 13 had micro albuminuria.

20. RELATION BETWEEN BLOOD SUGAR VALUES WITH MORTALITYAND MORBIDITY (TABLE20)

RBS(mg/dl)	DEATH	>2WEEK	TOTAL
<400mg/dl	0	1	1
400-500	1	7	8
500-600	10	15	25
>600	5	2	7

Figure 20 RELATION OF BLOOD SUGAR WITH OUTCOMES



Among patients with DKA blood sugar values ranged from 380-659mg/dl. Mortality increased with high RBS value .More death were observed when RBS

value >500mg/dl.10death were observed with RBS range 500-600.In this RBS range- prolonged hospital stay were observed in15 patients.5 death were occurred when RBS morethan600mg/dl. Below 500mg/dl; one death was observed. So higher the blood sugar values more mortality and morbidity; p value< 0.001 was statistically significant

21. RELATION BETEEN MORTALITY AND ASSOSIATED COMORBIDITIES.(TABLE21).

RISK FACTORS	Death
CAD	5
CVA	1
CKD	3
COPD	0
SEPSIS	7
TOTAL	16

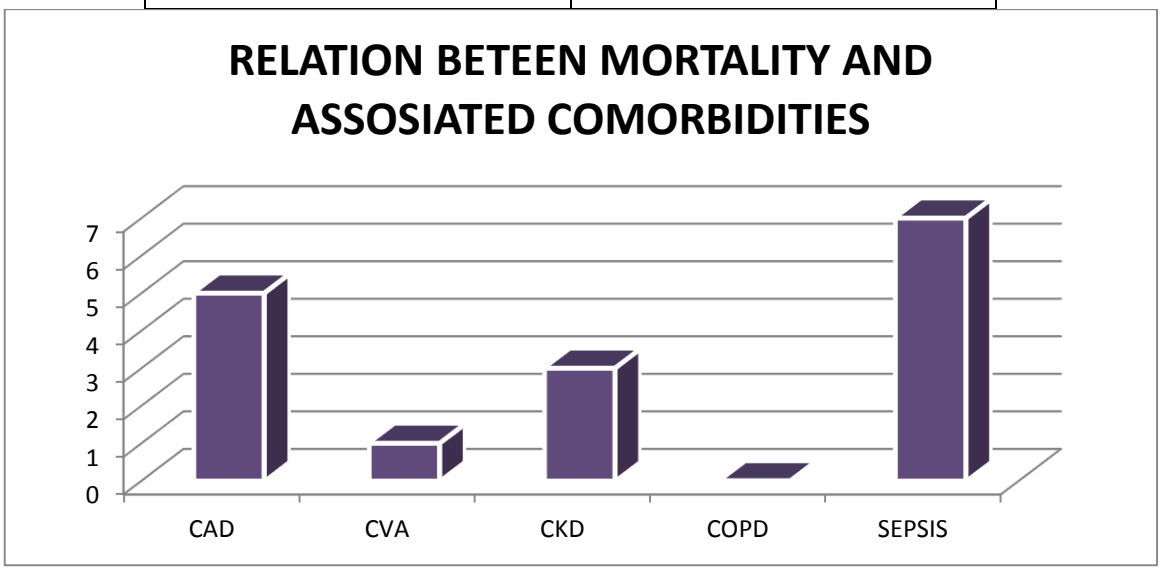


Figure. 21 RELATION BETWEEN MORTALITY AND ASSOSIATED COMORBIDITIES

Among 16 deaths most of them had associated with other comorbid illness.5 patients had suffered coronary artery disease. Severe sepsis observed among 7 patients.3 patients had chronic kidney disease

22. RELATION BETWEEN DIABETES DURATION & MORTALITY AND MORBIDITY(TABLE22).

	Death	<2 Weeks	>2WEEKS
<5Years	3	44	11
5 -9years	7	14	11
≥10Years	6	1	3
TOTAL	16	59	25

. New onset diabetes also included in less than 5yr

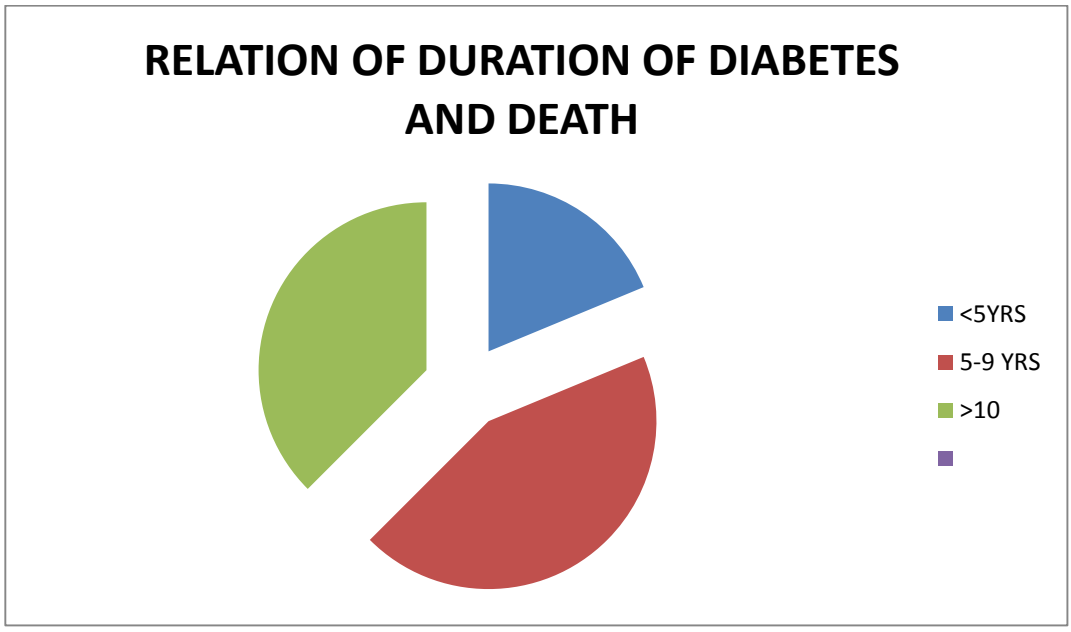
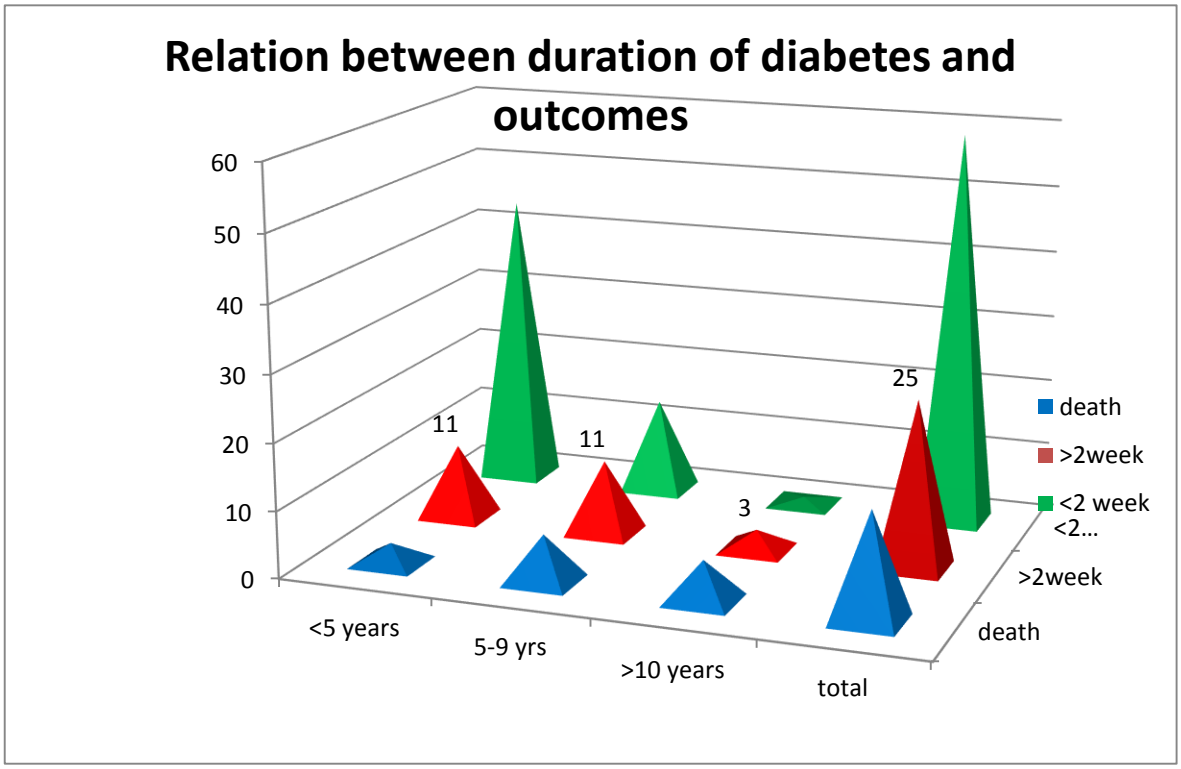


Figure 22 RELATION BETWEEN DURATION OF DM AND DEATH

Figure 23 RELATION BETWEEN DURATION AND OUTCOMES



Among 58 patients with <5 years diabetes duration 3 deaths; 11 patients had prolonged hospital stay observed. Among 32 patients with diabetes 5 to 9yrs 7 deaths; 11 patients had prolonged hospital stay due to complications observed. Out of 10 patients with diabetes more than 10 years 6 deaths occurred; 3 patients had prolonged hospital stay observed. So duration of diabetes had significant relation between DKA mortality and morbidity[50] p value< 0.001; was statistically significant**.**

DISCUSSION

Diabetic keto acidosis is a serious acute complication of diabetes.

DKA--- a hyperglycemic emergency which is an important cause of morbidity and mortality in patients with diabetes. This study has been done with regard to risk factors in the predominantly rural population.

DKA is common among Type 1 diabetes patients; may be a first presenting feature of type1 DM. In Type 2 diabetes patients with prolonged duration; can develop DKA in conditions, such as infections[51],sepsis and stressful situations like myocardial infarction, stroke .Most important precipitating factors include noncompliance to drugs or insulin omission.

In this study male to female ratio 58:42. DKA was most commonly seen among males. Among 58 males; 16 had type 1 diabetes, 42 had type 2 diabetes. Among 42 females; 14 had type 1 diabetes, 18 had type 2 diabetes. In this study DKA were more common among males.70 patients had type 2 diabetes; 30 patients had type 1 diabetes. Among 100 patients 9% patients (9 cases) had new onset of diabetes presented as DKA.

In this study among Type 2 DM patients DKA were common between 50-60yrs. In our study among type 2DM cases 59%were below 70 years. Type 1 DM DKA were common between 14 -23 yrs.

Most of patients had more than one symptoms. Common symptoms include fever, vomiting, altered sensorium, specific symptoms for infections like urinary tract infections[52], lower respiratory infections. Most of the patients had more than one symptom. With regarding to presenting complaint 38% had symptoms specific to focus of infection like dysuria, cough, loose stools, no healing ulcers and 37 patients had altered sensorium, 30 patients had fever as presenting symptom .Among the focus of infections13 had symptoms urinary tract infection,7 had lower respiratory tract infections[53],10patients were suffered from soft tissue infections. 8 had acute gastroenteritis/acute diarrheal disease. Diabetes were more prone for common infections like urinary tract infections and soft tissue infections. Patients with uncontrolled hyperglycemia were more prone for infections. In Nigerian study by Andrew about DKA common symptoms observed; fever, altered sensorium, breathlessness etc.

Among 70 patients with type2 diabetes 49patients were on oral anti diabetic drugs , 15patients were on insulin and oral antidiabetic drugs .Among 30 type1 patients all were taking insulin only. Among diabetes patients most of them not taking treatment regularly .In this study 48% DKA patients were not on regular treatment.

In any acute stressful conditions like infections, sepsis, MI; omission of insulin[54] cause DKA. In this study the predominant precipitating factors were related to insulin therapy (missed insulin injection, change in insulin dose or

regimen), noncompliance and infections. In this study drug noncompliance (48%) was most common precipitating factor for DKA. Many patients had more than one risk factors. Among 100 patients; 38 had infections, 14 had sepsis, 10 patients were on steroids. Acute stressful condition like MI[55], stroke were precipitating factors among 8% patients. Among alcoholics 8% patients with irregular treatment led to DKA. Noncompliance in rural population was due to low socioeconomic status and lack of knowledge .Most of the diabetic patients had no proper follow up; and also unaware of complications.

Many of the patients had more than one risk factors. Overall pvalue<0.001 for risk factors, so was statistically significant. In a similar study done in New jersey[56], most important precipitating factors were related to drug therapy, insulin omission.

In a Nigerian study by Andrew and American study[57] by John; most significant risk factor mentioned were non-compliance and infections. In Nigerian study malarial infections was included as a risk factor; it was not found in this study.

DKA had various complications like electrolyte abnormalities, sepsis, respiratory failure, severe acidosis. DKA is an important cause of mortality in diabetes patients. In this study among 100 patients most of patients had more than one complication.16 patients were died due to various complications. Morbidity assessed by prolonged hospital stay>2week duration with various

complications.26 patients had prolonged hospital stay. Electrolyte disturbances like hypokalemia, hyponatremia, hyperkalemia were occurred among 32 patients. Respiratory failure [58] were occurred in16 patients.14 patients had developed altered sensorium due to metabolic encephalopathy.14 patients had developed severe sepsis. In a study conducted in Nigeria[59] by Andrew, hyponatremia and hyperkalemia, hypokalemia were commonly observed. .In a similar study done in Israel[60], complications observed were death, respiratory failure, multiorgan failure, electrolyte abnormalities, acidosis, altered sensorium etc. Association of comorbidities also mentioned in that study.

Most of the diabetic patients had chronic complications like CKD, STROKE, and CAD. In our study, comorbidities like coronary artery disease was found in 12% of the cases and chronic kidney disease in 9% patients stroke in 7% cases, 2% hypertension 10% patient had COPD. Associated comorbidities also led to death and prolonged hospital stay. Only in few cases, death occurred due to DKA. Associated comorbidities and complications led to more mortality. In this study most of patients with single comorbidity was selected. More than one comorbid illness can confound the mortality risk, hence patients with more than one comorbid illness not included in this study.

Among 16 deaths most of them had associated with other comorbid illness.5 patients had suffered from coronary artery disease. Severe sepsis occurred in 7 died patients.3 patients had CKD.

In this study 58% had diabetes of less than 5 years duration,32% had 5 to9 years duration;10% had diabetes of more than 10 years .New onset diabetes was included in <5year duration category. In this study diabetes with more than 5yrs; 13 deaths occurred. 14 patients had prolonged hospital stay. Patients with prolonged duration of diabetes have more mortality and morbidity[61].They had prolonged hospital stay due to more complications[62].This association was statistically significant with

p value <0.001.

Diabetes with micro albuminuria was observed in 28 patients; it was associated with morbidity and mortality of DKA. 13patients with prolonged hospital stay more than 2 week had micro albuminuria. Among total 16 death; 9 patients had micro albuminuria.

In this study patients with oral anti diabetic drugs had high mortality and prolonged hospital stay>2week and complications. Among total 16 deaths , 11 were on oral diabetic drugs. 4death were observed in patients were taken both insulin and oral drugs. Patients were on insulin; 1 death observed. So patients with oral antidiabetic drugs had more mortality compared to insulin .Association was statistically significant with p value <0.001.In a similar study; Diabetic ketoacidosis and clinical outcome conducted by department of family medicine

Israel; similar results were obtained .Patients with oral drugs were prone to more complications

Among patients with DKA blood sugar values ranged from 380-659. Mortality increased with high blood sugar value .More death were observed when blood sugar value more than 500 mg/dl. One death was seen in blood sugar value less than 500 mg/dl.15 death occurred in patients with RBS more than 500mg/dl. Prolonged hospital stay was observed in17 patients with RBS value morethan[63]500 mg/dl. Relation between RBS and mortality was statistically significant with pvalue<0.001.

According to PH value, serum bicarbonate levels patients were classified into mild, moderate, severe acidosis. PH VALUE[64] more than7.25 with mild acidosis were occurred among 35 patients.PH less than7 with severe acidosis occurred in 8 patients.57 patients had pH value ranged from 7 to7.25 grouped into moderate acidosis. Among the patients with severe acidosis 4 patients were died. More severe the acidosis more complications were observed .In this study more patients presented with moderate acidosis, among them 10 death occurred. Association between severity of acidosis and mortality was statistically significant with p value <0.001

There may be many shortcomings in this study. But this study will definitely give us a fair idea of the risk factors and prognosis of DKA in the rural Indian population.

SUMMARY

- In our study it was found,
- Males are more commonly affected.
- DKA common in type 1DM and also it can occur in type2[65] diabetes.
- Most important precipitating factors are noncompliance to drug ,inadequate insulin therapy, infections[66] followed by drugs like steroids, alcohol ,acute stressful events like MI, STROKE
- Associated comorbid illness like stroke, MI, CKD cause more mortality and complications in DKA patients
- DKA have many complications like sepsis, electrolyte disturbances, metabolic encephalopathy, respiratory failure[67], severe acidosis etc. These complications cause more mortality and prolonged hospital stay contribute to morbidity.
- Diabetic patients with prolonged duration had significant mortality and morbidity, they were prone for complications.
- Patients with severe acidosis had more mortality and more complications.
- Patients with high blood sugar value had high mortality and morbidity.
- Patients with oral anti diabetic drugs had significant mortality and morbidity, compared to insulin therapy.

CONCLUSION

Diabetic ketoacidosis is an important acute complication[68] of diabetes. Precipitating factors include omission of insulin, noncompliance to drugs, infections[69], sepsis, myocardial infarction, stroke ,other drugs like steroids, alcoholism etc. DKA have multiple complications ; that can led to death and prolonged hospital stay. Complications include sepsis, respiratory failure, electrolyte abnormalities, acidosis , altered sensorium etc. Treatment mainly includes adequate fluid management, insulin administration, and correction of electrolyte abnormality .Identification and treatment of precipitating factors are more important .Patient education[70] plays a crucial role in prevention of DKA.

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67. MURRAY AND NADELS TEXT BOOK OF RESPIRATORY MEDICINE,5 TH EDITION,ROBERT,THOMAS,PART 3
68. DIABETIC EPIDEMIC IN INDIA, MD AASIF SIDDIQUE AHMED KHAN1 , ASHRAFA , SHABANA PARVEEN , INSTITUTE OF MEDICAL SCIENCES AND RESEARCHCENTRE,HYDERABAD., NIMRACOLLEGEOFPHARMACY,NIMRANAGAR,VIJAYAWADA

DEPARTMENT OF PHARMACEUTICAL SCIENCES, COIMBATORE MEDICAL COLLEGE

69. INDIAN JOURNAL OF ENDOCRINOLOGY AND METABOLISM, ,2012 MARCH INFECTIONS IN PATIENTS WITH DIABETES MELLITUS: A REVIEW OF PATHOGENESIS JULIANA CASQUEIRO, JANINE CASQUEIRO, AND CRESIO ALVES

70. TEXT BOOK OF SOCIAL AND PREVENTIVE MEDICINE,PARK,23 RD EDITION

ANNEXURE

1) PROFORMA

2) Consent Form

3) MASTER CHART

PROFORMA

1) NAME OF THE PATIENT
2) AGE
3) SEX
4) IP NUMBER
5) ADDRESS

HISTORY:

.MODE OF ONSET

.DURATION OF DIABETES

GENERALISED WEAKNESS

FATIGUE

NAUSEA AND VOMITING

ABDOMINAL PAIN

BREATHLESSNESS

ALTERED SENSORIUM

ANY FOCUS OF INFECTION

FEVER

ON REGULAR MEDICATION/NOT

PAST HISTORY

- DIABETES

- HOW LONG
- TREATED OR NOT
- WHICH MEDICATION
- DOSE

  - HYPERTENSION,CKD ,MI,CVA,COPD
  - OTHER MEDICATIONS

CLINICAL EXAMINATION

Vitals

General appearance

Sign of acidosis----shallow/rapid breathing

Sign of dehydration---weak and rapid pulse

Dry skin

Fruity/acetone smell to breath

SYSTEM EXAMINATION

RESPIRATORY SYSTEM

CVS

ABDOMINAL EXAMINATION

CNS

<u>INVESTIGATIONS</u>

CBC

RBS

RFT

ELECTROLYTES

LFT

URINE ANALYSIS

URINE ACETONE

ECG

CHEST X –RAY

URINE ANALYSIS

URINE CULTURE

ABG

ABBREVATIONS

BMI	-	BODY MASS INDEX
BP	-	BLOOD PRESSURE
CAD	-	CORONARY ARTERY DISEASE
CKD	-	CHRONIC KIDNEY DISEASE
CVA	-	CEREBRO VASCULAR ACCIDENT
DKA	-	DIABETIC KETO ACIDOSIS
DM	-	DIABETETS MELLITUS
GLP-1	-	GLUCAGONLIKE PEPTIDE–1
LRI	-	LOWER REPIRATORY TRACT INFECTION
MI	-	MYOCARDIAL INFRACTION
OHA	-	ORAL HYPOGLYCEMIC AGENTS
PR	-	PULSE RATE
SGLT-2	-	SELECTIVE SODIUM-GLUCOSE TRANSPORTER-2
TZD	-	THIOZOLIDINEDIONES
UTI	-	URINARY TRACT INFECTIONS

INFORMED CONSENT FORM

Study Title _____

Study Number _____

Subject's Full Name _____

Date of Birth/Age_____

Address _____

1. I confirm that I have read and understood the information sheet dated for the above study and have had the opportunity to ask questions.
   OR I have been explained the nature of the study by the Investigator and had the opportunity to ask questions
2. I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason and without my medical care or legal rights being affected.
3. I understand that the sponsor of the clinical trial/project, others working on the Sponsor's behalf, the Ethics Committee and the regulatory authorities will not need my permission to look at my health records both in respect of the current study and any further research that may be conducted in relation to it, even if I withdraw from the trial. However, I understand that my Identity will not be revealed in any information released to third parties or published.
4. I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s)
5. I agree to take part in the above study

Signature (or Thumb impression) of the Subject/Legally Acceptable Representative: _____

Signatory's Name _____ Date _____

Signature of the Investigator _____ Date _____

Study Investigator's Name _____

Signature of the Witness _____ Date _____

Name of the Witness

s.no	Name	Age	Sex m/f	History <1 week / >1 week	vomiting	fever (Y/N)	altered sensorium (Y/N)	focus of UTI Y/N	focus infection		ADD / AGE	PAST H/O Type1/2 new	DM DURAT years	COMPLI Y/N	Insulin	INSULINAND OHA	OHA	CAD Y/N	CVA Y/N	SHT Y/N	CKD	COPD	STEROIDS	oth drug	ALCOHOL Y/N	SMOKING Y/N	Weight	Height	BMI	DEHYDRA Y/N	Pulse Rate	Resp Rate	Temp High/ normal(N)	SIRS Y/N For sepsis	TC	RBS >300-	RFT UREA	CREATINE	Na 130-135	K 3.5-5.5	urine, alb umin, positive/not	CS URINE growth/no Y/N	blood cs Y/N	ABG ACIDOSIS SEVERITY	S HCO3	PH	STAY	Outcome DEATH/Y RECOVERD	repiratory failure y/n
									LRI	soft tissu																																						
1	MARI	54	F	<1	no	Y	no	Y	no	no	no	2	6	Y	no	no	Y	y	N	N	N	N	N	N	N	N	56	152	24.2	y	78	16	h	y	14000	450	42	1.4	136	3.4	y	y	N	moderate	10	7.02		DEATH	N
2	Parvathy	60	f	<1	no	no	no	Y	no	no	no	2	7	y	Y	no	y	y	N	N	y	N	N	N	N	N	68	162	25.9	N	90	18	N	N	9900	530	78	2.9	136	3.9	y	N	N	moderate	12	7.22	>2	y	N
3	JANANI	17	F	<1	no	no	no	no	no	no	no	1	3	N	y	no	no	N	N	N	N	y	y	N	N	N	50	164	18.6	y	102	22	N	N	10500	512	38	1.2	128	3.5	N	N	N	acidosis	15	7.3	<2	Y	N
4	SUKANYA	15	F	<1	no	no	no	no	no	no	no	1	3	N	Y	no	no	N	N	N	N	N	N	N	N	N	45	164	16.7	y	88	18	n	N	4560	490	34	1.1	138	3.9	N	N	N	moderate	12	7.2	<1	Y	N
5	alagammal	16	f	<1	y	no	Y	no	no	no	Y	1	2	N	Y	no	no	N	N	N	N	N	N	N	N	N	52	160	20.3	y	88	18	N	N	8900	540	34	1.3	132	3.2	N	N	N	moderate	11	7.12	<1	Y	N
6	PETCHI	59	f	<1	no	no	no	no	no	no	no	2	6	N	no	no	Y	N	N	N	y	N	y	Y	N	N	62	154	26.1	N	90	16	N	N	11200	428	86	1.8	134	3.9	y	N	N	mild	15	7.3	>2	Y	y
7	KALYANI	57	f	<1	no	no	no	no	no	y	no	2	7	Y	no	no	Y	N	y	N	N	N	N	N	N	N	62	162	23.6	y	104	22	HIGH	N	7890	470	46	1.2	130	4.5	N	N	N	mild	16	7.28	<2	Y	N
8	SUBBU	62	f	<1	no	no	no	Y	no	no	no	2	5	y	no	no	no	N	N	N	N	N	N	N	N	N	68	172	23.0	y	78	16	N	N	5678	560	38	0.9	132	3.6	N	N	N	mild	15	7.3	<2	Y	N
9	Kali	65	f	>1	no	no	no	no	no	y	no	2	6	N	no	no	Y	N	y	N	N	N	N	N	N	N	68	160	26.6	y	84	16	N	N	11280	380	52	1.8	134	3.9	N	N	N	moderate	12	7.22	>2	Y	N
10	Athiselvi	47	f	>1	no	Y	no	no	no	no	no	new2	0	Y	no	no	no	N	N	N	N	N	N	N	N	N	54	164	20.1	y	92	24	N	N	9980	460	34	1.4	134	4.2	N	N	N	moderate	12	7.2	>2	Y	N
11	shanmugammal	56	f	<1	no	no	no	Y	no	no	no	2	6	N	Y	no	no	N	N	N	N	N	N	N	N	N	68	164	25.3	N	88	16	N	N	6780	458	48	0.8	136	3.9	N	N	N	mild	15	7.3	<1	Y	N
12	abirami	20	f	>1	y	Y	y	no	no	no	y	1	5	y	y	no	no	N	N	N	N	N	N	N	N	N	46	156	18.9	y	98	22	HIGH	y	13400	606	68	3.4	134	2.8	y	N	N	SEVERE	6	6.02		DEATH	y
13	manjula	45	f	>1	no	no	no	no	no	no	no	2	2	N	no	no	y	N	N	Y	N	N	N	Y	N	N	66	152	28.6	N	88	16	N	N	6789	620	60	2.3	135	4.2	N	N	N	moderate	12	7.2	<1	y	N
14	esakiammal	59	f	<1	no	Y	no	Y	no	no	no	2	8	N	no	no	y	N	N	Y	N	N	N	Y	N	N	58	160	22.7	Y	78	16	N	N	10890	560	58	1.8	134	4.5	y	N	N	mild	16	7.29	>2	y	N
15	susheela	58	f	<1	y	y	y	no	no	y	no	2	6	N	no	no	y	N	N	Y	N	N	N	Y	N	N	56	168	19.8	N	78	18	N	y	12300	540	86	3.4	134	4.6	N	N	N	mild	15	7.26	>2	Y	N
16	kovilammal	80	f	>1	no	no	no	y	no	no	no	2	16	N	no	no	Y	N	y	N	N	y	N	y	N	N	63	164	23.4	N	88	24	N	N	5679	490	42	1.8	136	4.7	N	N	N	mild	16	7.28	>2	Y	y
17	mariammal	50	f	<1	y	no	no	Y	no	no	no	2	3	Y	no	no	Y	N	N	N	N	N	N	N	N	N	64	172	21.6	Y	88	18	N	N	7890	460	46	1.4	135	5.3	y	N	N	moderate	12	7.2	<1	Y	N
18	lalitha	55	F	>1	no	no	no	no	no	no	no	2	1	N	no	no	Y	N	N	N	Y	N	N	N	N	N	64	158	25.6	N	88	18	N	N	6780	560	94	2.8	136	4.2	N	N	N	mild	15	7.28	<1	y	N
19	ranjithm	16	F	<1	no	y	y	no	y	no	no	1	4	y	Y	no	no	N	N	N	N	N	N	N	N	N	46	154	19.4	Y	102	22	HIGH	y	12400	568	38	0.8	134	4.3	N	N	N	moderate	11	7.1	>2	y	N
20	bhadrakali	18	f	>1	no	no	y	no	no	no	no	new1	0	Y	no	no	no	N	N	N	N	N	N	N	N	N	64	160	25.0	y	88	16	N	N	8790	560	42	0.9	134	4.1	N	N	N	moderate	12	7.2	<2	Y	N
21	muthulakshmi	48	F	<1	no	no	no	Y	no	no	no	2	3	y	no	no	y	N	N	N	N	N	N	N	N	N	64	160	25.0	y	78	18	N	N	9800	456	52	1.9	134	5.1	y	N	N	moderate	11	7.24	<1	y	N
22	selvi	22	F	<1	no	Y	Y	no	no	no	no	1	4	N	Y	no	no	N	N	N	y	N	N	N	N	N	50	168	17.7	y	96	24	high	Y	16070	560	64	2.1	124	3.8	N	N	N	SEVERE	6	6.22	>2	y	y
23	mery	48	F	<1	no	no	no	no	no	no	no	2	4	Y	no	no	no	N	N	N	N	N	N	N	N	N	64	169	22.4	Y	88	18	N	N	8560	565	52	2.1	134	3.9	N	N	N	mild	15	7.28	<1	Y	N
24	vijayakumari	63	F	<1	no	no	no	no	no	no	no	2	3	N	no	no	Y	N	N	N	N	N	N	N	N	N	64	174	21.1	U	78	14	N	N	5670	650	42	1.4	136	6.2	y	N	N	moderate	10	7.1	>2	Y	N
25	Satyh path	16	F	<1	no	no	no	no	no	no	no	1	2	N	Y	no	no	N	N	N	N	N	N	N	N	N	48	154	20.2	y	86	18	N	N	8900	460	46	1.2	136	3.9	y	N	N	mild	15	7.28	>2	Y	N
26	guruvammal	67	F	>1	no	Y	Y	Y	no	no	no	2	12	Y	no	Y	Y	Y	N	N	N	N	N	N	N	N	62	152	26.8	N	92	24	HIGH	y	14060	520	64	2.3	130	4.9	y	y	N	moderate	10	7.02		DEATH	y
27	mariammal	53	F	>1	no	no	no	no	no	no	no	2	6	Y	no	Y	N	N	N	N	y	y	N	N	N	N	62	160	24.2	y	88	16	N	N	8750	530	43	1.2	132	5.2	N	N	N	mild	16	7.3	<1	Y	N
28	rajalekshmi	54	F	<1	no	no	no	no	no	no	no	2	1	N	no	no	Y	N	N	N	N	N	N	N	N	N	62	156	25.5	N	82	18	N	N	7690	480	38	1.3	132	5.2	N	N	N	moderate	11	7.24	<1	Y	N
29	fathima	22	f	<1	y	y	no	no	no	no	Y	1	3	y	y	no	no	N	N	N	N	N	N	N	N	N	62	164	23.1	y	96	24	high	N	8900	490	46	1.4	136	3.8	N	N	N	moderate	12	7.12	<2	y	N
30	nancy	18	f	>1	no	Y	Y	no	no	no	no	1	2	N	Y	no	no	N	N	N	N	N	N	N	N	N	52	157	21.1	N	94	18	HIGH	N	5680	460	42	1.3	136	3.9	N	N	N	SEVERE	7	6.22	>2	Y	y
31	BANU	19	F	<1	no	no	no	Y	no	no	no	1	4	y	y	no	no	N	N	N	N	N	N	N	N	N	48	154	20.2	Y	88	18	N	N	10200	530	48	1.4	134	4.2	N	N	N	mild	16	7.3	<1	y	N
32	Saritha	48	F	.>1	y	no	no	no	no	no	no	new2	0	Y	no	no	no	N	N	N	N	N	N	N	N	N	48	152	20.8	y	108	24	high	N	8870	520	46	1.2	132	3.2	N	N	N	moderate	12	7.24	<1	y	N
33	RAJAMMAL	75	F	>1	no	y	no	no	no	no	Y	2	8	N	no	Y	y	y	y	N	N	N	y	N	N	N	56	158	22.4	y	92	16	N	N	7990	515	48	1.4	132	4.8	y	N	N	SEVERE	8	6.02	>2	y	y
34	MEENACHI	72	F	<1	no	no	no	Y	no	no	no	2	6	y	no	no	y	Y	N	N	N	N	N	N	N	N	67	156	27.5	y	88	22	N	y	18900	590	64	2.4	126	4.3	y	y	N	moderate	12	7.24	>2	y	N
35	SARASWATHY	65	F	>1	y	no	y	no	no	no	no	2	4	N	no	no	y	N	N	N	N	N	N	N	N	N	60	168	21.3	y	92	22	N	N	5690	650	48	1.8	126	5.7	y	N	N	severe	8	6.28	>2	y	y
36	ASANFATH	18	F	<1	y	no	no	Y	no	no	no	1	3	Y	y	no	no	N	N	N	N	N	N	N	N	N	60	166	21.8	y	84	18	N	N	8790	601	46	1.2	132	4.2	N	N	y	mild	17	7.3	<2	y	y
37	janath	16	f	<1	y	no	y	no	no	no	no	1	2	N	y	no	no	N	N	N	N	N	N	N	N	N	59	148	26.9	N	78	18	N	N	7890	560	44	1.4	132	3.9	N	N	N	moderate	12	7.22	<1	Y	N
38	muppidati	68	f	>1	no	no	y	no	no	y	no	2	12	y	no	no	y	Y	N	N	N	N	N	N	N	N	59	165	21.7	N	70	18	N	N	8960	650	34	1.4	134	6.2	y	N	y	moderate	10	7.1		DEATH	N
39	pitchammai	56	f	<1	no	no	no	no	no	no	no	2	4	y	no	no	no	N	N	N	N	N	y	N	N	N	59	162	22.5	N	92	18	high	N	7850	620	38	1.4	136	4.6	N	N	N	mild	16	7.29	<1	Y	N
40	banumaty	58	f	<1	y	y	y	no	no	no	no	new 2	0	y	no	no	no	N	N	N	N	N	N	N	N	N	59	164	21.9	N	78	18	N	N	6789	560	48	1.5	134	5.2	N	N	N	moderate	12	7.1	<1	y	N
41	Ramalachmi	18	f	<1	no	y	no	no	no	no	no	1	6	N	Y	no	no	N	N	N	N	N	N	N	N	N	58	149	26.1	N	90	16	N	N	7840	480	42	1.5	134	3.2	N	N	N	mild	17	7.3	<1	y	N
42	parumma	48	f	>1	no	no	no	no	no	no	no	NEW2	0	Y	no	no	no	N	N	N	N	Y	N	N	N	N	46	154	19.4	Y	84	18	N	N	7869	450	42	1.4	134	3.9	N	N	N	mild	16	7.3	<1	Y	N
43	sonydas	55	male	>1	y	no	no	no	no	no	no	NEW2	0	Y	no	no	no	N	N	N	N	N	y	y	N	N	58	159	22.9	N	68	16	N	N	8760	460	46	1.4	132	4.2	N	N	N	mild	15	7.3	<1	Y	N
44	petchidevar	65	m	>1	no	no	no	no	no	y	no	2	7	y	no	no	y	N	N	N	y	N	N	N	y	N	58	170	20.1	y	106	22	HIGH	N	22030	520	86	2.3	132	4.6	y	N	N	mild	16	7.29	>2	y	N
45	RAMASAMY	70	m	>1	no	y	y	no	Y	no	no	2	12	N	no	Y	no	y	N	N	N	N	N	N	N	y	58	164	21.6	y	86	16	N	y	14590	659	42	1.4	134	5.2	N	N	N	moderate	10	7.2		DEATH	y
46	MOHAN	52	m	<1	no	no	no	no	no	no	no	2	10	y	no	no	y	N	N	N	y	y	N	Y	Y	58	164	21.6	Y	88	18	high	N	7680	480	44	1.3	136	4.5	N	N	N	moderate	12	7.24	>2	y	N
47	MURUGAN	62	m	<1	y	no	no	no	no	no	no	2	3	N	no	no	y	N	N	N	N	N	N	Y	y	58	164	21.6	y	88	18	N	N	8790	560	54	1.9	134	4.6	y	N	N	mild	15	7.28		DEATH	N
48	chelladurai	50	m	<1	no	no	no	no	no	no	no	2	3	y	no	no	y	N	N	N	N	Y	N	N	Y	58	168	20.5	y	83	16	N	N	9860	560	50	1.2	136	4.8	N	N	N	moderate	12	7.24	<1	y	N
49	mariappan	65	M	<1	no	y	no	no	no	no	Y	2	4	N	no	Y	y	N	N	N	y	N	N	N	y	56	150	24.9	y	98	18	N	N	9870	630	78	1.8	134	6.2	y	N	N	moderate	10	7.22	<1	y	N
50	pandiaraj	70	M	<1	no	y	no	no	no	no	no	2	5	y	no	Y	no	Y	N	N	N	y	N	N	N	y	56	160	21.9	N	88	24	N	N	14320	580	56	1.8	136	4.3	N	N	N	severe	8	6.03	>2	Y	y
51	kankaraj	47	M	<1	y	no	y	no	no	no	no	2	16	y	no	no	y	N	N	N	N	N	N	Y	y	56	148	25.6	N	88	16	N	N	8690	540	42	1.4	128	4.3	N	N	N	mild	15	7.3	<1	Y	N
52	mani	40	M	>1	no	no	no	no	no	y	no	2	2	N	no	no	y	N	N	N	N	N	N	Y	N	N	56	150	24.9	Y	88	18	N	N	8970	560	46	1.3	136	3.2	y	N	N	moderate	10	7.12	<2	y	N
53	murugan	55	M	>1	no	no	no	no	no	no	Y	2	2	N	no	no	y	Y	N	N	N	N	N	y	N	N	56	160	21.9	y	88	28	N	N	7850	546	56	1.9	132	3.1	y	N	N	severe	7	6.03	>2	y	y
54	RAMAR	65	M	<1	y	y	no	no	no	no	no	2	2	Y	no	Y		N	N	N	Y	N	N	y	N	N	56	148	25.6	y	78	18	N	N	9870	620	84	3.8	134	5.3	y	N	N	moderate	12	7.23		DEATH	N
55	DURAIRAJ	58	M	<1	no	no	no	no	no	no	no	2	4	N	no	no	y	N	N	N	N	N	N	N	N	N	56	152	24.2	Y	94	22	high	N	8970	480	46	1.5	136	4.4	N	N	N	mild	16	7.3	<2	Y	N
56	muthu	48	M	<1	y	no	no	no	no	no	no	new 2	0	Y	no	no	no	y	N	N	N	N	N	N	Y	Y	56	154	23.6	Y	86	18	N	N	9800	578	48	1.4	134	4.6	N	N	N	moderate	12	7.22	<1	Y	N
57	MURUGESAN	54	M	>1	no	no	no	Y	no	no	no	2	5	y	no	Y	no	N	N	N	Y	N	N	Y	y	56	160	21.9	y	78	16	HIGH	y	18790	532	54	2.1	134	2.8	N	N	N	moderate	10	7.12	>2	Y	N
58	PAPANASAM	54	M	.>1	Y	Y	Y	no	no	y	no	2	4	y	no	no	y	N	N	N	N	Y	N	y	Y	56	164	20.8	N	86	18	N	N	7860	523	58	1.9	126	4.6	N	N	N	moderate	11	7.23	<2	Y	N
59	RAMASAMY	68	m	<1	y	no	no	no	no	no	no	2	10	N	no	no	y	N	Y	N	N	N	N	N	Y	N	56	159	22.2	N	88	18	N	N	8765	620	56	1.8	132	4.7	N	N	N	moderate	12	7.2		DEATH	N
60	beeran	56	m	>1	Y	Y	Y	no	Y	no	no	2	4	N	no	no	Y	N	N	N	N	Y	N	Y	Y	56	162	21.3	Y	88	24	N	N	9870	570	54	1.4	134	3.2	N	N	N	mild	16	7.29	>2	Y	N
61	surendren	62	m	>1	no	y	no	no	no	y	no	2	3	y	no	no	y	N	N	N	N	Y	N	N	y	56	172	18.9	y	88	22	N	N	7869	470	48	1.4	124	4.5	N	N	N	moderate	12	7.22	>2	y	N
62	esakimutu	56	m	<1	y	no	no	Y	no	no	no	2	7	y	no	no	y	N	N	N	N	N	N	N	Y	56	158	22.4	N	84	16	N	y	22100	563	88	1.8	132	4.6	y	N	N	moderate	10	7.12	>2	Y	N
63	Najipkhan	72	m	>1	no	no	no	no	no	no	no	2	14	N	no	no	y	N	N	N	N	N	N	N	N	N	54	156	22.2	N	78	18	N	N	6780	560	43	1.4	136	4.7	y	N	N	moderate	12	7.23		DEATH	N
64	mydeen	45	m	<1	y	no	no	no	no	no	no	2	1	y	no	Y		N	N	N	N	Y	N	N	y	N	54	160	21.1	N	92	22	N	N	9809	490	47	1.4	132	4.6	N	N	N	moderate	12	7.22	<2	Y	N

65	Prem	48	m	<1	y	no	no	no	no	no	no	2	2	y	no	no	y	N	N	N	N	N	N	N	N	y	54	162	20.6	y	86	18	N	N	8970	489	48	1.4	132	4.7	N	N	N	moderate	12	7.23	<1	y	N
66	Nambirajan	56	m	<1	no	no	no	no	no	no	Y	2	6	N	no	no	y	N	N	N	N	N	N	N	N	Y	52	153	22.2	N	78	16	N	N	8760	482	42	1.5	128	5.1	N	N	N	moderate	12	7.23	<1	y	N
67	muthu	52	m	<1	no	no	y	no	no	no	no	new2	0	Y	no	no		N	N	N	N	N	N	N	N	N	52	159	20.6	N	88	20	N	N	7690	480	43	1.4	134	5.3	y	N	N	mild	15	7.28	<1	y	N
68	Raghu	62	m	<1	y	y	y	no	no	y	no	2	6	y	no	no	y	N	N	N	N	N	N	N	N	Y	50	148	22.8	y	88	18	N	Y	19870	560	56	2.2	132	4.6	y	N	y	severe	8	6.04		DEATH	y
69	samiduri	67	m	>1	y	y	no	no	no	no	no	2	6	N	no	no		N	N	N	N	N	N	N	N	Y	50	156	20.5	y	92	22	N	N	7124	530	46	1.7	134	2.9	N	N	N	mild	16	7.3	<2	Y	N
70	sahulhameed	72	m	>1	y	y	y	no	no	no	no	2	6	Y	no	no	y	Y	N	N	N	N	N	Y	N	Y	48	146	22.5	y	86	16	N	N	7650	520	42	1.4	136	4.3	N	N	N	moderate	12	7.23		DEATH	y
71	kalyanasundaram	68	m	>1	y	y	y	no	no	no	no	2	7	y	no	no		N	N	N	N	N	N	N	N		48	150	21.3	N	103	22	N	y	18940	510	56	1.9	134	4.6	y	N	y	moderate	10	7.22		DEATH	y
72	vadivel	62	m	<1	no	no	no	no	no	no	no	2	2	N	no	no		N	N	N	y	N	N	N	N		48	150	21.3	N	86	16	N	N	8960	540	88	3.9	136	5.9	N	N	N	moderate	11	7.12		DEATH	N
73	chermakani	52	m	>1	y	no	no	no	no	no	no	NEW2	0	Y	no	no		N	N	N	N	N	Y	N	N		48	152	20.8	N	88	16	N	N	9510	460	46	1.5	134	3.2	N	N	N	moderate	12	7.12	<1	Y	N
74	CHINNADURAI	77	m	>1	y	no	no	no	no	y	no	2	10	N	no	Y	y	N	N	N	N	N	N	N	N		48	146	22.5	N	86	16	N	Y	21890	520	64	2.1	136	4.6	y	N	Y	moderate	10	7.24		DEATH	N
75	esakimutu	56	m	<1	y	no	no	no	no	no	no	2	2	y	no	Y	y	N	N	N	N	N	N	N	N	Y	48	156	19.7	y	84	16	N	N	6789	530	43	1.4	128	4.6	N	N	N	moderate	12	7.22	<1	y	N
76	KASIM	76	m	>1	no	no	no	Y	no	no	no	2	12	N	no	Y		N	Y	N	N	N	N	N	Y	Y	48	158	19.2	Y	82	14	N	y	19800	540	68	1.9	134	4.6	y	y	N	moderate	12	7.23	>2	Y	N
77	CHINNAESAKI	58	m	<1	Y	no	no	no	no	no	no	2	3	N	no	no	Y	N	N	N	N	N	N	N	N	Y	48	160	18.8	N	86	18	N	N	5678	567	43	1.4	132	4.2	N	N	N	mild	16	7.28	<1	Y	N
78	THANKARAJ	67	m	>1	no	no	no	no	no	no	no	2	4	N	no	no	Y	N	N	N	N	N	N	N	N	N	46	152	19.9	N	88	16	N	N	6780	450	43	1.4	128	4.8	N	N	N	mild	16	7.29	<1	Y	N
79	MARIMUTU	53	m	<1	no	no	no	no	no	no	no	2	2	Y	no	Y	y	N	N	N	N	N	N	N	N	Y	46	148	21.0	y	106	24	high	N	7860	532	56	1.8	134	3.1	N	N	N	moderate	12	7.12	<1	Y	N
80	PANDIYAN	82	m	>1	no	Y	no	no	no	no	no	2	6	N	no	no	Y	N	N	N	N	N	N	N	N	N	46	162	17.5	Y	96	20	N	N	8970	580	56	1.8	136	4.3	y	N	N	moderate	11	7.22		DEATH	N
81	KOPALAN	58	m	,<1	y	no	no	no	no	no	no	2	7	Y	no	Y	Y	N	N	N	N	N	N	N	N	N	46	152	19.9	y	92	18	N	N	7890	530	43	1.4	134	4.2	N	N	N	moderate	10	7.1	<1	Y	N
82	MURUGAN	52	m	<1	y	no	no	no	no	no	no	2	6	N	no	no	Y	N	N	N	N	N	N	N	N	N	46	152	19.9	Y	78	24	N	N	8760	510	44	1.4	132	5.1	N	N	N	moderate	12	7.23	<1	y	N
83	selvam	68	m	>1	y	no	no	no	no	no	no	2	8	N	no	Y	Y	N	N	N	N	N	N	N	N	Y	46	156	18.9	Y	88	18	N	N	8760	395	50	1.5	134	4.3	N	N	N	moderate	12	7.22	<1	y	N
84	saravanan	79	m	>1	no	y	y	no	no	no	no	2	6	Y	no	no	Y	N	N	N	N	N	N	N	N	Y	43	152	18.6	y	88	18	N	N	9870	590	45	1.3	136	4.5	N	N	N	moderate	11	7.22		DEATH	y
85	sorimutu	18	m	<1	no	no	no	no	no	no	no	1	3	N	y	no	no	N	N	N	N	N	N	N	N	Y	38	148	17.3	Y	86	18	N	N	6789	610	43	1.4	134	3.2	N	N	N	moderate	10	7.23	<1	Y	N
86	ponnuesaki	19	m	<1	no	no	y	no	no	no	no	1	2	N	y	no	no	N	N	N	N	N	N	N	N	Y	42	142	20.8		88	16	N	N	7890	567	42	1.4	136	2.9	N	N	N	moderate	12	7.22	<2	Y	N
87	arun	20	m	<1	y	no	y	no	no	no	no	1	5	N	y	no	no	N	N	N	N	N	N	N	N	Y	48	145	22.8	y	90	18	N	N	6789	567	44	1.4	134	5.2	N	N	N	moderate	12	7.22	<1	Y	N
88	vijay	18	m	<1	no	no	no	no	no	no	no	1	4	Y	y	no	no	N	N	N	N	N	N	N	Y	N	50	156	20.5	Y	78	18	N	N	4567	505	38	1.2	132	4.6	y	N	N	mild	17	7.3	<1	Y	N
89	shek muhamad	16	m	<1	no	no	y	no	no	no	no	1	3	N	y	no	no	N	N	N	N	N	N	N	N	N	48	156	19.7	Y	90	18	N	N	3456	460	43	1.3	134	4.3	N	N	N	mild	16	7.28	<1	Y	N
90	beeran	17	m	<1	no	y	no	no	no	no	no	1	4	N	y	no	no	N	N	N	N	N	N	N	N	N	46	156	18.9	Y	86	18	N	N	5678	450	44	1.3	136	4.5	N	N	N	mild	16	7.28	<1	Y	N
91	tamilhovan	20	m	>1	no	no	y	no	no	no	no	1	5	N	y	no	no	N	N	N	N	N	N	N	N	N	49	158	19.6	Y	78	16	N	N	7680	460	42	1.3	132	4.6	N	N	N	moderate	12	7.22	<1	Y	N
92	sankaran	22	m	>1	no	no	y	no	no	no	no	1	5	N	y	no	no	N	N	N	N	N	N	N	N	N	55	162	21.0	Y	86	18	N	N	6784	520	42	1.3	134	4.7	N	N	N	mild	16	7.3	<1	Y	N
93	selvam	17	m	<1	no	no	y	no	no	no	no	1	2	Y	y	no	no	N	N	N	N	Y	Y	N	N	Y	52	167	18.6	Y	88	24	high	N	7890	530	50	1.4	136	4.6	N	N	N	moderate	10	7.12	>2	y	N
94	pandiraj	14	m	<1	no	no	y	no	no	no	no	1	4	N	y	no	no	N	N	N	N	N	N	N	N	Y	51	168	18.1	Y	82	14	N	N	4560	430	46	1.5	126	3.6	N	N	N	moderate	12	7.22	<2	Y	N
95	amalan	18	m	<1	no	no	y	no	no	no	no	1	3	Y	y	no	no	N	N	N	N	N	Y	N	N	N	52	159	20.6	Y	84	16	N	N	6780	395	46	1.4	132	3.8	N	N	N	moderate	12	7.23	<1	Y	N
96	gobi	19	m	>1	y	no	no	no	Y	no	no	1	4	Y	y	no	no	N	N	N	N	N	N	N	N	N	57	164	21.2	Y	80	18	N	N	7680	394	42	1.4	134	3.8	N	N	N	mild	17	7.3	<1	Y	N
97	vasanth	20	m	<1	y	no	y	Y	no	no	no	1	4	Y	y	no	no	N	N	N	N	N	N	N	N	N	46	157	18.7	N	88	20	N	N	7650	520	43	1.5	136	3.9	N	N	N	mild	15	7.28	<2	Y	N
98	murugan	22	m	>1	y	no	no	no	no	no	Y	1	1	N	y	no	no	N	N	N	N	N	N	N	N	N	48	156	19.7	N	76	16	N	N	8670	450	44	0.9	134	4.6	N	N	N	mild	15	7.29	<2	Y	N
99	vetrivel	22	m	<1	no	no	no	no	no	no	no	1	2	y	y	no	no	N	N	N	N	N	N	N	Y	Y	49	169	17.2	N	78	16	N	N	8790	520	46	0.8	134	4.6	N	N	N	mild	16	7.28	<1	Y	N
100	pandian	23	m	<1	no	no	no	no	no	no	no	1	5	Y	y	no	no	N	N	N	N	N	N	N	Y	Y	52	159	20.6	N	72	18	N	N	5678	389	38	1.2	134	4.9	N	N	N	moderate	12	7.23	<1	Y	N

Compli - Compliance || Temp - Temperature || SIRS - Systemic Inflammatory response syndrome || TC - Total Count