"A COMPARATIVE STUDY OF LONG TERM GLUCOSE CONTROL AND RISK OF SUPERFICIAL SURGICAL SITE INFECTION IN DIABETICS VS NON DIABETICS UNDERGOING GENERAL SURGERY"

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ENDORSEMENT

This is to certify that the dissertation entitled "**A COMPARATIVE STUDY OF LONG TERM GLUCOSE CONTROL AND RISK OF SUPERFICIAL SURGICAL SITE INFECTION IN DIABETICS VS NON DIABETICS UNDERGOING GENERAL SURGERY"** is a bonafide research work done by **REG NO. BHO111002.**

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ABSTRACT

Surgical site infections (SSI) are the infections present in any location along a surgical tract after a surgical procedure. They are the third most common hospital-acquired infection and account for 14% to 16% of all infections. Diabetic patients have shown impaired wound healing and thus have an increased risk of morbidity after surgery from SSI.Tight glucose control in diabetic patients is now widely accepted as the goal in long term management because it demonstrates improvement in microvascular complications. However, the impact of HbA1c on surgical outcome is still being defined. Studies have been done proving increasing HbA1C levels correlated with increasing complications and reduced long term survival in CABG patients. But,there are no previously reported analysis of surgical outcomes of patients based on preoperative HbA1c levels presenting for general surgery.Hence, this study was done to see HbA1c as a predictor of superficial surgical site infection and to know the correlation between them.

AIM:

To compare the HbA1c levels in diabetics and nondiabetics and the risk of surgical site infection after any elective general surgery.

PATIENTS AND METHODS:

All participants diagnosed with type 2 diabetes and equal number of non-diabetics undergoing elective surgical procedures were selected from January 2012 to December 2012.HbA1c levels of diabetic patients were taken within 30days of surgery and compared with HbA1c levels of those of non-diabetics.

Surgical site infection was assessed post operatively at 3^{rd}, 5^{th} , 7^{th}, 15^{th} , 30^{th} days.

RESULTS:

High levels of HbA1c were correlated with rate of SSI and p value was <0.005

CONCLUSION:

Plasma hemoglobin A1c is an indicator of measuring glycaemia over 2-3 months. Increase in this levels has shown to be associated with significantly higher rate of SSI and medical morbidity. Hence, a strict control on glycemia must be performed preoperatively and postoperatively to reduce such complications and to obtain better outcome in diabetic patients.

KEYWORDS: Surgical site infections, Plasma hemoglobin A1c, elective surgery, non-diabetics, diabetics, surgical tract, wound healing, microvascular complications.

TABLE OF CONTENTS

LIST OF TABLES

LIST OF GRAPHS

INTRODUCTION

Presently million surgical procedures are performed each year and trends in nosocomial infections are studied. SSIs holds the third most commonly reported nosocomial infection, accounting for 14% to 16% of all nosocomial infections among hospitalized patients. Among surgical patients, SSIs are the most common nosocomial infection, accounting for 38% of all such infections. Of these SSIs, two thirds are confined to the incision. When surgical patients with nosocomial SSI die, 77% of the deaths are reported to be related to the infection.[1,2]

Advances in infection control practices take account of improved operating room ventilation, sterilization procedures, barriers, surgical technique, and efficient antimicrobial prophylaxis. Despite these actions, SSIs remain a significant cause of morbidity and mortality among hospitalized patients.[1,3]

This may be partially explained by the emergence of antimicrobial-resistant pathogens and the increased numbers of surgical patients who are elderly and/or have a wide variety of chronic, debilitating, or immunocompromising underlying diseases. Thus, to reduce the risk of SSI,a systematic but realistic approach must be applied with the awareness that the risk is also influenced by characteristics ofthe patient. In surgery, patient characteristics have been strongly associated with an increased risk of an SSI.[1]

Among these, diabetes becomes an important consideration because of its high prevalence. Recent preliminary findings from study of patients who underwent coronary artery bypass graft showed a significant relationship between increasing levels of HgA1c and SSI rates but, the contribution of diabetes to SSI risk is controversial, because the independent role of diabetes to SSI risk has not been assessed after controlling for likely confounding factors.[1]Since, more studies are

needed to assess the efficacy of perioperative blood glucose control as a preventive measure, we are carrying out a comparative study of long term glucose control and risk of superficial surgical site infection in diabetics vs nondiabetics undergoing general surgery.

AIM AND OBJECTIVES

To compare the HbA1c levels in diabetics and non-diabetics and the risk of surgical site infection after any elective general surgery.

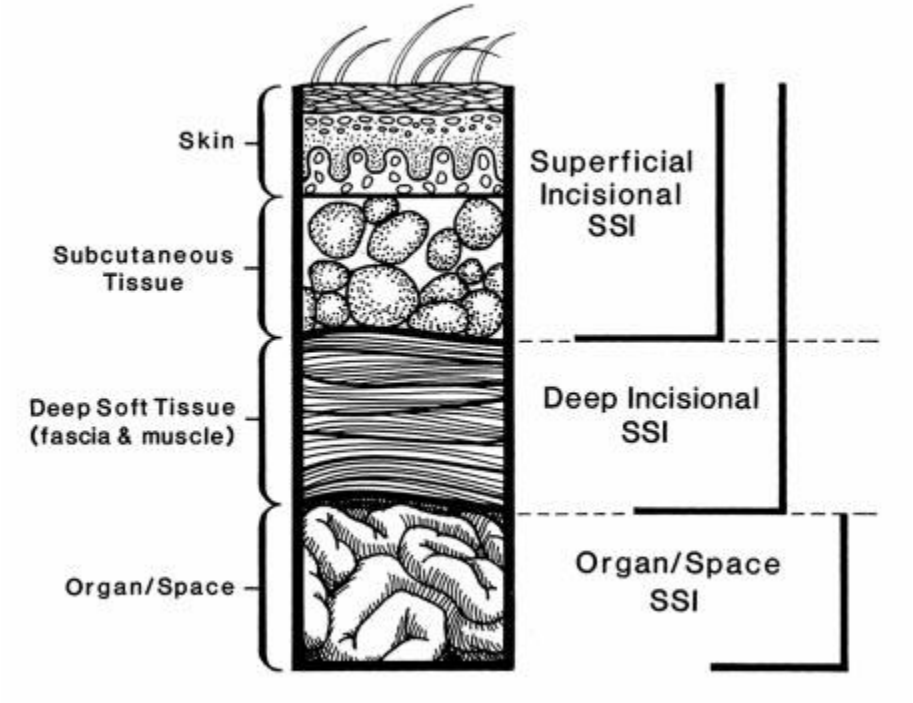
REVIEW OF LITERATURE

Before the mid-19th century, surgical patients often suffered post-operative fever, followed by purulent drainage from the incision sites, sepsis and frequently death. In late 1860s, after Joseph Lister introduced the principles of antisepsis, the postoperative infectious morbidity reduced significantly. Lister's work drastically transformed surgery from an activity related with infection and death to a discipline that could abolish suffering and prolong life. [1]

Presently, million surgical procedures are done each year [1,2] with recognition of nosocomial infections in hospitals. Based on CDC's National Nosocomial Infections Surveillance (NNIS) system reports, SSIs are the third most commonly reported nosocomial infection, accounting for 14% to 16% of all nosocomial infections amongst hospitalized patients. [1,3] Among the surgical patients, Surgical site infection (SSIs) were the most common nosocomial infection, accounting for 38% of them. [1]

The identification of SSI involves understanding of clinical and laboratory findings, and it is essential that a surveillance program uses definitions that are consistent, reliable and standardized. Otherwise, incorrect or uninterpretable SSI rates will be computed and reported. The CDC NNIS definitions of SSIs have been applied constantly by surveillance and surgical personnel in many settings and are a national standard. [4] By these criteria, SSIs are classified as incisional and organ/space. Incisional SSIs are further divided into those involving only skin and subcutaneous tissue (superficial incisional SSI) and those involving deeper soft tissues of the incision (deep incisional SSI). Organ/space SSIs involve any part of the anatomy other than incised body wall layers that was opened or manipulated during the surgery. For example, in a

patient who had an appendectomy and consequently developed an intraabdominal abscess not draining through the incision, the infection will be reported as an organ/space SSI at the intra-abdominal site. Failure to use objective criteria to define SSIs, substantially affects reported SSI rates. [5,6]



Superficial Incisional SSI

- Infection occurs within 30 days after the surgery.
- infection involves only skin or subcutaneous tissue of the incision and at least one of the following:
  - Purulent drainage, with or without laboratory confirmation, from the superficial incision.
  - Organisms isolated from an aseptically obtained culture of fluid or tissue from superficial incision.

- At least one of the following signs or symptoms of infection i.e. pain, localized swelling, redness or heat
- Superficial incision is purposely opened by surgeon, unless incision is culture-negative.
- Diagnosis of superficial incisional SSI by the surgeon or attending physician.

Do not report the following conditions as SSI:

- Stitch abscess (minimal inflammation and discharge restricted to the points of suture penetration).
- Infection of an episiotomy or newborn circumcision site.
- Infected burn wound.
- Incisional SSI that extends into the fascial and muscle layers.

Note: Specific criteria are used for identifying infected episiotomy and circumcision sites and burn wounds. [1,4]

Deep Incisional SSI

- Infection occurs within 30 days after the surgery if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation.
- infection involves deep soft tissues (e.g., fascial and muscle layers) of the incision and at least one of the following:
- Purulent drainage from the deep incision but not from the organ/space component of the surgical site.

- A deep incision spontaneously dehisces or is purposely opened by a surgeon when the patient has at least one of the following signs or symptoms: fever (>38ºC), localized pain, or tenderness, unless site is culture-negative.
- An abscess or other evidence of infection involving the deep incision is found on direct examination, during re-surgery or by histopathologic or radiologic examination.
- Diagnosis of a deep incisional SSI by a surgeon or attending physician.

Notes:

1. Report infection that involves both superficial and deep incision sites as deep incisional SSI.

2. Report an organ/space SSI that drains through the incision as a deep incisional SSI. [1,4]

Organ Space SSI

- Infection occurs within 30 days after the operation if no implant is left in place or within 1 year if implant is in place and the infection appears to be related to the operation.
- infection involves any part of the anatomy (e.g., organs or spaces), other than the incision, which was opened or manipulated during an operation and at least one of the following:
- Purulent drainage from a drain that is placed through a stab wound into the organ/space.
- Organisms isolated from an aseptically obtained culture of fluid or tissue in the organ/space.

- An abscess or other evidence of infection involving the organ/space that is found on direct examination, during reoperation or by histopathologic or radiologic examination.
- Diagnosis of an organ/space SSI by a surgeon or attending physician. [1,4]

According to data from the NNIS system,[8,9] Staphylococcus aureus, coagulase-negative staphylococci, Enterococcus and Escherichia coli remain the most commonly isolated pathogens. A rising fraction of SSIs are caused by antimicrobial-resistant pathogens, such as methicillin-resistant S. aureus (MRSA), [10,11] or by Candida albicans.[12]

Pathogen Sources for SSIs

Endogenous

1. Patient flora

– Skin

– Mucous membranes

– GI tract

2. Seeding from a distant focus of infection

Exogenous

1. Surgical personnel (surgeon and team)

– Soiled attire

– Breaks in aseptic technique

– Inadequate hand hygiene

2. Physical environment and ventilation

3. Tools, equipment, materials brought to operative field

Challenge in Detecting SSIs is attributed to lack of standardized methods for post-discharge/ outpatient surveillance due to -

– Increased number of outpatient surgeries

– Shorter post-op stays

PATIENT AND SURGERY CHARACTERISTICS THAT MAY EFFECT THE RISK OF SURGICAL SITE INFECTION

Patient

Age

Nutritional status

Diabetes

Smoking

Obesity

Coexistent infections at a remote body site

Colonization with microorganisms

Altered immune response

Length of preoperative stay

Surgery

Duration of surgical scrub

Skin antisepsis

Preoperative shaving

Preoperative skin prep

Duration of operation

Antimicrobial prophylaxis

Operating room ventilation

Inadequate sterilization of instruments

Foreign material in the surgical site

Surgical drains

Surgical technique

Poor hemostasis

Failure to obliterate dead space

Tissue trauma [7,13,14]

An SSI prevention measure can be defined as one or many actions intentionally taken to decrease the risk of an SSI. Many techniques are directed at diminishing microbial contamination of the patient's tissues or sterile surgical instruments such as using antimicrobial prophylaxis or avoiding pointless traumatic tissue dissection. Most beneficial application of SSI prevention measures requires a variety of patient and surgery characteristics to be carefully considered. [1]

Modifiable Risk Factors for SSIs

Antimicrobial prophylaxis

– Inappropriate choice (procedure specific)

– Improper timing (pre-incision dose)

– Poor dose based on body mass index, procedures more than 3 hours or increased blood loss

Skin or site preparation ineffective

– Hair removal with razors

Colorectal procedures

– Inadequate bowel prep/antibiotics

– Improper intraoperative temperature regulation

Treat infections at remote site prior to surgery

Inadequate wound dressing protocol

Improper glucose control

Colonization with preexisting microorganisms

Inadequate intraoperative oxygen levels

Out of these, elevated serum glucose levels related with diabetes mellitus is a very well recognized risk factor among the risk factors for SSI.[15-22] It alters host immune responses, causing a well-documented increase in the predisposition to infectious processes. The cumulative effect of age-related immune senescence, can lead to serious and life-threatening infections in elderly patients with DM.

Diabetes Mellitus

Diabetes was one of the earliest diseases described,[23] with Egyptian manuscript from 1500 B.C. .[24] The term "diabetes" or "to pass through" was originally used in 230 BC by the Greek Appollonius of Memphis.[24] The original surviving work with a meticulous reference to diabetes is of Aretaeus of Cappadocia .[25]

Type 1 and type 2 diabetes were recognized as separate conditions for the first time by the Indian physicians Sushruta and Charaka in 400-500 CE with type 1 associated with youth and type 2 with overweight.[24] The term "mellitus" or "from honey" was added by the Briton John Rolle in the late 1700s. Successful treatment was

not developed until the early part of the 20th century, when Canadians Frederick Banting and Charles Herbert Best isolated and purified insulin in 1921 and 1922.[24]

Diabetes mellitus

Universal blue circle symbol for diabetes.[26]

Table 338-1 Etiologic Classification of Diabetes Mellitus
I. Type 1 diabetes
A. Immune-mediated
B. Idiopathic
II. Type 2 diabetes
III. Other specific types of diabetes
A. Genetic defects of cell function characterized by mutations in:
1. Hepatocyte nuclear transcription factor (HNF) 4 (MODY 1)
2. Glucokinase (MODY 2)
3. HNF-1 (MODY 3)
4. Insulin promoter factor-1 (IPF-1; MODY 4)
5. HNF-1 (MODY 5)
6. NeuroD1 (MODY 6)
7. Mitochondrial DNA
8. Subunits of ATP-sensitive potassium channel
9. Proinsulin or insulin conversion
B. Genetic defects in insulin action
1. Type A insulin resistance
2. Leprechaunism
3. Rabson-Mendenhall syndrome
4. Lipodystrophy syndromes
C. Diseases of the exocrine pancreas—pancreatitis, pancreatectomy, neoplasia, cystic fibrosis, hemochromatosis, fibrocalculous pancreatopathy, mutations in carboxyl ester lipase
D.Endocrinopathies—acromegaly, Cushing's syndrome, glucagonoma, pheochromocytoma, hyperthyroidism, somatostatinoma, aldosteronoma
E. Drug- or chemical-induced— pentamidine, nicotinic acid, glucocorticoids, thyroid hormone, diazoxide, -adrenergic agonists, thiazides, phenytoin,, protease inhibitors, clozapine
F. Infections—congenital rubella, cytomegalovirus, coxsackie
G.Rare immune-mediated diabetes—"stiff-person" syndrome, anti-insulin receptor antibodies
H. Genetic syndromes sometimes associated with diabetes—Down's syndrome, Klinefelter's syndrome, Turner's syndrome, Wolfram's syndrome, Friedreich's ataxia, Huntington's chorea, Laurence-Moon-Biedl syndrome, myotonic dystrophy, porphyria, Prader-Willi syndrome

IV. Gestational diabetes mellitus (GDM)

Note: MODY, maturity onset of diabetes of the young. [28]

Globally, as of 2010, an estimated 285 million people had diabetes, with type 2 making up about 90% of the cases.

Type 1 diabetes

Type 1 DM results from the body's failure to produce insulin and was formerly referred to as "insulin-dependent diabetes mellitus" (IDDM) or "juvenile diabetes". Type 1 diabetes mellitus is characterized by loss of the insulin-producing beta cells of the islets of Langerhans in pancreas causing to insulin deficiency. This is classified as immune-mediated or idiopathic. Majority is of the immune-mediated type in which beta cell failure is due to a T-cell-mediated autoimmune attack.[29] A genetic element in individual has been traced to particular HLA genotypes. Even in those who have inherited the susceptibility, type 1 DM seems to need an environmental trigger. The onset of type 1 diabetes is not related to lifestyle. Responsiveness to insulin is normal, especially in the early stages. Type 1 diabetes can affect children, traditionally termed "juvenile diabetes". [29]

"Brittle" diabetes or unstable diabetes or labile diabetes, is a term used to explain the dramatic and repeated swings in glucose levels, occurring for no apparent reason in insulin-dependent diabetes.[30,31]

Type 2 diabetes

Type 2 DM results from insulin resistance. The cells fail to use insulin, sometimes combined with a complete insulin deficiency. This form was previously referred to as non insulin-dependent diabetes mellitus (NIDDM) or "adult-onset diabetes".

A number of lifestyle factors are recognized to be important for the development of type 2 diabetes, including: obesity(body mass index > 30), lack of physical activity, poor diet, stress, and urbanization.[32] Other causes are-

- Genetic defects of β-cell function
    - Maturity onset diabetes of the young
    - Mitochondrial DNA mutations
- Genetic defects in insulin processing or insulin action
    - Defects in proinsulin conversion
    - Insulin gene mutations
    - Insulin receptor mutations
- Exocrine pancreatic defects
    - Chronic pancreatitis
    - Pancreatectomy
    - Pancreatic neoplasia
    - Cystic fibrosis
    - Hemochromatosis
    - Fibrocalculous pancreatopathy

- Endocrinopathies
    - Growth hormone excess (acromegaly)
    - Cushing syndrome
    - Hyperthyroidism
    - Pheochromocytoma
    - Glucagonoma
- Infections
    - Cytomegalovirus infection
    - Coxsackievirus B
- Drugs
    - Glucocorticoids
    - Thyroid hormone
    - β-adrenergic agonists
    - Statins [28]

Comparison of type 1 and 2 diabetes[32]

Feature	Type 1 diabetes	Type 2 diabetes
Onset	Sudden	Gradual
Age at onset	Mostly in children	Mostly in adults
Body habitus	Thin or normal[33]	Often obese
Ketoacidosis	Common	Rare
Hyperosmolar state	Rare	Common
Autoantibodies	Usually present	Absent
Endogenous insulin	Low or absent	Normal, decreased or increased
Concordance in identical twins	50%	90%
Prevalence	~10%	~90%

Gestational diabetes

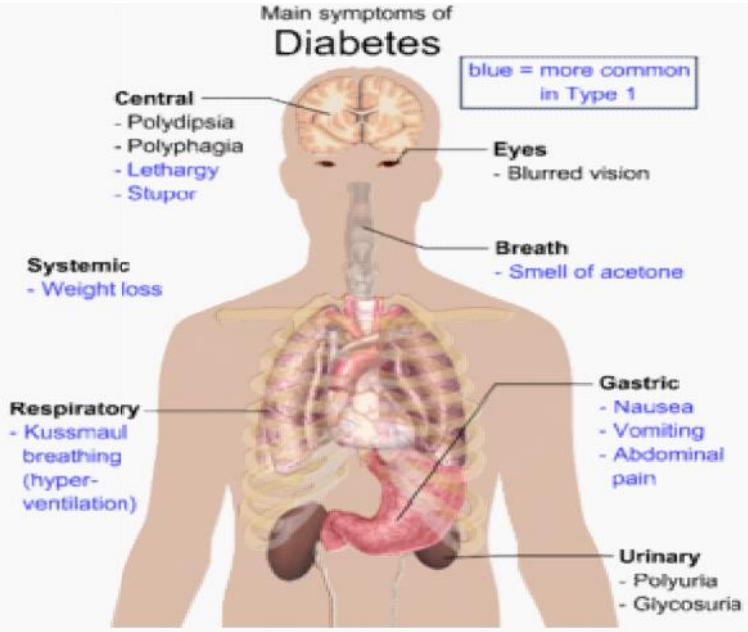
Gestational diabetes mellitus (GDM) resembles type 2 diabetes, involving a combination of relatively inadequate insulin secretion and responsiveness of receptors. It occurs in about 2–5% of all pregnancies and may improve after delivery. Gestational diabetes is completely treatable, but requires careful medical supervision throughout the pregnancy. About 20–50% of affected women develop type 2 diabetes later in life.

Though it may be transient, if untreated, it can damage the health of the fetus or mother. Risks to the baby include macrosomia, congenital cardiac and central nervous system defects, skeletal muscle malformations, respiratory distress syndrome and perinatal death may occur due to poor placental perfusion due to vascular impairment.

Other types

Prediabetes is a condition with a person's blood glucose levels elevated than normal but not high enough for a diagnosis of type 2 DM. Many people destined to develop type 2 DM live many years in a state of prediabetes which has been termed as "America's largest healthcare epidemic."[34,35]

Signs and symptoms



Overview of the most significant symptoms of diabetes

Complications

Table 338-7 Chronic Complications of Diabetes Mellitus
Microvascular
Eye disease
Retinopathy (nonproliferative/proliferative)
Macular edema
Neuropathy
Sensory and motor (mono- and polyneuropathy)
Autonomic
Nephropathy
Macrovascular
Coronary artery disease

Peripheral arterial disease
Cerebrovascular disease
Other
Gastrointestinal (gastroparesis, diarrhea)
Genitourinary (uropathy/sexual dysfunction)
Dermatologic
Infectious
Cataracts
Glaucoma
Periodontal disease

[28]

Although chronic hyperglycemia is an significant etiologic factor leading to complications of DM, the mechanism by which it leads to such diverse cellular and organ dysfunction is unknown. Four prominent theories have been proposed to explain how hyperglycemia leads to the chronic complications of DM. [28]

One theory is that raised intracellular glucose leads to the formation of advanced glycosylation end (AGE) products through the nonenzymatic glycosylation of intra- and extracellular proteins that results from the interaction of glucose with amino groups on proteins. The serum level of AGEs correlates with the level of glycemia and these products gather as glomerular filtration rate declines.

A second theory is based on the observation that hyperglycemia raises glucose metabolism via the sorbitol pathway. Intracellular glucose is predominantly metabolized by phosphorylation and consequent glycolysis, but when raised, some glucose is converted to sorbitol by the enzyme aldose reductase. Raised sorbitol concentration alters redox potential, increases cellular osmolality, thus, generates reactive oxygen species.

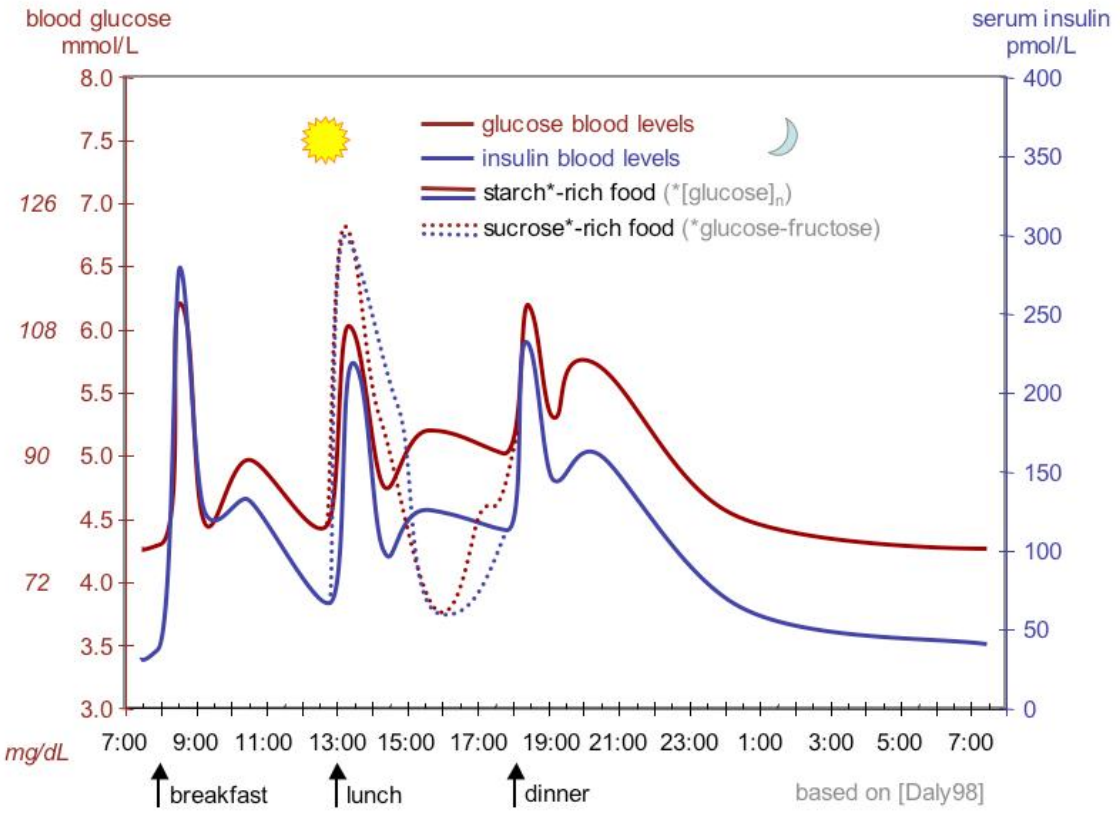
A third hypothesis proposes that hyperglycemia accelerates the formation of diacylglycerol leading to activation of protein kinase C (PKC). PKC alters the transcription of genes for fibronectin, type IV collagen, contractile proteins, and extracellular matrix proteins in endothelial cells and neurons.

A fourth theory proposes that hyperglycemia increases the flux through the hexosamine pathway, which generates fructose-6-phosphate, a substrate for O-linked glycosylation and proteoglycan production. The hexosamine pathway may modify function by glycosylation of proteins such as endothelial nitric oxide synthase or by changes in gene expression of transforming growth factor or plasminogen activator inhibitor-1 (PAI-1).

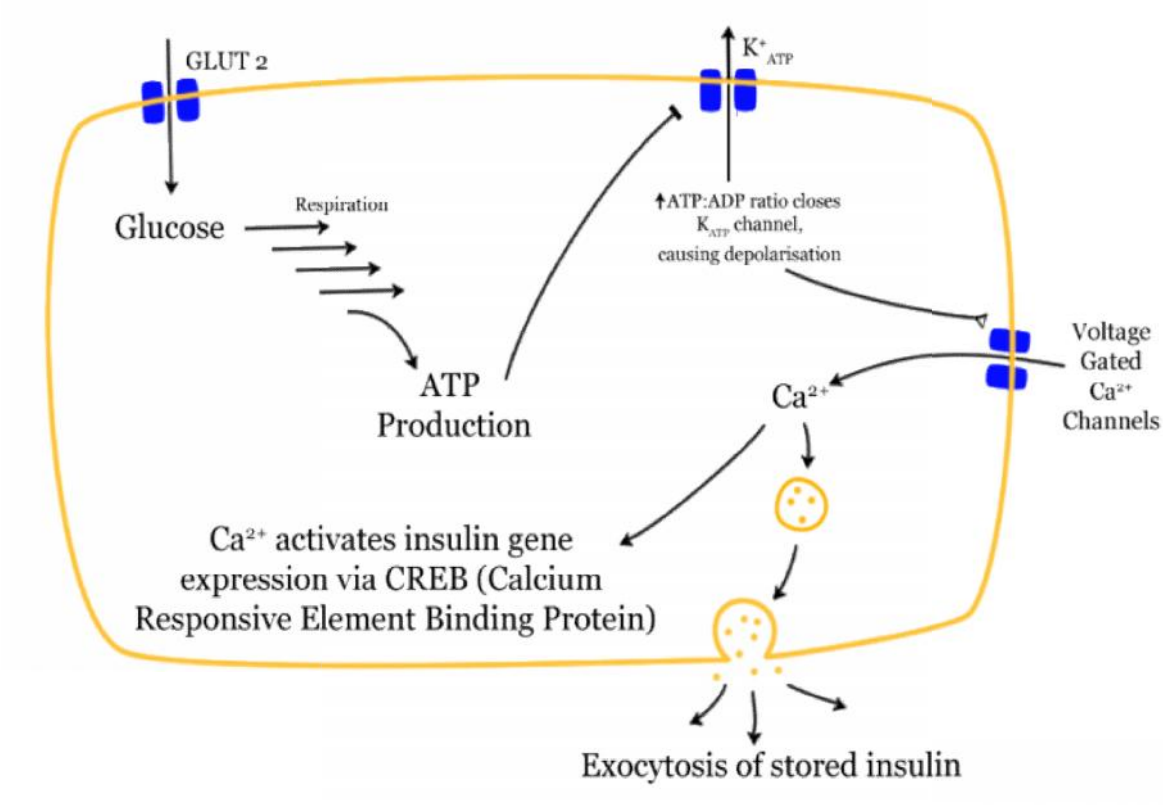
Growth factors appear to play an important role in DM-related complications, and their production is amplified by most of these proposed pathways. Vascular endothelial growth factor A (VEGF-A) is increased locally in diabetic proliferative retinopathy and reduces after laser photocoagulation. TGF is raised in diabetic nephropathy and stimulates basement membrane production of collagen and fibronectin by mesangial cells. [28]

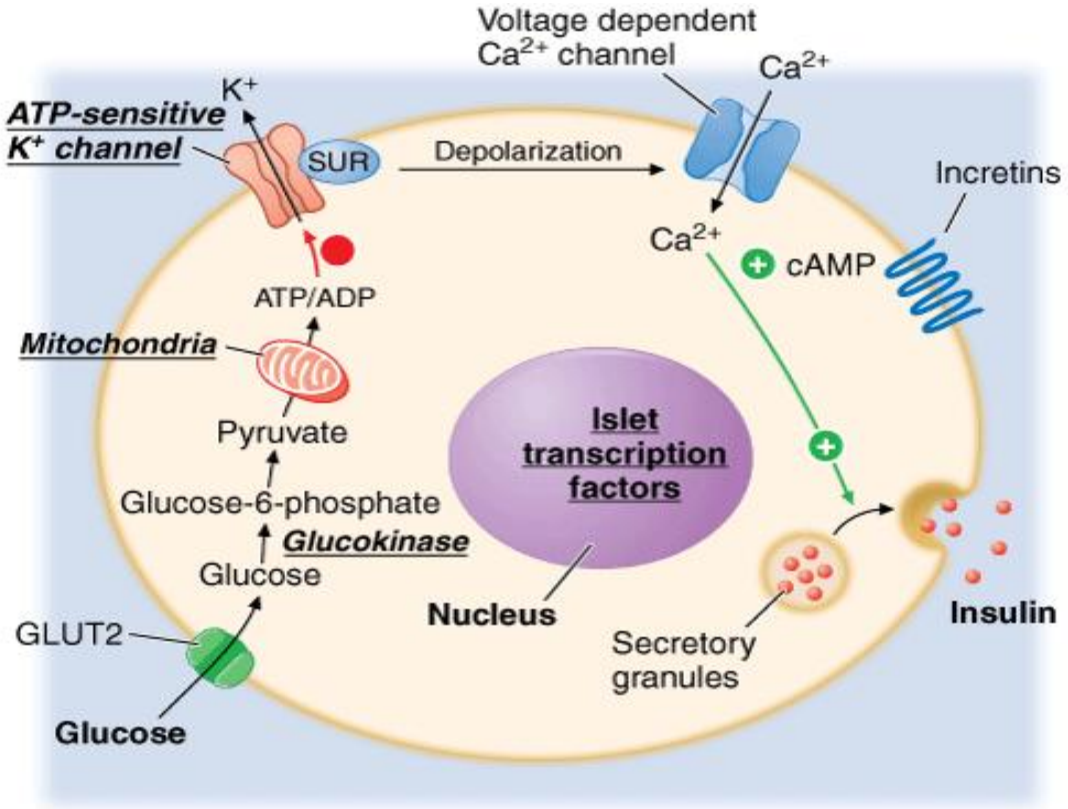
Individuals with DM have a greater incidence and severity of infection. The reasons for this include partly defined abnormalities in cell-mediated immunity and phagocyte function associated with hyperglycemia, as well as diminished vascularization. Diabetic patients also have a greater risk of postoperative wound infections. Strict glycemic control reduces postoperative infections in diabetic individuals and should be the goal in all diabetic patients with an infection. [28]

Pathophysiology-



The fluctuation of blood sugar (red) and the sugar-lowering hormone insulin (blue) in humans during the course of a day with three meals - one of the effects of a sugar-rich vs a starch-rich meal is highlighted.


GLUT 2
K^+_{ATP}
Glucose
Respiration
↑ATP:ADP ratio closes K_{ATP} channel, causing depolarisation
Voltage Gated Ca^{2+} Channels
ATP Production
Ca^{2+}
Ca^{2+} activates insulin gene expression via CREB (Calcium Responsive Element Binding Protein)
Exocytosis of stored insulin


Voltage dependent Ca^{2+} channel
Ca^{2+}
K^+
ATP-sensitive K^+ channel
SUR
Depolarization
Incretins
Ca^{2+}
cAMP
ATP/ADP
Mitochondria
Pyruvate
Islet transcription factors
Glucose-6-phosphate
Glucokinase
Glucose
Nucleus
Insulin
GLUT2
Secretory granules
Glucose

Glucose is the key regulator of insulin secretion by the pancreatic beta cell, although amino acids, ketones, various nutrients, gastrointestinal peptides, and neurotransmitters also influence insulin secretion. Glucose levels > 3.9 mmol/L (70 mg/dL) stimulate insulin synthesis, primarily by enhancing protein translation and processing. Glucose stimulation of insulin secretion begins with its transport into the beta cell by the GLUT2 glucose transporter. Glucose phosphorylation by glucokinase is the rate-limiting step that controls glucose-regulated insulin secretion. Further metabolism of glucose-6-phosphate via glycolysis generates ATP, which inhibits the activity of an ATP-sensitive K^+ channel. This channel consists of two separate proteins: one is the binding site for certain oral hypoglycemics (e.g., sulfonylureas, meglitinides); the other is an inwardly rectifying K^+ channel protein. Inhibition of this K^+ channel induces beta cell membrane depolarization, which opens voltage-dependent calcium channels (leading to an influx of calcium), and stimulates insulin secretion. Insulin secretory profiles reveal a pulsatile pattern of hormone release, with small secretory bursts about every 10 min, superimposed upon greater amplitude oscillations of about 80–150 min. Incretins are released from neuroendocrine cells of the gastrointestinal tract following food ingestion and increase glucose-stimulated insulin secretion and suppress glucagon secretion. Glucagon-like peptide 1 (GLP-1), the most effective incretin, is released from L cells in the small intestine and stimulates insulin secretion only when the blood glucose is above the fasting level. Incretin analogues, such as exena-tide, are being used to improve endogenous insulin secretion. [28]

Lowered glucose levels result both in the reduced release of insulin from the β-cells and in the reverse conversion of glycogen to glucose when glucose levels decrease.

This is mainly controlled by the hormone glucagon, acting opposite to insulin. Glucose thus forcibly produced from internal liver cell stores as glycogen, re-enters the bloodstream. Normally, liver cells do this when the level of insulin is low (which normally correlates with low levels of blood glucose).

Higher insulin levels increase some anabolic processes, such as cell growth and duplication, protein synthesis, and fat storage. Insulin is the principal signal in converting many of the bidirectional processes of metabolism from a catabolic to an anabolic and vice versa. A low insulin level is the trigger for entering or leaving ketosis .

If the amount of insulin available is insufficient, defective or if cells have insulin insensitivity or resistance, then glucose will not have its usual effect, so it will not be absorbed by the cells that require it, nor will it be stored in the liver and muscles. The net effect is constant high levels of blood glucose, poor protein synthesis, and other metabolic derangements, such as acidosis.

Diagnosis

Table 338-2 Criteria for the Diagnosis of Diabetes Mellitus
• Symptoms of diabetes plus random blood glucose concentration 11.1 mmol/L (200 mg/dL)[a] *or* • Fasting plasma glucose 7.0 mmol/L (126 mg/dL)[b] *or* • Two-hour plasma glucose 11.1 mmol/L (200 mg/dL) during an oral glucose tolerance test[c] • Glycated hemoglobin (Hb A1C) ≥ 6.5%

[a]Random is defined as without regard to time since the last meal.

[b]Fasting is defined as no caloric intake for at least 8 h.

[c]The test should be performed using a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water; not recommended for routine clinical use. [28]

A positive result, in the absence of unequivocal hyperglycemia, should be confirmed by a repeat of any of the above methods on a different day. It is preferable to measure a fasting glucose level due to the ease of measurement and the time commitment of formal glucose tolerance testing, which takes two hours to complete with no prognostic advantage over the fasting test.[35-41] According to the current definition, two fasting glucose measurements above 126 mg/dl (7.0 mmol/l) is considered diagnostic for diabetes mellitus.

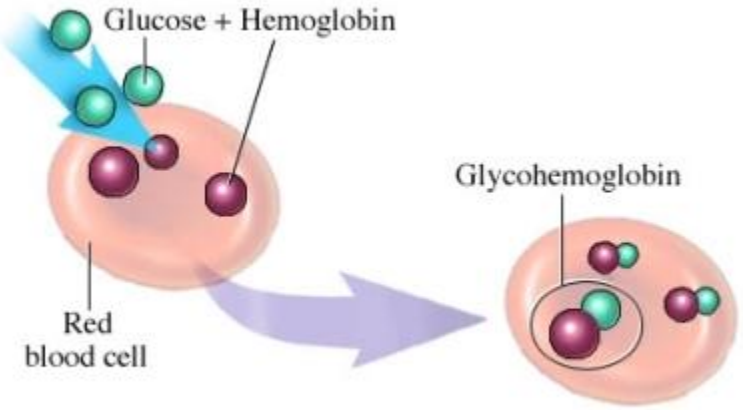
People with fasting glucose levels from 110 to 125 mg/dl (6.1 to 6.9 mmol/l) are said to have impaired fasting glucose.[42] Patients with plasma glucose at or above 140 mg/dL (7.8 mmol/L), but not over 200 mg/dL (11.1 mmol/L), two hours after a 75 g oral glucose load are said to have impaired glucose tolerance.[43]

Glycated hemoglobin is better than fasting glucose for determining long term status and risks of cardiovascular disease and death from any cause.[44]

Glycated hemoglobin (HbA1c)

It is a form of hemoglobin that is measured principally to identify the plasma glucose concentration over prolonged periods of time. It is formed in a non-enzymatic glycation pathway by hemoglobin's exposure to plasma glucose. Normal levels of glucose create a normal amount of glycated hemoglobin. As the average amount of plasma glucose rises, the fraction of glycated hemoglobin rises in a expected way. This serves as a marker for average blood glucose levels over the earlier months prior to the measurement. Monitoring HbA_{1c} in type 1 diabetic patients may improve outcome[45]

Hemoglobin A1c was first separated from other forms of hemoglobin by Huisman and Meyering in 1958 with a chromatographic column.[46] Its rise in diabetes was first described in 1969 by Samuel Rahbar et al.[47,48] The use of hemoglobin A1c for monitoring the control of glucose metabolism in diabetic patients was proposed in 1976 by Anthony Cerami, Ronald Koenig and coworkers.[49,50]



Measurement of glycated hemoglobin is the standard method for assessing long-term glycemic control. When plasma glucose is consistently elevated, there is an increase

in nonenzymatic glycation of hemoglobin; this alteration reflects the glycemic history over the previous 2–3 months, since erythrocytes have an average life span of 120 days (glycemic level in the preceding month contributes about 50% to the A1C value). There are numerous laboratory methods for measuring the various forms of glycated hemoglobin, and these have significant interassay variations. Since glycated hemoglobin measurements are usually compared to prior measurements, it is essential for the assay results to be comparable.

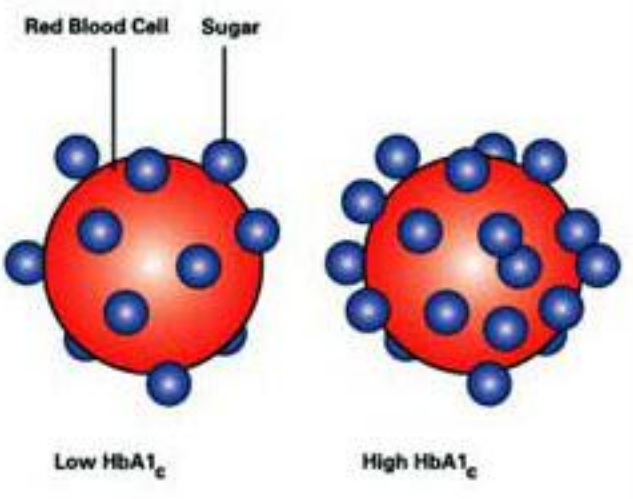
A 1% rise in the A1C translates into a 2.0-mmol/L (35 mg/dL) increase in the mean glucose. In patients achieving their glycemic goal, the ADA recommends measurement of the A1C at least twice per year. More frequent testing (every 3 months) is warranted when glycemic control is inadequate, when therapy has changed, or in most patients with type 1 DM. The degree of glycation of other proteins, such as albumin, can be used as an alternative indicator of glycemic control when the A1C is inaccurate (hemolytic anemia, hemoglobinopathies). The fructosamine assay (measuring glycated albumin) reflects the glycemic status over the prior 2 weeks. Alternative assays of glycemic control (including the 1,5 anhydroglucitol assay) should not be routinely used since studies demonstrating that it accurately predicts the complications of DM are lacking.

Principle

Glycation of hemoglobin, a nonenzymatic reaction occurs between glucose and the N-end of the beta chain. This forms a Schiff base which is itself transformed to 1-deoxyfructose. This reorganization is known as Amadori rearrangement.

When blood glucose levels are high, glucose molecules bind to the hemoglobin in red blood cells. The longer hyperglycemia occurs in blood, the more glucose binds to hemoglobin in the red blood cells and the higher is the glycated hemoglobin level.

Once a hemoglobin molecule is glycated, it remains as it is. A buildup of glycated hemoglobin within the red cell, thus, reflects the average level of glucose to which the cell has been exposed during its life-cycle. Measuring glycated hemoglobin assesses the efficiency of therapy by monitoring long-term serum glucose regulation. The HbA_{1c} level is proportional to average blood glucose concentration over the preceding four weeks to three months. Some studies state that the major proportion of its value is weighted toward the most recent 2 to 4 weeks.[51]



Measuring HbA1C

A number of techniques are used to measure HbA1C.

Laboratories use:

- High-performance liquid chromatography (HPLC): The HbA1c result is calculated as a ratio to total hemoglobin by using a chromatogram.
- Immunoassay
- Enzymatic
- Capillary electrophoresis
- Boronate affinity chromatography [52-54]

Interpretation of results

Laboratory results may differ with the analytical technique, age of the subject, and biological variation among individuals. Two individuals having same average blood sugar can have A1C values that differ by as much as 3 percentage points. Results can be variable in many circumstances, such as after blood loss, high erythrocyte turnover, in the presence of chronic renal or liver disease, after administration of high-dose vitamin C or erythropoetin.[55]In general, the reference range is 20–40 mmol/mol (4–5.9%).[56]

The International Diabetes Federation and American College of Endocrinology recommend HbA_{1c} values below 48 mmol/mol (6.5%), where as American Diabetes Association recommends below 53 mmol/mol (7.0%).[57] Recent results from large trials suggest that a target below 53 mmol/mol (7%) may be excessive and the intensive glycemic control required to reach this level leads to an increased rate of unsafe hypoglycemic episodes.[58,59]

HbA_{1c}		**eAG (estimated average glucose)**	
(%)	**(mmol/mol)**[24]	**(mmol/L)**	**(mg/dL)**
5	31	5.4 (4.2–6.7)	97 (76–120)
6	42	7.0 (5.5–8.5)	126 (100–152)
7	53	8.6 (6.8–10.3)	154 (123–185)
8	64	10.2 (8.1–12.1)	183 (147–217)
9	75	11.8 (9.4–13.9)	212 (170–249)
10	86	13.4 (10.7–15.7)	240 (193–282)
11	97	14.9 (12.0–17.5)	269 (217–314)
12	108	16.5 (13.3–19.3)	298 (240–347)
13	119	18.1 (15–21)	326 (260–380)
14	130	19.7 (16–23)	355 (290–410)
15	140	21.3 (17–25)	384 (310–440)
16	151	22.9 (19–26)	413 (330–480)
17	162	24.5 (20–28)	441 (460–510)
18	173	26.1 (21–30)	470 (380–540)
19	184	27.7 (23–32)	499 (410–570)

[55,60]

Indications and use

Glycated hemoglobin testing is recommended for both

(a) checking the blood sugar control in people who might be pre-diabetic.

(b) monitoring blood sugar control in patients with diabetes mellitus.

For a single blood sample, it gives far more information on glycemic behavior than a fasting blood sugar value. However, fasting blood sugar tests are essential in making management decisions. The American Diabetes Association guidelines are similar to others in advising that the glycated hemoglobin test be performed at least two times a year in patients with diabetes that are meeting management goals and quarterly in patients with diabetes whose treatment has changed or that are not meeting glycemic goals.[61,62]

All the chief institutions like International Expert Committee Report, drawn from the International Diabetes Federation (IDF), the European Association for the Study of diabetes (EASD), and the American Diabetes Association (ADA), suggests the A1C level of 48 mmol/mol (6.5%) as a diagnostic level.[63] The Committee Report states that when A1C testing cannot be performed, the fasting and glucose tolerance tests be done.

Management of Diabetes

Treatment concentrates on keeping blood sugar levels as close to normal, euglycemic state without causing hypoglycemia. This can generally be accomplished with diet, exercise, and use of appropriate medications.

Table 338-8 Treatment Goals for Adults with Diabetes	
Index	**Goal**
Glycemic control[b]	
HbA1C	<7.0[c]
Preprandial capillary plasma glucose	5.0–7.2 mmol/L (90–130 mg/dL)
Peak postprandial capillary plasma glucose	<10.0 mmol/L (<180 mg/dL)[d]
Blood pressure	<130/80[e]
Lipids[f]	
Low-density lipoprotein	<2.6 mmol/L (<100 mg/dL)
High-density lipoprotein	>1.1 mmol/L (>40 mg/dL)[g]
Triglycerides	<1.7 mmol/L (<150 mg/dL)

[a]As recommended by the ADA; Goals should be developed for each patient. Goals may be different for certain patient populations.

[b] HbA1C is primary goal.

[c]While the ADA recommends an A1C < 7.0% in general, in the individual patient it recommends an ". . . HbA1C as close to normal (<6.0%) as possible without significant hypoglycemia. . . ." Normal range for HbA1C—4.0–6.0 [d] One-two hours after beginning of a meal.

[e]In patients with reduced GFR and macroalbuminuria, the goal is <125/75.

[f]In decreasing order of priority.

[g]For women, some suggest a goal that is 0.25 mmol/L (10 mg/dL) higher. [28]

Patient education, perception, and participation is vital, since the complications of diabetes are less common and less severe in people who have well-managed blood sugar levels.[64,65] The goal of treatment is an HbA1C level of 6.5%.[66] Consideration is also paid to other health problems that may accelerate the deleterious effects of diabetes such as smoking, elevated cholesterol levels, obesity, high blood pressure, and lack of regular exercise.[66,67]

It has been observed that diabetic patients with poor postoperative glycemic control (defined as a mean 48-h postoperative capillary glucose (MCG) >11.0 mmol/L or 200 mg/dL) have 2.5 times higher risk for having surgical site infection than those with a 48-h MCG < or =11.0 mmol/L.

The contribution of diabetes to SSI risk is controversial, because the independent contribution of diabetes to SSI risk has not typically been assessed after controlling for potential confounding factors. Findings from study of patients who underwent coronary artery bypass graft showed a significant relationship between increasing levels of HbA1c and SSI rates. [20] Also, increased glucose levels (>200 mg/dL) in the immediate postoperative period (<48 hours) were associated with increased SSI risk. [21,22] Studies are done to assess the efficacy of perioperative blood glucose control as a prevention measure but we also need to know if long term high glucose levels predicted as increase the rate of SSI .

MATERIAL & METHODS

Patients of all age groups fulfilling the inclusion criteria and coming to the emergency of KLE'S Dr. Prabhakar Kore Charitable Hospital were included in the study. Informed consent was taken from the subjects. History was taken and routine general and systemic examination was done. A detailed history was recorded. Blood sample was collected from antecubital vein immediately after admission. Subsequently, the subjects were treated according to the standard treatment protocol of our hospital. The blood samples were immediately taken to the laboratory and HbA1c levels estimated along with other investigations.

The study design was a Cross sectional study.

The sample Size was 60 patients. Equal number of patients, that is, 30 in each group were taken with type 2 diabetes and non-diabetics undergoing elective surgical procedures from January 2012 to December 2012.

Inclusion Criteria:

All participants with Type 2 Diabetes Mellitus (age30 -70 years) and equal number of nondiabetics admitted under General Surgery care in KLES Prabhakar Kore Hospital, Belgaum for definitive surgery.

Exclusion Criteria:

1) Immunocompromised patients
2) Patients with co-morbidities like severe anemia, renal failure, cardiac diseases, bleeding disorders
3) Emergency surgical procedures

All consented patients fulfilling the inclusion criteria were interviewed using a pre-designed questionnaire for relevant detailed history including treatment history, history of drug intake, and examined for all important physical findings.

The primary data for this study was to carry out the blood investigations of these patients. Blood sample was collected from antecubital vein and the following tests were carried out for participants undergoing elective surgery.

Investigations performed were:-

Investigations	Method
Haemoglobin estimation	Automated cell counter
Red blood cell count	
Hematocrit	
Total leucocyte count	
Differential leucocyte count	
Platelet count	
Erythrocyte Sedimentation Rate	Westergren method
Peripheral smear	Wright staining

Other Important Investigations-

- Blood Glucose
- Serum Urea
- Blood Urea Nitrogen
- Serum Creatinine

S.electrolytes

- Serum Sodium
- Serum Potassium
- Serum Chloride
- Serum Bicarbonate

Liver Function Tests-

- Total Bilirubin
- Direct and indirect Bilirubin
- SGOT and SGPT
- Alkaline phosphate
- Total Proteins

HbA1c level

Seropositivity for HbsAg

Seropositivity for HIV

Urine examination (routine & microscopy) was also done on urine samples of all.

The comparative outcomes were tabulated and risk of infection was assessed.

Analysis-

HbA1c levels of diabetic patients was done within 30 days of surgery with those of non-diabetics.The results of HbA1c levels were grouped as ‘Normal’ or ‘High’ or ‘Raised’ as per the below reference levels:

Reference Range of HbA1c

Criteria	Range
Normal	<7%
High	>7-8.9%
Very High	>9%

Wound site was assessed under the following –

Grades	Appearances		Interval				
			Day 3	Day 5	Day 7	Day 15	Day 30
0		**Normal Healing**					
I		**Normal Healing with mild bruising or erythema**					
	A	Some bruising					
	B	Considerable bruising					
	C	Mild erythema					
II		**Erythema Plus Other signs of inflammation**					
	A	At one point					
	B	Around sutures					
	C	Along wound					
	D	Around wound					
III		**Clear or Heamoserous dis charge**					
	A	At one point only (<2cm)					
	B	Along wound (2cm)					
	C	Large Volume					
	D	Prolonged (>3 Days)					
IV		**Major Complications**					
	A	At one point (<2cm)					
	B	Along wound (2cm)					
V		**Deep severe wound infection with or without tissue breakdawn; Haematoma requiring aspiration**					

The discharge was sent for culture and sensitivity. The report was interpreted as Positive or Negative. If positive then, following details were taken into account.

Name of the organism ----------

Sensitive to ----------

OBSERVATION & RESULTS

Out of 60 patients, 24 were females and 36 were males. The age of females ranged from 40-68 years, the mean age being 52.54 years. The age of males ranged from 40-75 years, the mean age being 54.9 years. Combining the males and females, the age ranged from 40-75 years with the average age being 54.9 years. Thirty diabetics and thirty non-diabetics were studied. Among diabetics, 19 were males and 11 were females. Among non-diabetics, 17 were males and 13 were females.

Table 1.Sex ratio of the patients

Sex	No.of patients	Percentage (%)
Males	36	60
Females	24	40
Total	60	100

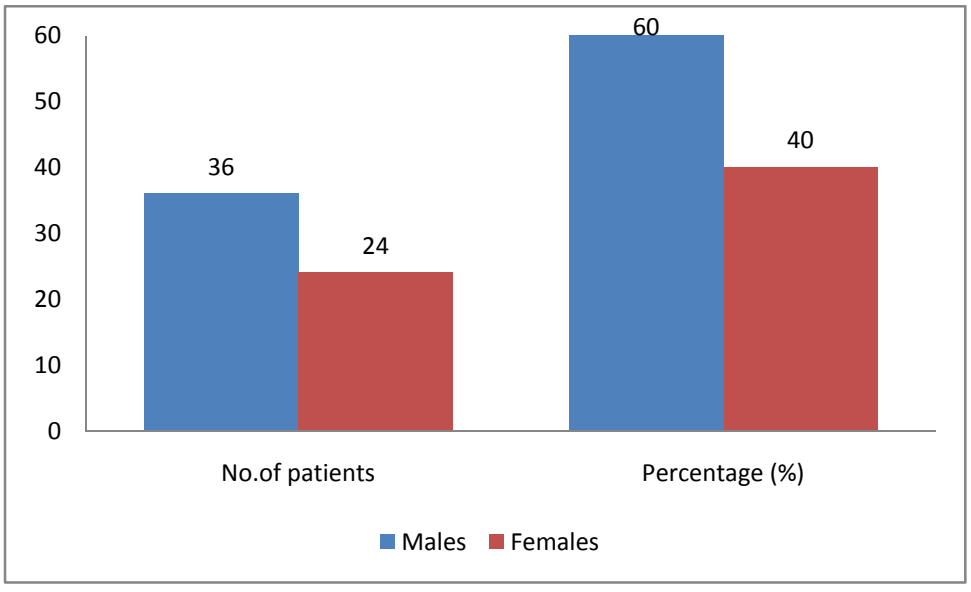


Figure 1 Sex ratio of the patients

Table 2.Male-Female ratio in diabetics and non-diabetics

Disease	Total no. of cases	No. of males	No. of females	M:F
Diabetics	30	19	11	1.72:1
Non-diabetics	30	17	13	1.31:1

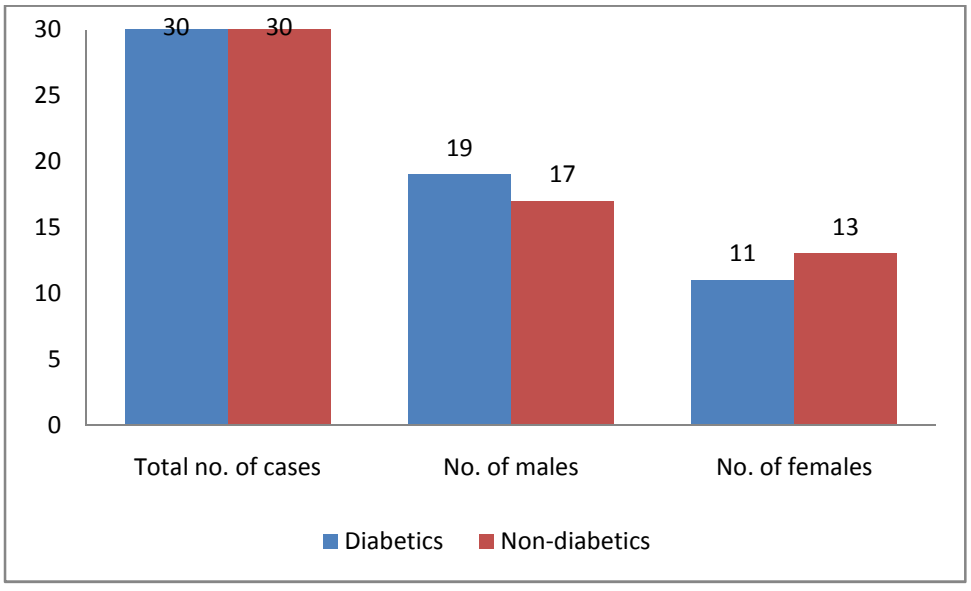


Figure 2 Male-Female ratio in diabetics and non-diabetics

Table 3.Range of Age and Mean Age of the patients

Sex	Age Range	Mean age
Males	40-75 yrs	54.9 yrs
Females	40-68 yrs	52.54 yrs
Overall	40-75 yrs	54.9yrs

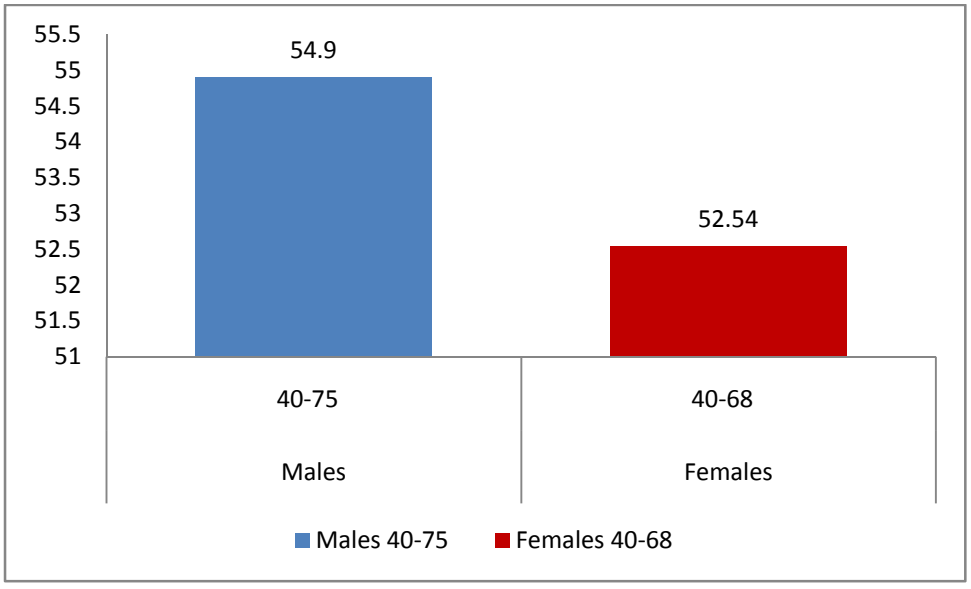


Figure 3 Range of Age and Mean Age of the patients

Table 4.Age distribution of the patients

Age group	No. of patients
0-10yrs	0
11-20yrs	0
21-30yrs	0
31-40yrs	3
41-50yrs	18
51-60yrs	25
61-70yrs	10
71-80yrs	4
81-90yrs	0

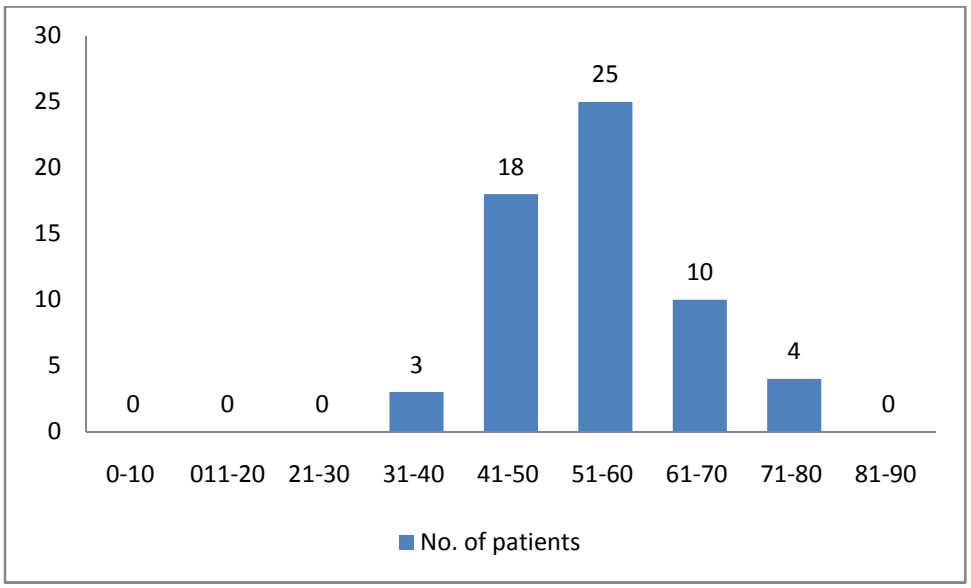


Figure 4 Age distribution of the patients

Table 5. HbA1C levels of all patient

Patients	Patients with HbA1C levels ≤7%	Patients with HbA1C levels >7%
Diabetics (30 Patients)	8	22
Non-Diabetics (30 Patients)	30	0
Total 60	38	22

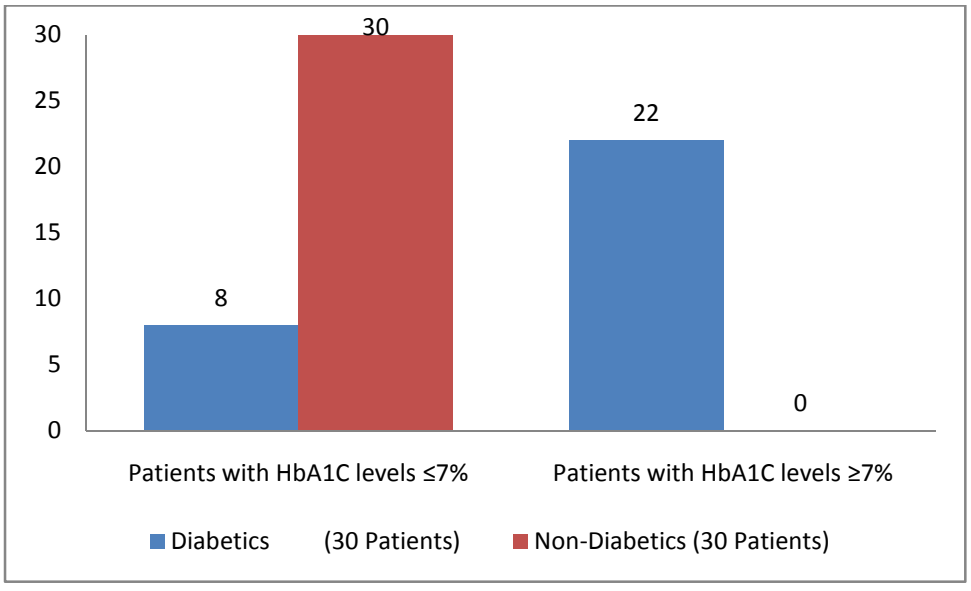


Figure 5 HbA1C levels of all patient

Table 6: Surgical Site Infection in Diabetic Patients with Different HbA1C Values

Sl. No	RBS Values (mg/dL)	HbA1C in Diabetic patients (%)	Surgical Site Infection	No Surgical Site Infection
1	187	8.9	+	
2	190	9.2	+	
3	175	8.5		+
4	135	6.6		+
5	181	9.1	+	
6	136	6.6		+
7	170	9.0	+	
8	183	8.6	+	
9	179	8.5		+
10	130	6.7		+
11	144	7		+
12	196	9.7	+	
13	185	8.3		+
14	187	8.3		+
15	190	9.2	+	
16	177	8.4		+
17	140	7.0		+
18	186	8.6	+	
19	195	10.0	+	
20	141	7.0		+
21	200	10.0	+	

22	172	8.5		+
23	194	9.6	+	
24	150	7.0	+	
25	187	8.7		+
26	190	9.2	+	
27	183	8.5		+
28	133	6.8		+
29	188	8.7	+	
30	197	10.0	+	
Total			15	15

Out of 30 non-diabetics, none of the patients showed high HbA1C levels and 3 of them had SSI.

Table 7: HbA1C and presence of SSI in diabetics

HbA1C Level %	SSI present	SSI absent	Total
HbA1C ≤7	1	7	8
HbA1C >7	14	8	22
Total	15	15	30

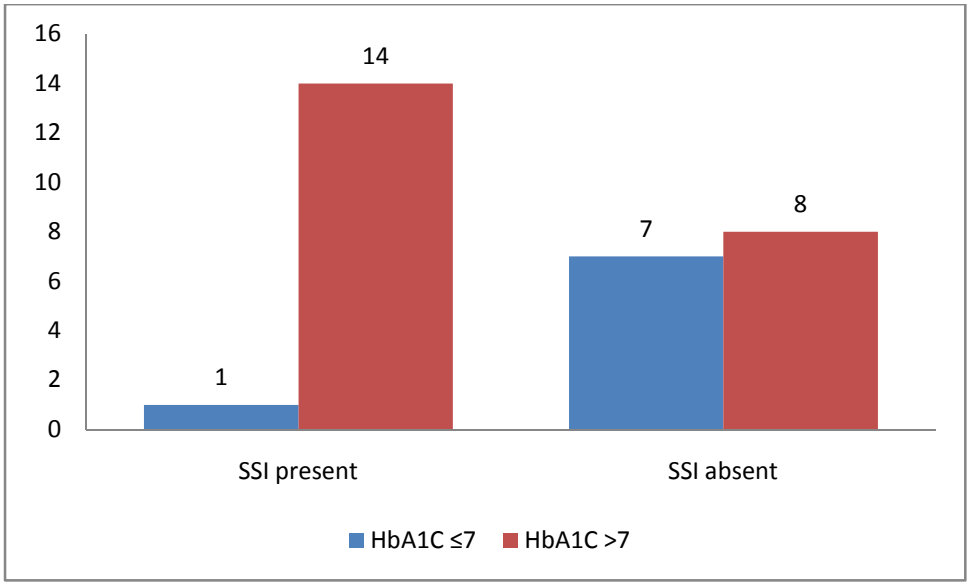


Figure 6 HbA1C and presence of SSI

Table 8: Range of HbA1C Value and SSI in Diabetics

HbA1C in Diabetic patients (%)	SSI
6.0-7.0	1
7.1-9.0	5
9.1-11.0	9
TOTAL	15

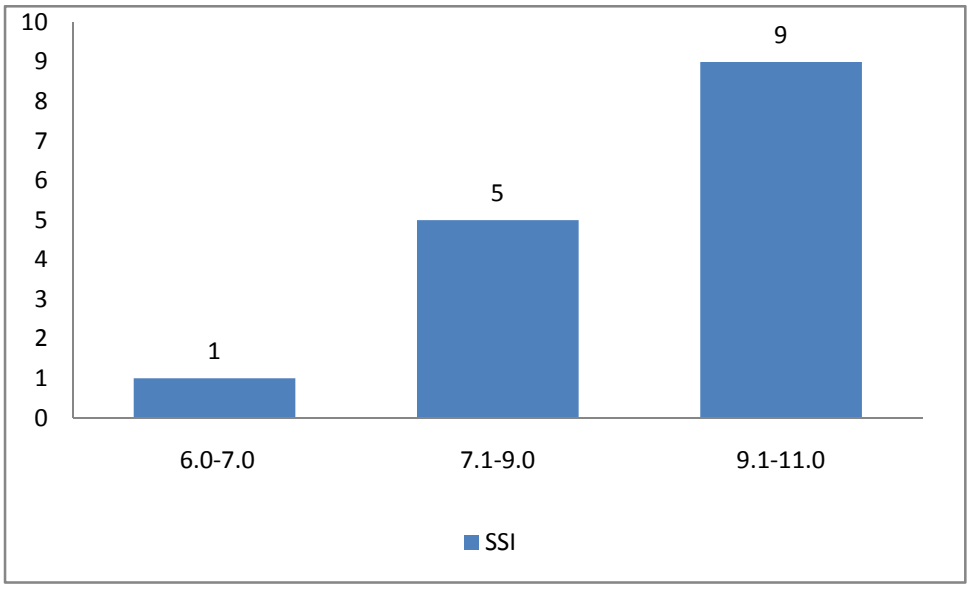


Figure 7 Range of HbA1C Value and SSI

Out of 30 diabetics, 22 showed high HbA1C levels and 14 of the patients with high HbA1C levels had SSI. Eight diabetics had normal HbA1C levels, out of which 1 had SSI. K-W test was done. Chi square value was 6.136 and p value was 0.0132. There was a correlation between HbA1C levels of patient and rate of SSI. The p value was 0.0132 which was statistically significant.

Out of 30 non-diabetics, none of the patients showed high HbA1C levels and 3 (10%) of the patients had SSI.

In addition the percentage of diabetic patients with normal HbA1C having SSI is 12.2% which is closely similar to percentage of SSI in normal HbA1C non-diabetics i.e 10%. Also, the risk of SSI in diabetic patients with high HbA1C (>7%) is 63%.

Hence the study shows that poor long term glycemic control depicted as high HbA1C levels were associated with high rates of SSI. It also shows that the diabetics who had good glycemic control i.e. HbA1C levels less than 7% had similar risk of SSI as the non-diabetics.

DISCUSSION

Millions of surgical procedures are performed each year worldwide,[1,2] with reports recognizing nosocomial infections in hospitals. Based on CDC's National Nosocomial Infections Surveillance (NNIS) system reports, SSIs are the third most commonly reported nosocomial infection, accounting for 14% to 16% of all nosocomial infections amongst hospitalized patients. [1,3] Among surgical patients, SSIs were the most common nosocomial infection, accounting for 38% of all such infections.[1] Of these SSIs, two thirds were limited to the incision.[1]

In 1980, Cruse estimated that an SSI increased a patient's hospital stay by approximately 10 days. [4,5] Other studies support that increased length of hospital stay and cost are related with SSIs. [6,7]

There are several patient and surgery characteristics that may affect the risk of surgical site infection. Out of the patient characteristics, elevated serum glucose level associated with diabetes mellitus is a very well recognized risk factor among the risk factors for surgical site infections.[15-22] It alters host immune responses, causing a well-documented rise in the predisposition to infectious processes.

Individuals with DM have a greater frequency and severity of postoperative wound infection. Hyperglycemia aids the colonization and growth of a variety of organisms. However, gram-negative organisms, S. aureus, and Mycobacterium tuberculosis are more frequent pathogens. Escherichia coli, several yeast species (Candida and Torulopsis glabrata) are commonly observed. Poor glycemic control is a common denominator in individuals with these infections. The cumulative effect of age-related immune senescence, can lead to serious and life-threatening infections in elderly patients with DM.

Our data show that good preoperative glycemic control, as measured by HbA1c levels less than 7%, is associated with a significantly lower risk of postoperative infections when adjusted for other factors that are known to influence this outcome. Not only that we also observed that diabetics patients with controlled HbA1c have close realtion to risk of SSI as non diabetics as 12.5% to 10% in latter.

To date, only handful of articles have explored the question of whether long-term control of blood glucose affects the occurrence of postoperative infections. Each of these has been limited to the study of surgical site infections (SSIs; wound infections) in a group of patients undergoing a specific type of operation. One such eg. Is of Bishop et al[68] who after confirming these findings in penile prosthetic patients recommended denying operations on the basis of poor glycaemic control. This finding was widely adopted and, according to this research group, was "used as the 'standard of care' in several legal cases."

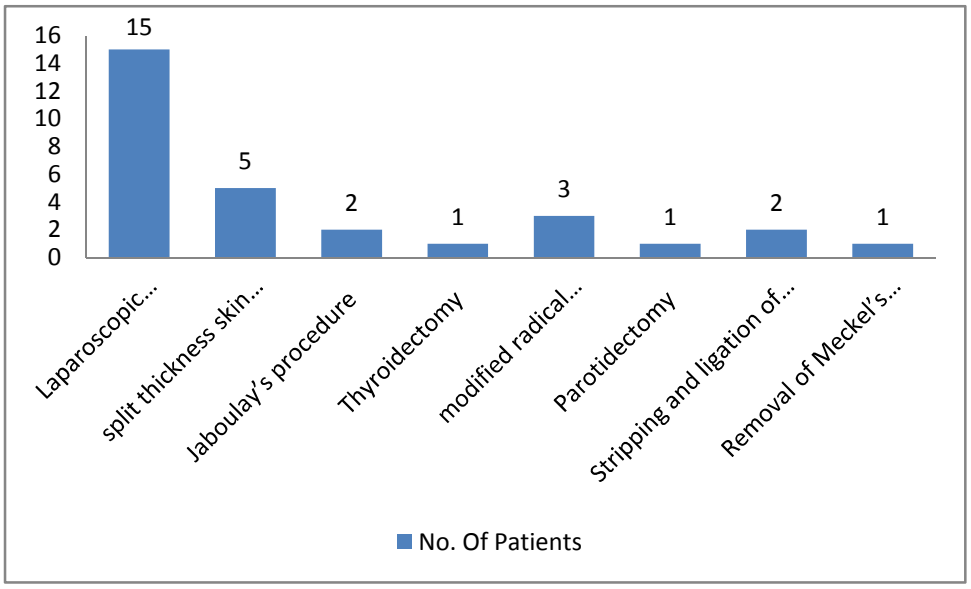
The benefit of normoglycemia seems to be independent of HbA1c values or pre-existent diabetes mellitus.[69]

In our analysis, we used the American Diabetes Association target of HgA1c levels of less than 7% to define good control. We took total 60 cases, 30 diabetics and 30 non-diabetics. The patients were predominantly male with a mean age of 71 year. Out of 60 patients, 24 were females and 36 were males. The age of females ranged from 40-68 years, the mean age being 52.54 years. The age of males ranged from 40-75 years, the mean age being 54.9 years. Combining the males and females, the age ranged from 40-75 years with the average age being 54.9 years. Thirty diabetics and thirty non-diabetics were studied. Among diabetics, 19 were males and 11 were females. Among non-diabetics, 17 were males and 13 were females.

Tabular Representation of Diferent types of surgeries done on diabetic patients

Sl. No	Types of surgery in diabetics	No. Of Patients
1	Laparoscopic Procedures (cholecystectomy and appendicectomy)	15
2	split thickness skin grafting	5
3	Jaboulay's procedure	2
4	Thyroidectomy	1
5	Modified radical mastectomy	3
6	Parotidectomy	1
7	Stripping and ligation of varicose veins	2
8	Removal of Meckel's diverticulum (Open)	1
Total		30

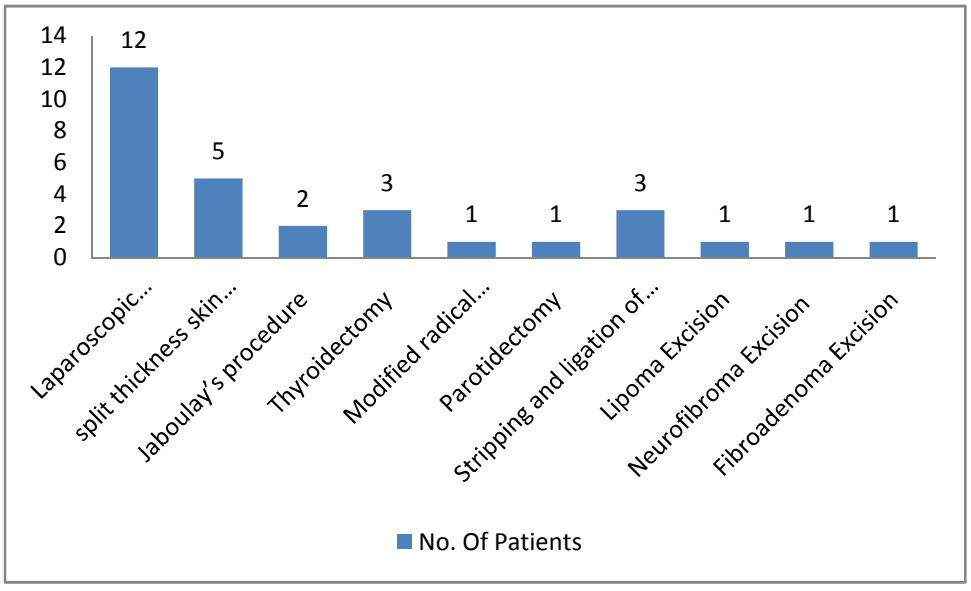
Graph Representing the Same



Tabular Representation of Diferent types of surgeries done on non-diabetic patients

Sl. No	Types of surgery in non-diabetics	No. Of Patients
1	Laparoscopic Procedures (cholecystectomy and appendicectomy)	12
2	split thickness skin grafting	5
3	Jaboulay's procedure	2
4	Thyroidectomy	3
5	Modified radical mastectomy	1
6	Parotidectomy	1
7	Stripping and ligation of varicose veins	3
8	Lipoma Excision	1
9	Neurofibroma Excision	1
10	Fibroadenoma Excision	1
Total		30

Graph Representing the Same



The cases of laparoscopic cholecystectomy, cases of laproscopic appendicectomy, cases of split thickness skin grafting, cases of surgeries done for treatment of varicose veins, cases of Jaboulay's procedure, cases of thyroidectomy, cases of modified radical mastectomy, cases of parotidectomy, case of lipoma excision, case of excision of Meckel's diverticulum, case of excision of neurofibromaand case of fibroadenoma excision were taken.

Out of 30 diabetics, 22 showed high HbA1C levels and 14 of the patients with high HbA1C levels had SSI. Eight diabetics had normal HbA1C levels, out of which 1 had SSI. K-W test was done. Chi square value was 6.136 and p value was 0.0132.There was a correlation between HbA1C levels of patient and rate of SSI. The p value was 0.0132 which was statistically significant. Out of 30 non-diabetics, none of the patients showed high HbA1C levels and 3 (10%) of the patients had SSI. Out of 8 diabetics with normal HbA1C levels and 1 had SSI.

There are a significant number of elective surgical patients who have previously undiagnosed pre-diabetes or a provisional diagnosis of diabetes. These patients are at considerable risk for unrecognized postoperative hyperglycemia and the associated adverse outcomes. Among patients with a history of diabetes, the majority have sub-optimal glycemic control. Relative to HbA1c, RBS testing has limited value in identifying patients with poor glycemic control in pre-surgical screening. These results suggest that HbA1c may be a more appropriate test for the preoperative assessment of diabetic patients.[70]

The question of why preoperative glycemic control is associated with postoperative infections can be considered from 2 perspectives. One is the likelihood of better postoperative glucose control in patients with good preoperative control.

Latham et al[71] demonstrated that hyperglycemia occurs significantly less often in patients with HbA1c levels less than 8% than in those with higher levels. Acute hyperglycemia has clearly been shown to be associated with poor outcome in a variety of clinical settings, including acute myocardial infarction,[72] stroke,[73] critical illness[74,75] and cardiac surgery.[76-78] Tight control of glucose in the postoperative period results in fewer complications, including infections, and decreased mortality in both diabetic and nondiabeticpatients.[79-84] In diabetic patients, the association between hyperglycemia and the susceptibility to infection is well established.[85] Neutrophil adherence , chemotaxis, phagocytosis, and intracellular bactericidal activity are all impaired.[86-88] The degree of neutrophil impairment correlates with the degree of hyperglycemia.[89]

The other possibility for decreased postoperative infection with long-term glucose control is the overall improvement in general health and metabolic milieu of the well controlled diabetic patient.

Latham and colleagues[71] did a study that pointed out, that each group of hyperglycemic patients (diabetic and nondiabetic) experienced an approximately twofold higher SSIrate than the comparable group that did not experiencehyperglycemia. Thirty percent of the excess infectionsattributable to hyperglycemia occurred in non-diabeticpatients. There would have been a reduction of 19infections out of 72, or 26%. That is a difference worth confirmingand achieving. [103]

Many such studies kept establishing and re-establishing the increased surgical site infection rates in diabetics. These studies doneon a variety of patientsundergoing variety of surgeries also showed that not just diabetics but non-diabetics also carry

increased incidence of SSI if the perioperative serum glucose levels are high. This fact raises the query if long term glucose control has any significance or its only the perioperative glucose levels that increases the risk of SSI.

The only study worth mentioning here is that of Annika et al[90] to assess if good preoperative glycemic control i.e. hemoglobin A1c [HbA1c] levels less than 7 % is associated with decreased postoperative infections. It was a retrospective study and 490 diabetic patients who underwent major non-cardiac surgery were analyzed.The details of age and sex were not mentioned in the study.A wide spectrum of general surgeries were included in the study. For analysis, they used the American Diabetes Association target of HbA1c levels of less than 7% to define good control. We followed the same criteria. The results show that an HbA1c level of less than 7% was significantly associated with decreased infectious complications with an adjusted odds ratio of 2.13 and a *P* value of .007. They concluded that good preoperative glycemic control is associated with a decrease in infectious complications across a variety of surgical procedures. The drawback of this study was that it is a retrospective studyand the details for age and sex were not mentioned.

In addition the percentage of diabetic patients with normal HbA1C having SSI is 12.2% which is closely similar to percentage of SSI in normal HbA1C non-diabetics i.e 10%. Also, the risk of SSI in diabetic patients with high HbA1C (>7%) is 63%.

Hence the study shows that poor long term glycemic control depicted as high HbA1C levels were associated with high rates of SSI. It also shows that the diabetics who had good glycemic control i.e. HbA1C levels less than 7% had similar risk of SSI as the non-diabetics.

SUMMARY

Surgical site infections (SSI) are the infections present in any location along a surgical tract after a surgical procedure. They are the third most common hospital-acquired infection. Diabetic patients have shown impaired wound healing and thus have an increased risk of morbidity after surgery from SSI.Tight glucose control in diabetic patients is now widely accepted as the goal in long term management because it demonstrates improvement in microvascular complications.

However, the impact of HbA1c on surgical outcome is still being defined and studies have been done proving increasing HbA1C levels correlated with increasing complications and reduced long term survival in CABG patients. But, there were no previously reported analysis of surgical outcomes of patients based on preoperative HbA1c levels presenting for general surgery.Hence, this study was done to see HbA1c as a predictor of superficial surgical site infection and to know the correlation between them.

In this study we studied all participants diagnosed with type 2 diabetes and equal number of non-diabetics undergoing elective surgical procedures from January 2012 to December 2012. Tests were carried out for participants undergoing elective surgery under General Surgery care and admitted to KLES Dr. Prabhakar Kore Hospital, Belgaum. HbA1c levels of diabetic patients were taken within 30days of surgery and compared with HbA1c levels of those of non-diabetics. Surgical site infection was assessed post operatively at 3^{rd}, 5^{th} , 7^{th}, 15^{th} 30^{th} days.

In present study, high levels of HbA1c were correlated with rate of SSI and p value was <0.005. Plasma hemoglobin A1c is an indicator of measuring glycaemia

over 2-3 months. The present study shows that increase in these levels is associated with significantly higher rate of SSI and medical morbidity. A strict control on glycemia must be performed preoperatively and postoperatively to reduce such complications and to obtain better outcome in diabetic patients.

In addition the percentage of diabetic patients with normal HbA1C having SSI is 12.2% which is closely similar to percentage of SSI in normal HbA1C non-diabetics i.e 10%. Also, the risk of SSI in diabetic patients with high HbA1C (>7%) is 63%.

Hence the study shows that poor long term glycemic control depicted as high HbA1C levels were associated with high rates of SSI. It also shows that the diabetics who had good glycemic control i.e. HbA1C levels less than 7% had similar risk of SSI as the non-diabetics.

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90. Annika S. Dronge, MD; Melissa F. Perkal, MD; Sue Kancir, RN; John Concato, MD, MPH; Michaela Aslan, PhD; Ronnie A. Rosenthal, MS, MD Long-term Glycemic Control and Postoperative Infectious Complications

INFORMED CONSENT

Title Of Research Study: A comparative study of long term glucose control and risk of superficial surgical site infection in diabetics v/s non diabetics undergoing general surgery

Principal Investigator:-

_____

Post Graduate Student,
Department Of General surgery
J.N.Medical College, Belgaum.

Surgical patients with diabetes are at increased risk of postoperative complications, including infections. The relationship between HbA1c levels and superficial surgical site infections is unclear. Hence the purpose is to derive a conclusion amongst them

The procedure is to subject all participants who voluntary want to get recruited of type 2 diabetes and equal number of non-diabetics undergoing general surgery, to compare and analyze the HbA1c levels amongst them in relation to surgical site infections and to derive a conclusion representing the same in context to specificity, sensitivity, positive predictive value and negative predictive value.

The benefits include to predict superficial surgical site infections, decreased hospital stay , and decreased morbidity amongst diabetics.

There are no risks involved.

I Mr./Ms._____ have been explained about the research study, the need of the study, the diagnostic intervention, their risks, benefits and alternatives available in my own vernacular language.

Taking part in this study is voluntary. I may choose not to take part in the study, or withdraw from the study anytime later. My decisions will not change the present or future health care or any service I receive. The study doctor or sponsor may stop my participation in the study without any consent. While taking part in the study I will be told of any important new findings that may change my willingness to continue or take part. If I choose not to take part in the study I will receive the standard treatment for patients with my conditions.

In the event that I become injured as a result of taking part in this study, treatment will be offered to me or I will be given information about where to receive medical care: but my insurance company or I will be responsible for the costs. No reimbursement, compensation or free medical care will be given.

All information collected about me during the course of the study will be kept confidential to the extent, permitted by law. I will be identified in research records by a code number. Information of the study may be published but my identity will be kept confidential in any publication.

You are voluntarily agree to participate in this study by signing up this form below. You may withdraw at any time from this study. You are not giving any of your legal rights by signing up this form. Your signature / thumb impression below indicates that you have read or information in the consent

been read to you including the risks and benefits and have cleared your doubts. You will be given a copy of this consent form.

In case of any queries, you can contact the following:

_____

Post Graduate Student)
Department of Surgery
Jawaharlal Nehru Medical College
Nehru Nagar, KLE Hospital Road
Belgaum 590 010

_____

(Professor)
Department of Surgery
Jawaharlal Nehru Medical College
Nehru Nagar, KLE Hospital Road
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_____

Chairman
College Ethical Dissertation & Research Committee
Jawaharlal Nehru Medical College
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Belgaum 590 010
Mobile no:+91 9880219404

Signature of the study participant:

Name of the study participant :

Signature of the witness:

Relationship to the patient:

Signature of the investigator:

Date:

PROFORMA / QUESTIONNAIRE TO BE USED FOR DATA COLLECION

The proposed proforma / questionnaire to be used for data collection for the study tilted

"A comparative study of long term glucose control and risk of superficial surgical site infection in diabetics vs nondiabetics undergoing general surgery" is as:

	Name:	Age: ☐☐ yrs. Sex: ☐ M ☐ F
	Address:	
	Educational Status:	Occupation:
	IP No.	Date of Admission: ☐☐ / ☐☐ / ☐☐☐☐
	Ward:	Date of Discharge: ☐☐ / ☐☐ / ☐☐☐☐

Chief Complaints:

Diabetes mellitus ☐ present ☐ not present
If yes then
Time of treatment ☐☐ days/weeks/months/years

Type of treatment ☐ oral hypoglycemics ☐ insulin

Past 4history:

Any history of hypertension ☐ Yes ☐ No

Any history of urinary complaints ☐ Yes ☐ No

Any history of visual disturbances ☐ Yes ☐ No

General examination:

	Built : ☐ Thin ☐ Moderate Obese Pulse : BP : Respiratory rate : Jaundice : Pallor / Clubbing / Cyanosis / Lymphadenopathy
Systemic Examination:	
	CNS: CVS: RS: P/A:
Clinical Diagnosis: **Proposed operation:**	

Investigations:

- Blood Glucose
- Serum Urea
- Blood Urea Nitrogen
- Serum Creatinine

S.electrolytes

- Serum Sodium
- Serum Potassium
- Serum Chloride
- Serum Bicarbonate

Liver Function Tests-

- Total Bilirubin
- Direct and indirect Bilirubin
- SGOT and SGPT
- Alkaline phosphate
- Total Proteins

	Serological Investigations HbA1c levels	□ . □ %

Grades		Appearances	Interval				
			Day 3	Day 5	Day 7	Day 15	Day 30
0		**Normal Healing**					
I		**Normal Healing with mild bruising or erythema**					
	A	Some bruising					
	B	Considerable bruising					
	C	Mild erythema					
II		**Erythema Plus Other signs of inflammation**					
	A	At one point					
	B	Around sutures					
	C	Along wound					
	D	Around wound					
III		**Clear or Heamoserous dis charge**					
	A	At one point only (<2cm)					
	B	Along wound (2cm)					
	C	Large Volume					
	D	Prolonged (>3 Days)					
IV		**Major Complications**					
	A	At one point (<2cm)					
	B	Along wound (2cm)					
V		**Deep severe wound infection with or without tissue breakdawn; Haematoma requiring aspiration**					

Culture and sensitivity report:

Positive Negative

If positive then:- Name of the organism ----------

Sensitive to ----------