"EVALUATION OF PREOPERATIVE SERUM ALBUMIN AND BODY MASS INDEX AS PREDICTORS OF POSTOPERATIVE MORBIDITY AND MORTALITY IN ELECTIVE MAJOR SURGERIES"

By

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IN

GENERAL SURGERY

Under the Guidance of

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ABSTRACT

INTRODUCTION:

Although a variety of nutritional indices have been found to be valuable in predicting patient outcome from surgery, serum albumin level is the most readily available and clinically useful parameter.

A decrease in serum albumin from concentrations greater than 46g/L to less than 21g/L is associated with an exponential increase in mortality rates from less than 1% to 29% and morbidity rates from 10% to 65%, hence serum albumin concentration is the Strongest predictor of mortality and morbidity for surgery as a whole.[1]

There is a substantial evidence to show that patients who have signs of malnutrition have a higher risk of complications and an increased risk of death in comparison with patients who have adequate nutritional reserves.[2]

Serum albumin is the most valuable test in the diagnosis of protein- calorie malnutrition. Most of the patients with depletion of protein intake will have low serum albumin levels. Patients with altered parameters will have increased risk of poor clinical outcomes.[3]

Nutritional assessment is necessary for identifying patients who are at risk of developing complications secondary to malnutrition.

A personal dietary history, physical examination (height and weight), and relevant investigations are the appropriate tools essential for an accurate evaluation of a patient's nutritional status preoperatively.

Serum albumin level is easily available and a helpful clinical parameter. A serum albumin level of 3.5 g% and above indicates adequate protein stores and it confers a protective effect through several biological mechanisms. It predicts the postoperative morbidity and mortality.[8]

The impact of BMI and tobacco smoking on outcome after open appendicectomy, concluded that tobacco smoking and a BMI of 27.5 or more were associated with more postoperative complications after open appendicectomy in patients with non-perforated appendicitis.[40]

Patients with low BMI are seen to be at higher risk for postoperative complications following cardiac surgery than normal or even severly obese patients. [44]

OBJECTIVES OF THE STUDY:

To determine the preoperative serum albumin levels and observe its association with postoperative morbidity and mortality.

To determine preoperative body mass index (BMI) and observe its association with postoperative morbidity and mortality.

MATERIALS & METHODS:

This study is a clinical, prospective, observational study conducted at R L JALAPPA HOSPITAL AND RESEARCH CENTRE ATTACHED TO SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR during the period from January 2014 to August 2015. The data regarding patient particulars, diagnosis, investigations, and surgical procedures are collected in a specially designed case recording form and patients followed for complications. The observations were analyzed using a statistical method, Chi square test and Fischer's Exact test.

Calculated sample size n = 100

RESULTS AND CONCLUSIONS:

Of the 100 patients studied, 59% were female and 41% were male. Of the 59 female patients 27 (45.8%) had complications and of the 41 male patients 19 (46.3%) had complications.

Of the 100 patients, the age varied from 19-79 yrs. The number of patients in the 41 – 60 years age group was the highest (59%). Among patients studied highest number of complications were noted in the age group of 41-60 years (56.6%) compared to other age groups.

When comparing the complication rate to serum albumin, it was found that among patients with serum albumin<2.5g/dl had 70% complication. Patients with serum albumin 2.6-3.0g/dl had 73% complication rate, between 3.1-3.5g/dl had 31% and >3.5g/dl had 21% complication rate.

The complication rate was found to be high with BMI<18.5kg/m^2 . The complication rate with BMI < 18.5 was 100%, BMI of 18.5-24.9 was 45%, and BMI >24.9 was 25% .

CONCLUSION: Patient with serum albumin level <3.0gm/dl and with abnormal BMI will have increased complication rate.

So serum albumin is a relatively cost effective test that should be used more frequently as a prognostic tool to detect malnutrition and risk of adverse surgical outcomes.

KEY WORDS :

Serum albumin, BMI, Morbidity and Mortality.

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INTRODUCTION

INTRODUCTION

Wound healing requires energy and is an anabolic process. Patients who are severely malnourished demonstrate impaired wound healing.

Although a variety of nutritional indices have been found to be valuable in predicting patient outcome from surgery, serum albumin level is the most readily available and clinically useful parameter.

A decrease in serum albumin from concentrations greater than 46g/L to less than 21g/L is associated with an exponential increase in mortality rates from less than 1% to 29% and morbidity rates from 10% to 65%, hence serum albumin concentration is the strongest predictor of mortality and morbidity for surgery as a whole.[1]

According to WHO, Body Mass Index(BMI), is defined as the weight in kilograms divided by the square of the height in meters(kg/m^2) and the normal value of BMI is 18.5 to 24.99 in adults. BMI is a simple index of weight-for-height commonly used to classify underweight(BMI <18.5), overweight (BMI 25 to 30)and obesity(BMI >30) in adults.

There is substantial evidence to show that patients who have signs of malnutrition have a higher risk of complications and an increased risk of death in comparison with patients who have adequate nutritional reserves.[2]

Protein energy malnutrition occurs due to relative and absolute deficiency of energy and protein intake. It may be primary, due to inadequate protein intake or secondary , as a result of other associated diseases. In developing countries, Primary

PEM is the most significant health related problem. PEM affects almost all organs of human body system. The most obvious observations are loss of body weight, loss of adipose fat storage and loss of skeletal muscle bulk of the patient. Synthesis of serum protein from hepatic system decreases and decreased levels of circulating proteins are observed. This leads to changes in body's immunological system, which results in poor wound healing.[3] Most patients with depletion of protein intake will have low serum albumin levels. Patients with altered parameter will have increased risk of poor clinical outcomes.[3]

The adverse effects of malnutrition on the morbidity and mortality of patients was first recognized by Hippocrates(460 BC- 370 BC) many centuries ago. Malnutrition occurs in about 30% of surgical patients with gastrointestinal disorders and in 60% of those in whom hospital stay has been prolonged because of postoperative complications. Nutritional assessment is necessary for identifying patients who are at risk of developing complications secondary to malnutrition.[6]

The impact of BMI and tobacco smoking on outcome after open appendicectomy, concluded that tobacco smoking and a BMI of 27.5 or more were associated with more postoperative complications after open appendicectomy in patients with non-perforated appendicitis

The serum albumin level is the easily available and helpful clinical parameter. A serum albumin level of 3.5 g% and above indicates adequate protein stores and it confers a protective effect through several biological mechanisms. It predicts the postoperative morbidity and mortality.[8]

AIMS

AND

OBJECTIVES

AIMS AND OBJECTIVES

To determine the preoperative serum albumin levels and observe its association with postoperative morbidity and mortality.

To determine preoperative body mass index (BMI) and observe its association with postoperative morbidity and mortality.

REVIEW

OF

LITERATURE

REVIEW OF LITERATURE

NUTRITION IN PATIENTS FOR SURGERY

The incidence of post operative complications, such as intra –abdominal collection(infection) and anastomotic site leakage and post operative ileus, can be reduced by the use of preoperative nutritional support. Preoperative nutritional support is not routinely indicated for all patients. In patients with only mild to moderate malnutrition preoperative nutritional support is not indicated. Protein depletion will result in prolonged wound healing.

Malnutrition has significant role in the operative mortality rate and three fold increase in the post operative complications like infections. Current indication for nutritional support before elective surgery are : history of weight loss in excess of 10% of body weight or an anticipated prolonged post operative recovery period during which the patient will not take adequate nutrition orally.[9]

Protein calorie malnutrition results in decreased lean muscle mass, alterations in respiratory system, altered body immune mechanism and intestinal atrophy. These changes result in delayed wound healing, predisposition to infective complications and increased postoperative morbidity.[9]

The aim of nutritional support in the surgical patient is to reduce or reverse the catabolic effects of disorder or injury. Although several important biological parameters have been used to measure the efficacy of nutritional regimens, the ultimate result for nutritional support in surgical patients should be improvement in

patient outcome and restoration of normal body functions.[10]

ASSESSMENT OF NUTRITION IN PATIENTS:

A comprehensive nutritional assessment includes the initial history of disease, physical examination and laboratory results to provide an overview of the patient's recent nutritional health.

1) History :

The possibility of malnutrition is suggested by the underlying disease status or by a history of recent significant weight loss. Anorexia, nausea, vomiting, dysphagia, gastroesophageal reflux disease or a history of generalized muscle weakness and fatigue should prompt further thorough evaluation. Recent weight loss (5% in the last one month or 10% in the last 6 months) or a current body weight of 80 to 85% (or less) of ideal bodyweight suggests significant malnutrition.

2) Physical Examination:

May identify wasting muscle bulk (thenar and temporal muscles), loose or flabby skin (due to loss of subcutaneous fat), and generalized edema and/or ascites (due to hypoproteinemia). Subtle findings of nutritional deficiency include skin rashes, pallor, glossitis, hepatomegaly, bald tongue, changes in hair gingivobuccal lesions, neuropathy and altered mental status like dementia.

3) Laboratory tests that indicate malnutrition correlate with postoperative morbidity and mortality

- Serum albumin of less than 3.5 g% in a stable, well hydrated patient indicates malnutrition. Serum albumin levels are normal in presence of malnutrition in severely dehydrated patients. Two thirds of the total stores of body albumin are located in the extravascular compartment with remaining one third in the circulation. Patients with sepsis , trauma and diseases of malignant state (like catabolic states) have lower plasma levels of albumin. Serum albumin has a half life of 14 - 20 days in human body.

- Serum prealbumin is the most useful indicator of acute change in nutritional status of patient. It has a half life of 2-3 days.

  10-17 mg% - mild depletion

  5-10 mg% - moderate depletion

  <5mg% - severe depletion

- Serum transferring levels of <200 mg% suggests malnutrition. It has a half life of 8 -10 days.[6]

4) Anthropometric studies:

Anthropometry is the method of assessing body size, weight and proportions. Measurements like MAC (mid arm circumference) and TSF (triceps skin fold thickness) reflect the body muscle mass (protein) and fat reserve respectively.

DEXA (Dual-energy X Ray absorptiometry) is also available in health centres and can be used to assess various body compartments (mineral, fat, lean muscle mass) Measurements that can be easily performed in the hospital or at the bedside of patient include measurement of height and weight, than calculation of BMI, which is the most useful reliable indicator. These values assess the patients visceral and somatic protein mass and fat reserve.[9]

5) Malnutrition is associated with defective immune function of body. It can be assessed by

Delayed type hypersensitivity

Total lymphocyte count (TLC) which is calculated by the formula

TLC = %lymphocyte x WBC /100

1500 – 1800 cells/mm^3 –mild depletion

900 – 1500 cells/mm^3 – moderate depletion

<900 cells/mm^3 severe depletion

NUTRITIONAL INDICES:

They provide a means of risk stratification and objective comparison among patients. They help doctors in determining the appropriate time for intervention and the progress being made towards the goal of adequate nourishment.[9]

BODY MASS INDEX: (Quetelet's Index)

It was invented between 1830 and 1850 by the Belgian Adolphe Quetelet during the course of developing " Social Physics".

It is a statistical measure of the weight of a person compared according to height. BMI is defined as an individual's body weight divided by the square of the height in metres.

A frequent use of the BMI is to assess an Individuals body weight compared to their height, that is normal or desirable for a person. The weight excess or deficiency may, in part, be accounted for by body fat or muscle

$$BMI = \text{weight(Kg)} / \text{height}^2\text{(m)} = 703 \times \text{weight (lb)} / \text{height}^2\text{(in)}$$

It is a better estimate of body fat than body weight and has more advantages over the ideal body weight estimation, BMI well correlates with body fat. BMI correlates with morbidity. It is useful in both men and women.[12]

Both low and high BMI correlate with morbidity and mortality. BMI of low levels are associated with lethargy, fatigue and diminished work duration. The lowest survivable levels of BMI, as suggested by observation in starvation, famine and anorexia nervosa, or by theoretical models, have been estimated to be 12-13kg/m^2. Recent studies have suggested that independently living older individuals with a BMI less than 22 kg/m^2 are at higher risk of mortality due to all causes.[7] It has been demonstrated that patients with low BMI and inadequate nutritional intake are associated with decreased functional status among older patients. A BMI of less than 18.5 indicates nutritional impairment and a BMI below 15 is associated with significant hospital mortality.[2]

There are few limitations of BMI, it may overestimate total body fat in persons who are very muscular (athletes) and may underestimate body fat in persons who have lost muscle mass(elderly). It also inaccurately measures body fat in edematous states or in those individuals of short stature (<5 feet tall).

		BMI (kg/m^2)
Underweight	Grade III	<16
	Grade II	16 – 16.99
	Grade I	17 - 18.49
Normal		18.5 -24.9
Overweight		25-29.9
Obesity	Grade I	30 – 34.9
	Grade II	35 – 39.9
	Grade III	>40

BMI is also used in children but in different ways. Its calculation is same as in adults, but then compared to typical values for other children of the same age. Instead of set thresholds for underweight and overweight in adults, percentiles are used in children. The BMI percentile allows comparison with children of the same age and sex. A BMI that is less than the 5^{th} percentile is considered underweight, between 85^{th} and 95^{th} percentile at risk of being overweight and above the 95^{th} percentile is considered overweight. [13]

PROGNOSTIC NUTRITIONAL INDEX (PNI):

It has been validated in patients undergoing either major cancer or gastrointestinal surgery and found to accurately identify a subset of patients at increased risk of complications. Furthermore, preoperative nutritional repletion has been shown to reduce post operative morbidity in this patient group.

PNI =158 – [16.6 x Alb] – [0.78 x TSF] – [0.2 x TFN] – [5.8 x DH]

Alb- Albumin

TSF – Triceps skin fold thickness (mm)

TFN – Transferrin (mg/dl)

DH – Delayed cutaneous hypersensitivity ;
induration > 5mm =2
1 -5 mm =1
Anergy =0

PNI risk of complication

Low <40%

Intermediate 40 -49%

High >50 %

NUTRITION RISK INDEX (NRI):

The index successfully stratifies perioperative morbidity and mortality using serum albumin and weight loss as prediction of malnutrition. Of note, the NRI is not a tool for tracking the adequacy of nutritional support, since supplemental nutrition often fails to improve serum albumin levels.

NRI = [15.19 x Alb] +41.7 x [actual wt 9kg)/ Ideal wt (kg)]

NRI = Well nourished>100

Mild malnutrition 97.5 -100

Moderate malnutrition 83.5 -97.5

Severe Malnutrition <83.5

Catabolic Index (CI)

CI = [24 hr urine urea nitrogen excretion in g] – [0.5 x (dietary nitrogen intake in g)]

No physiologic stress	0
Mild stress	0-5
Moderate to severe stress	>5

ESTIMATION OF CALORIC REQUIREMENTS:

It is necessary to provide adequate substrates for healing and tissue repair. Failure to provide adequate amounts of both calorie and protein leads to further depletion of lean body mass.[8]

Basal Energy Expenditure (BEE) is calculated using a modification of the Harris Benedict equation: BEE in kcal/day in,

Males: 16.4+ [13.7 x weight (kg)] + [5.0 x height(cm)] – [6.8 x age(yr)]

Females: 655 + [9.6 x weight(kg)]+[1.7 x height(cm)] – [4.7 x age(yr)]

The actual caloric requirement is obtained by multiplying BEE by a specific stress factor. Most stressed patients require 25 -30 kcal/kg/day.[8]

TEE in kcal/day (Total energy expenditure) is calculated by using the formula

TEE = BEE x stress factor

Selective stress factors	Stress factor
Starvation	0.8 -1.0
Elective surgery	1.0 -1.1
Peritonitis/other infection	1.05 -1.25
ARDS or sepsis	1.3 - 1.35
Cardiopulmonary disease With major surgery	1.3 – 1.55
Pancreatitis or major burns	1.3-1.8

Estimation of protein requirement:

The protein requirement varies with age and the clinical condition of the patient.

The appropriate ratio of caloric: nitrogen is 150:1 (for a caloric: protein ratio of 24:1)

Clinical condition	Protein requirement (g/kg(body wt)/day)
Normal person	0.8
Elective hospitalization	1.00-1.10
Complicated post operative case, infection	1.20-1.40
Major trauma, sepsis, pancreatitis	1.50-2.50

Protein needs of a person can also be assessed based on serum albumin levels.[12]

Albumin(g/dl)		protein requirement(g/kg/day)
Normal nutritional status	>3.5	0.8
Mild depletion	2.8 -3.5	1.0 – 1.2
Moderate deplete	2.1- 2.7	1.2 -1.5
Severe depletion	<2.1	1.5 – 2.0

METABOLISM OF PROTEINS, CARBOHYDRATES AND FATS:

The body requires an energy source to remain in steady state. About 50% of the basal metabolic rate (BMR) reflects the work of ion pumping, 30% represents protein turnover, and the remainder represents recycling of amino acids, glucose, lactate and pyruvate. Energy consumed in physical activity constitutes 10-50% of the total in normal subjects but decreases to 10-20% for hospitalized patients.

The increase in energy expenditure above basal is about 10% for elective operations, 10-30% for trauma and 50-80% for sepsis. Metabolic energy can be derived from carbohydrates, proteins and fats.[9]

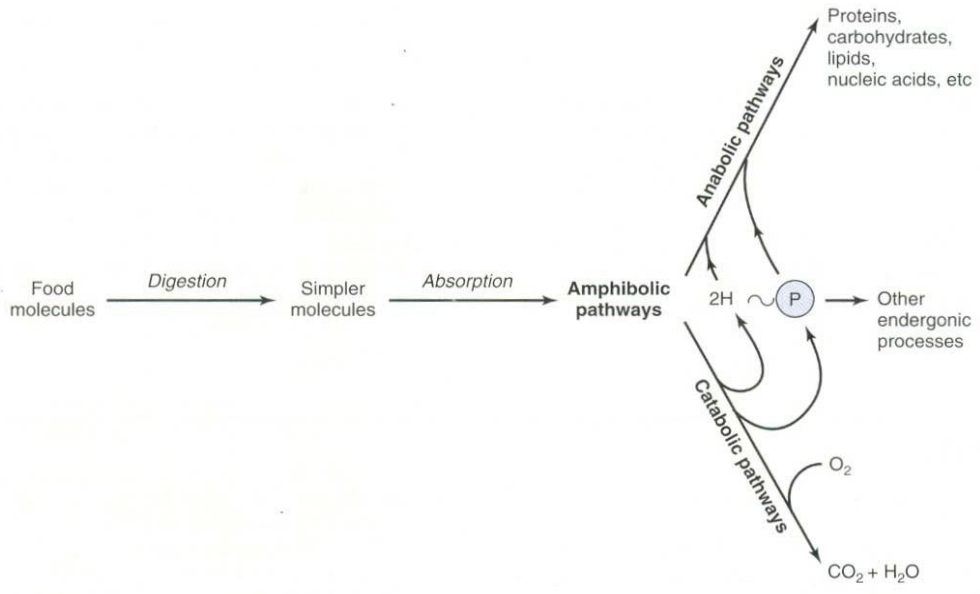


Fig.1 Metabolism of food molecules.

Carbohydrate metabolism:

Carbohydrates are the primary fuel source in the body under usual conditions, accounting for upto 30- 40% of total calorie intake. Each gram of enteric carbohydrate releases 4 kcal of energy after metabolism. Parenterally administered carbohydrate (example: IV dextrose) gives 3.4 kcal per gram.

Carbohydrate digestion is initiated by the enzymatic action of salivary amylase present in the oral cavity, and absorption normally occurs within the first 1.0 – 1.5 m of the small intestine i.e jejunum.

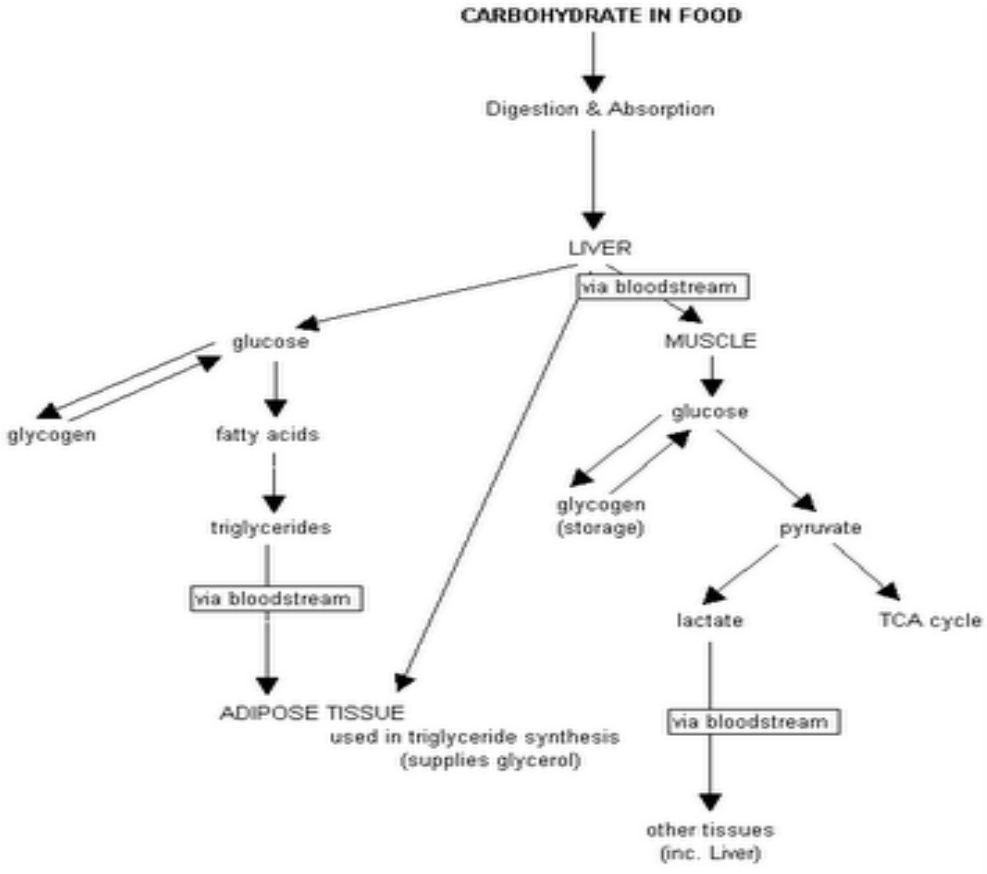


Fig.2:Overview of carbohydrate metabolism showing major pathways and endproducts.

Salivary and pancreatic amylases then divide starch into oligosaccharides after contact with food. Surface oligosaccharides then hydrolyze these further and help in transport of these molecules across the GI mucosa.

More than 75% of ingested carbohydrate is broken down into monosaccharides and absorbed in the form of glucose. Hyperglycemia stimulates insulin secretion from beta cells of pancreas, which influences protein synthesis. In a normal person minimum of 400 kcal of carbohydrate intake per day minimizes protein breakdown, particularly after adaptation to starvation. Glucose is essential for wound healing, but excessive carbohydrate intake or repletion with excessive amounts of glucose can cause hepatic steatosis and neutrophil dysfunction.

Excess glucose from refeeding, as reflected by RQ >1.0, can result in conditions such as glucosuria, thermogenesis and conversion to fat (lipogenesis). Excessive glucose administration results in elevated carbondioxide production, which is dangerous in patients with decreased pulmonary function, as well as hyperglycemia, which may contribute to increased risk of infection and suppression of immune function. [10]

Protein metabolism:

Proteins are composed of amino acids as subunits and its metabolism releases 4Kcal/gm of protein. Digestion of proteins produces dipeptides which is then broken down to single amino acids which are well absorbed by the gastrointestinal tract. The process of digestion of proteins is initiated by Gastric pepsin. Pancreatic proteases, activated by the enzyme enterokinase produced from intestine found throughout the duodenal mucosa, are the principal enzymes of protein digestion. After digestion, almost 50% of absorption of protein is achieved in duodenum and complete protein absorption occurs in the mid jejunum.

Total body protein in a 70 kg person is approximately 10-11 kg, concentrated predominately in skeletal muscle mass. Daily protein turnover in normal human being is about 250-300 gm, or approximately 3% of total body protein stores.

The daily protein requirement in healthy adults is about 0.8 to 1 gm/kg body weight. Protein synthesis or breakdown can be determined by measuring the nitrogen balance in the body. Protein intake of 6.25 g is equivalent to 1 g of nitrogen.

Nitrogen (balance) =Nitrogen (intake) – Nitrogen (output)

Nitrogen (intake) = gm protein (intake)/6.25

Nitrogen (output) = (UUNxVol) +3

Where, UUN is urine urea nitrogen, vol is volume of urine produced over time of measurement.

There are 20 amino acids divided into essential amino acids and non essential amino acids, depending on whether they can be synthesized de novo or provided from outside. Major role of amino acids are:

- Synthesis and recycling of proteins
- Catabolic reactions, resulting in energy generation and carbon-dioxide production
- Incorporation of nitrogen into non essential amino acids and nucleotides
- Transport and storage of small molecules and ions

Metabolism of absorbed amino acids, primarily by the liver, regulates accumulation of plasma amino acids. A baseline nitrogen loss of 10-15 g/day occurs through urinary excretion. Protein turnover decreases as the age increases from 25g/kg/day in the neonate to 3g/kg/day in the adult.

Glutamine: a non essential amino acid plays an important role in the metabolically stressed patient. It is a essential respiratory fuel for enterocytes of human body. The occurrence of decline of glutamine during injury or stress is more than that of any other amino acids and will persist during recovery period, after the concentration of other amino acids has normalized. It is very much essential for production of lymphocyte and function of macrophages.

Arginine: It is a essential substrate of urea cycle and nitric oxide production. It improves nitrogen balance and wound healing, stimulates response of T-cell to external factors and helps in reduction of infective complications. Arginine is also plays important role in albumin synthesis.

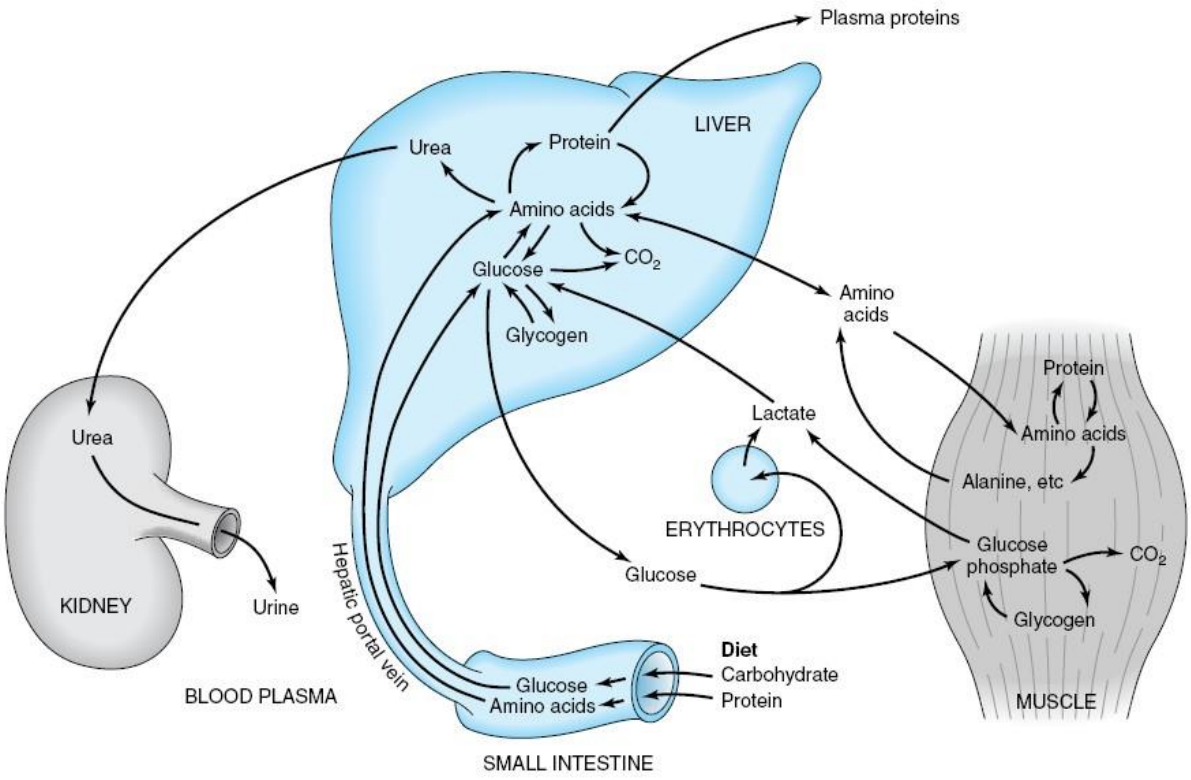


Fig.3:Overview of protein metabolism showing major pathways and endproducts

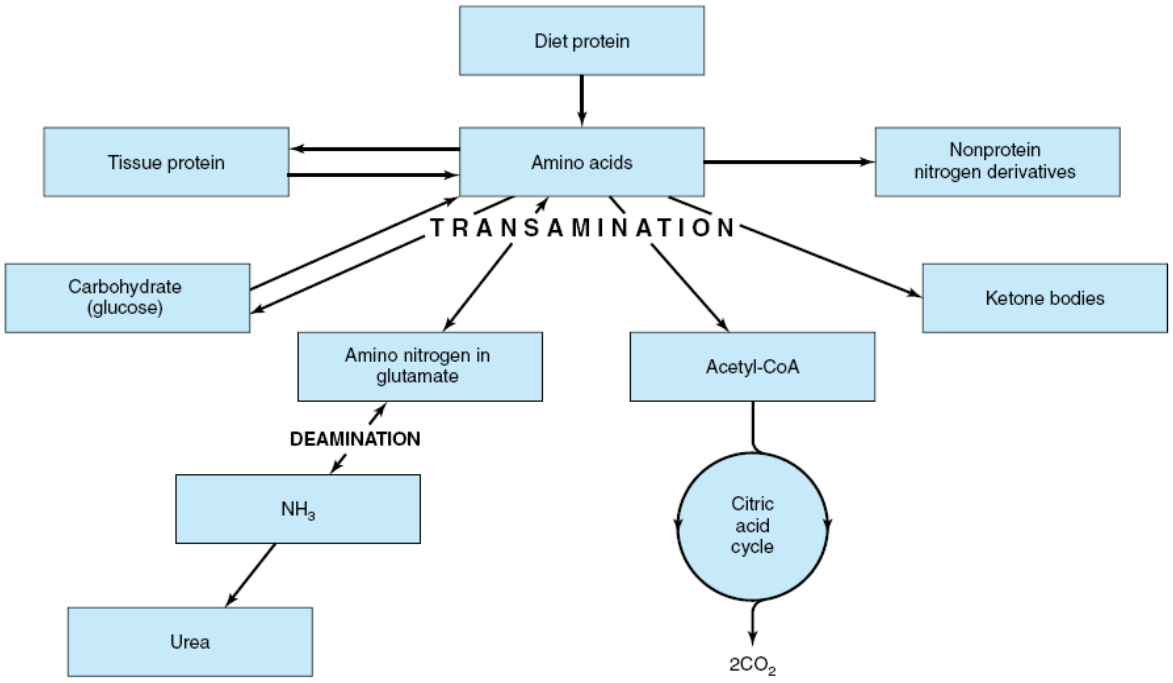


Fig.4:Overview of amino acid metabolism and major pathways.

Lipid metabolism:

Lipids comprise 25-45% of total calorie intake in the normal adult diet. Lipids release 9 kcal of energy for each gram. Digestion and absorption of lipids in humans is complex process and it requires the presence of biliary and pancreatic secretions as well as a functional jejunum and ileum.

The entry of fat into the duodenum stimulates the secretion of the enzymes cholecystokinin and secretin, which results in gall bladder contraction and release of pancreatic enzymes respectively. The environment of the duodenum is alkaline, which facilitates hydrolysis of triglycerides to diglycerides and monogycerides by lipase. Bile salts helps in emulsification of fats, than micelle formation is the most important step in absorption of lipids, facilitating absorption of fatty acids across the mucosal barrier. Reabsorption of bile salts is necessary to maintain the bile salt pool. The liver is able to compensate the moderate intestinal bile salt losses by increased synthesis from cholesterol. Ileal resection of major segment may lead to depletion of the bile salt pool and later fat malabsorption. Lipolysis is stimulated by stress steroids, catecholamine and glucagon but is inhibited by insulin.

The essential fatty acids like linoleic acid and linolenic acid are required for cell membrane integrity. Dietary fats are the only precursors to eicosanoid production. Clinical deficiency results in a generalized scaling rash, hepatic steatosis and bony changes.

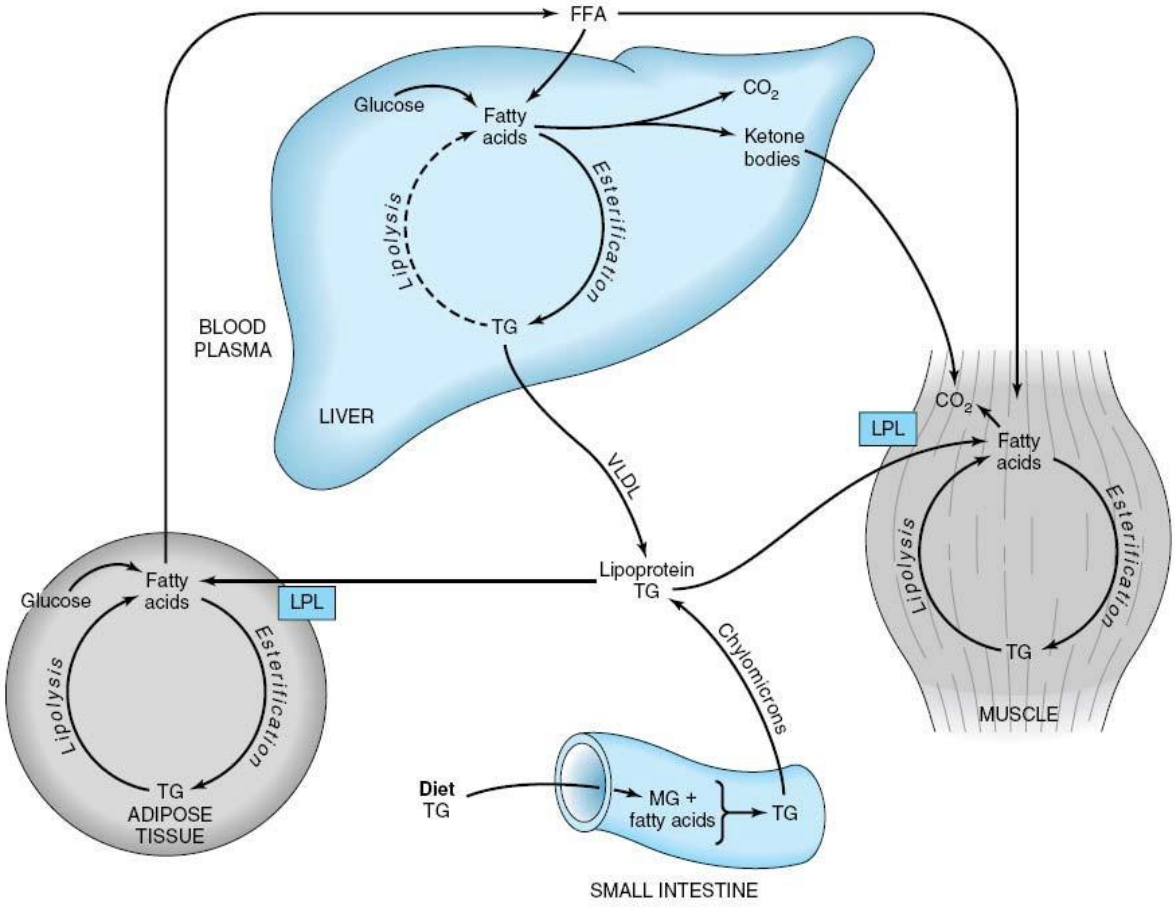


Fig.5:Overview of fat metabolism and major pathways.

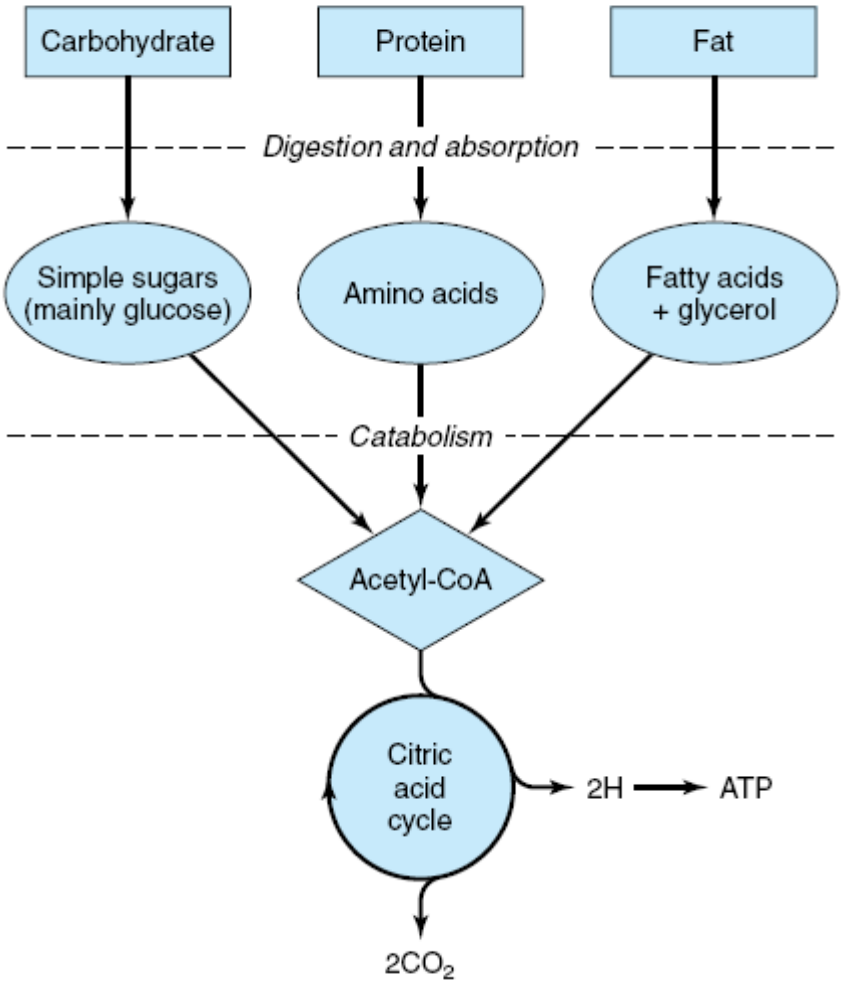


Fig.6:Outline of the pathways for the catabolism of dietary carbohydrate, protein, and fat. All the pathways lead to the production of acetyl-CoA, which is oxidized in the citric acid cycle, ultimately yielding ATP in the process of oxidative phosphorylation.

METABOLISM DURING STRESS :

Wound healing and recovery from critical illness all depend upon adequate nutrient intake.

Starvation: After an overnight fast, liver glycogen is rapidly depleted due to decreased plasma insulin and a rise in glucagon levels. Carbohydrate stores are exhausted after a 24 hr fast. Liver glycogen is used first, followed by muscle glycogen. Hepatic glucose metabolism must satisfy the energy demands of the hematopoietic and the central nervous system particularly the brain, which is dependant on glucose oxidation during acute starvation. Within approximately 10 days of starvation, the brain adapts to use fat as its fuel source. Thereafter brain depends mainly on ketone bodies produced by the liver and it has a protein sparing effect.

The adaptive changes to starvation are a decrease in basal energy expenditure (up to 30%), a change in the type of fuel consumed, and a relative preservation of protein.

Physiologic stress:

The interaction of metabolic and endocrine responses that result from major operation, trauma or sepsis can be divided into three phases

1) Catabolic phase: After major surgery or trauma, the metabolic demand is greatly increased and results in a significant rise in the excretion of urinary nitrogen. Following a major surgical procedure, protein depletion initially occurs because patients are commonly prevented from eating in addition to having an elevated basal metabolic rate. The hormonal response of

physiologic stress includes elevation in the serum levels of glucagon, glucocorticoids and catecholamines and reduction of insulin.

2)Early Anabolic phase: (cortical withdrawal phase) since it marks the shift from catabolism to anabolism, it ranges from several weeks to few months. This phase is marked by a positive nitrogen balance, and there is a rapid and progressive gain in weight and muscle strength. The total amount of nitrogen gained is equivalent to the amount lost in the catabolic phase.

3)Late Anabolic phase: It is the final period of recovery and may last from several weeks to months. Adipose stores are replenished gradually, and nitrogen balance equilibrates. Weight gain is much slower than in early anabolic phase due to higher caloric content of fat – the primary energy stores deposited during the early anabolic phase – as compared to protein. activation of hormone sensitive lipase by glucagon, epinephrine, cortisol and thyroid hormone accelerated catabolism, arise in proteolysis stimulated by cortisol and decreased peripheral glucose uptake due to insulin antagonism by GH and epinephrine.

Following elective operation, neural impulses carried via spinothalamic pathways activate the brain stem and thalamic and cortical centers which stimulate the hypothalamus. Hypothalamic stimulation triggers combined neural and endocrine discharges. Nor epinephrine is released from sympathetic nerve endings. Epinephrine from the adrenal medulla, Aldosterone from adrenal cortex, ADH from posterior pituitary, insulin and glucagon from the pancreas and ACTH, TSH and GH from the anterior pituitary. These hormones produce secondary elevations of cortisol, thyroid hormone and somatomedins. The effects of the heightened neuroendocrine secretion include a) Peripheral lipolysis from the synergistic activation of hormone sensitive lipase by

glucagon,epinephrine,cortisol and thyroid hormone.

b) Accelerated catabolism, arise in proteolysis stimulated by cortisol and

c) Decreased peripheral glucose uptake due to insulin antagonism by GH and epinephrine. The consequences are a marked rise in plasma concentration of free fatty acids, glycerol, glucose, lactate and amino acids.

The liver responds with an increase in substrate uptake and glucose production, as a result of glucagon stimulated glycogenolysis and enhanced gluconeogenesis induced by cortisol and glucagon. Operation and trauma are neuroendocrine driven processes. The BEE rises by 10% in post operative patients.

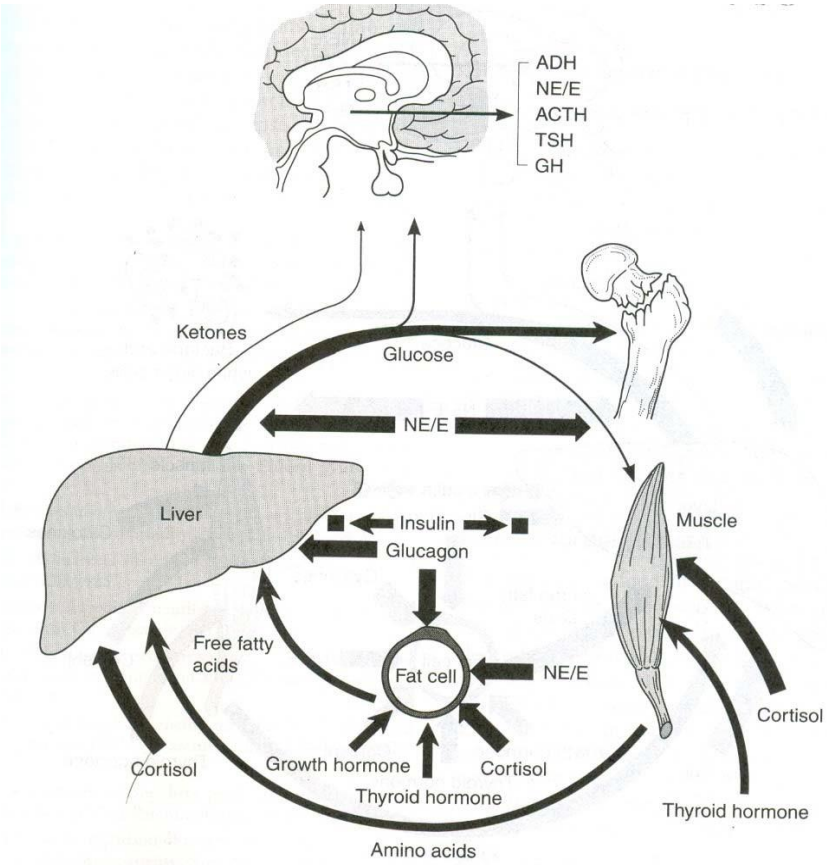


Fig.7:The metabolic response to trauma is a result of neuroendocrine stimulation, which accelerates protein breakdown, stimulates gluconeogenesis, and produce glucose intolerance.

WOUND HEALING:

Healing is a fibroproliferative process that helps in repair of tissue as a result insult to the body. It is a complex process but orderly phenomenon involving a number of steps.[23]

1. Induction of an inflammatory process in response to the initial injury with removal of damaged and dead tissue.
2. Proliferation and migration of parenchymal and connective tissue cells.
3. Formation of new blood vessels (angiogenesis) and granulation tissue.
4. Synthesis of ECM proteins and collagen deposition.
5. Tissue remodeling.
6. Wound contraction.
7. Acquisition of wound strength.

The earliest account of wound healing dates back to about 2000B.C.when Sumarius employed two modes of treatment: a spiritual method consisting of incantations and a physical method of applying poultice-like materials to the wound. The Egyptians were the first to differentiate between infected and diseased wounds compared to non- infectious wounds. The Greeks later equipped with this knowledge , went even further ahead Egyptians and classified wounds as acute or chronic wounds.[4]

Acute wound healing is the normal orderly process that occurs after a typical injury and it requires minimal intervention.

Chronic wound healing requires a variety of interventions to correct and keep the healing of wound in a normal phase of healing.

Acute wounds are usually traumatic or surgical in nature. These wounds occur suddenly,

moves rapidly and predictably through the repair process and result in durable closure. Chronic wounds are the ones that fail to proceed normally through the repair process.

Chronic wounds usually occur as a result of vascular compromise, chronic inflammation or repetitive damage to tissues and results in failure to close in a timely fashion or fail to result in durable closure. [15]

PHASES OF WOUND HEALING:

a. Inflammatory phase

b. Proliferative phase

c. Remodeling phase (Maturation phase)

The immediate response to injury is the inflammatory (also called reactive phase). The body's defenses are aimed at limiting the amount of damage and preventing further injury. The proliferative (also called regenerative or reparative phase) is the reparative process and consists of re-epithelialisation, matrix synthesis and neovascularisation to relieve the ischemia of the trauma itself . The final maturational (or remodeling) phase is the period of scar contraction with collagen cross linking, and loss of edema.

INFLAMATORY PHASE

After the immediate reaction of the tissue to injury, hemostasis and inflammation process occur. This phase plays a role in an attempt to limit damage by stopping the bleeding, sealing the surface of the wound and removing any necrotic tissue, foreign debris. During the phase of inflammation, vascular permeability is increased, migration of cells towards wound by occurs by chemotaxis, cytokines and growth factors secretion increased and increased activation of the migratory cells.

HEMOSTASIS

During an acute phase of injury blood vessels damage results in exposure of subendothelial collagen to platelets, which leads to platelet aggregation and activation of coagulation pathway. Initially there is local vasoconstriction of arterioles and capillaries which is followed by vasodilation and increased vascular permeability. Cessation of haemorrhage is aided by plugging of capillaries with erythrocytes and platelets which adhere to the damaged capillary endothelium.

Regeneration is the perfect restoration of the pre-existing tissue architecture in the absence of scar formation. Although regeneration is the goal of wound healing, it is found only in embryonic development or in certain tissue compartments such as bone and liver. However, in wound healing of adult humans, the accuracy of regeneration is sacrificed for inflammatory phase.

PROLIFERATIVE PHASE

As the acute responses of hemostasis and inflammation begin to resolve the scaffolding is laid for repair of wound through angiogenesis, fibroplasia and epithelialisation. This stage is characterized by formation of granulation tissue which consists of capillary bed, fibroblasts, macrophages and a loose arrangement of collagen, fibronectin and hyaluronic acid.

ANGIOGENESIS

It is a process of formation of new blood vessels and is essential to support wound healing environment after injury to tissue, endothelial cells are activated, that degrades the basement membrane of post capillary venules there by allowing the migration of cells. Division of these migrating endothelial cells results in tubule or lumen formation. Eventually deposition of the basement membrane occurs and results in capillary maturation.

FIBROPLASIA

Fibroblasts are specialized cells that differentiate from resting mesenchymal cells in connective tissue. After injury, the normal quiescent and sparse fibroblasts are attracted to the inflammatory site, where they divide and produce the components of extracellular matrix. The primary function of fibroblasts is to synthesize collagen. The time required for undifferentiated mesencymal cells to differentiate into highly specialised fibroblasts accounts for the delay between injury and the appearance of collagen in a healing wound. This period, generally 3 to 5 days, depending on the type of tissue injured is called the lag phase of wound healing.

The rate of collagen synthesis declines after 4 weeks and eventually balances rate of collagen destruction by collagenase. At this point the wound enters a phase of collagen maturation. The maturation phase continues for months or even years. Glycoprotein and mucopolysaccaride levels decrease during the maturation phase and new capillaries regress and disappear. These process changes the appearance of wound and increases its strength.

EPITHELIALISATION

Re-epithelialisation of wound begins within hours after injury and is completed within 2 days in case of approximated incised wounds. Initially wound is rapidly covered by formation of clot and later by epithelial cell migration towards the defect. Keratinocytes located at basal layer of the epidermis or in depths of dermal appendages epithelium migrate towards the wound. Epithelialisation involves a series of changes in the wound keratinocytes that is detachment, migration, proliferation, differentiation, and stratification. If the basement membrane zone is in close intact, wound epithelialisation proceeds very rapidly.

EXTRACELLULAR MATRIX

The ECM acts as a scaffold to stabilize the physical structure and shape of tissues. ECM cells produces the macromolecular constituents.

Following are the macromolecules:

- Glycosaminoglycans, are usually found covalently linked to protein in the form of proteoglycans.
- Fibrous proteins such as collagen, elastin, fibronectin and laminin.

The wound matrix accumulates and composition changes as healing progresses, there will be a balance between new deposition and degradation. The provisional matrix is scaffold for acellular migration and composed of fibrin, fibrinogen, fibrinonectin, and vitronectin. Glycosaminoglycans and proteoglycans are synthesized next and support further matrix deposition and remodeling.

WOUND CONTRACTION:

The wounds undergo some degree of contraction. The major cell responsible in this process is the myofibroblast, which appears from 6^{th} day of injury. The wound contraction starts almost immediately after injury. The movement of cells with concomitant reorganization of the cytoskeleton is responsible for contraction.

WOUND STRENGTH:

At the end of one week, the wound strength is approximately 10% that of a normal skin, which increases rapidly over the next one month. At the end of 3rd month it reaches a plateau at about 70% to 80% of the tensile strength of normal skin, this strength may persist for life. The recovery of tensile strength results from excess of collagen synthesis inspite of collagen degradation in first 2 months of healing process and at later by structural modification of collagen fibres (linking, altered fibre size) once synthesis of collagen ceases.

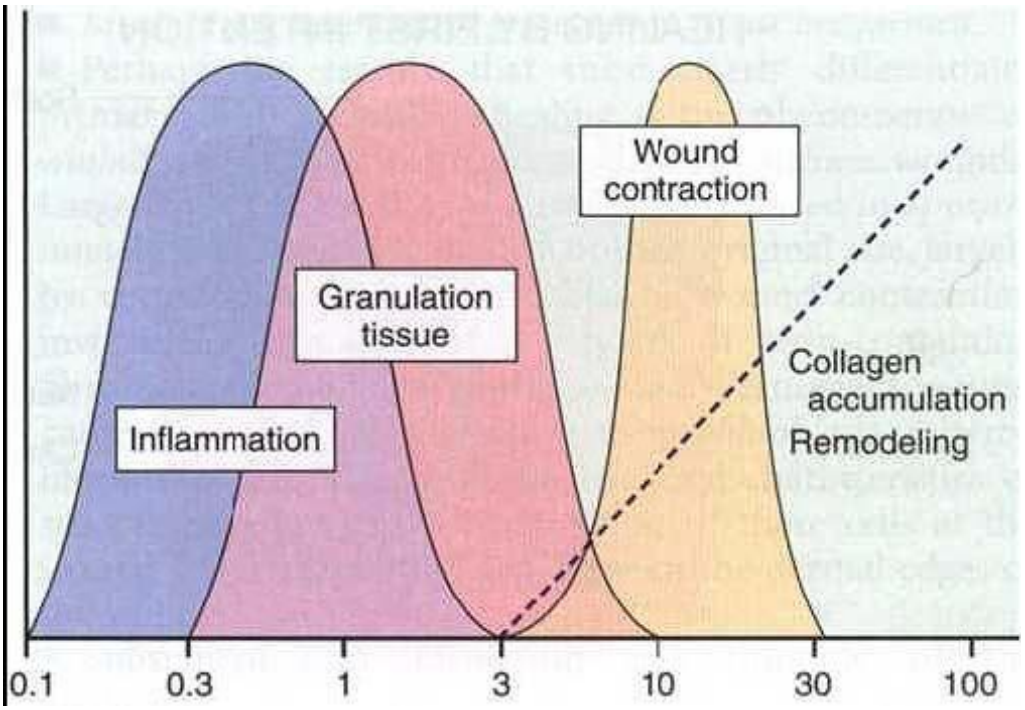
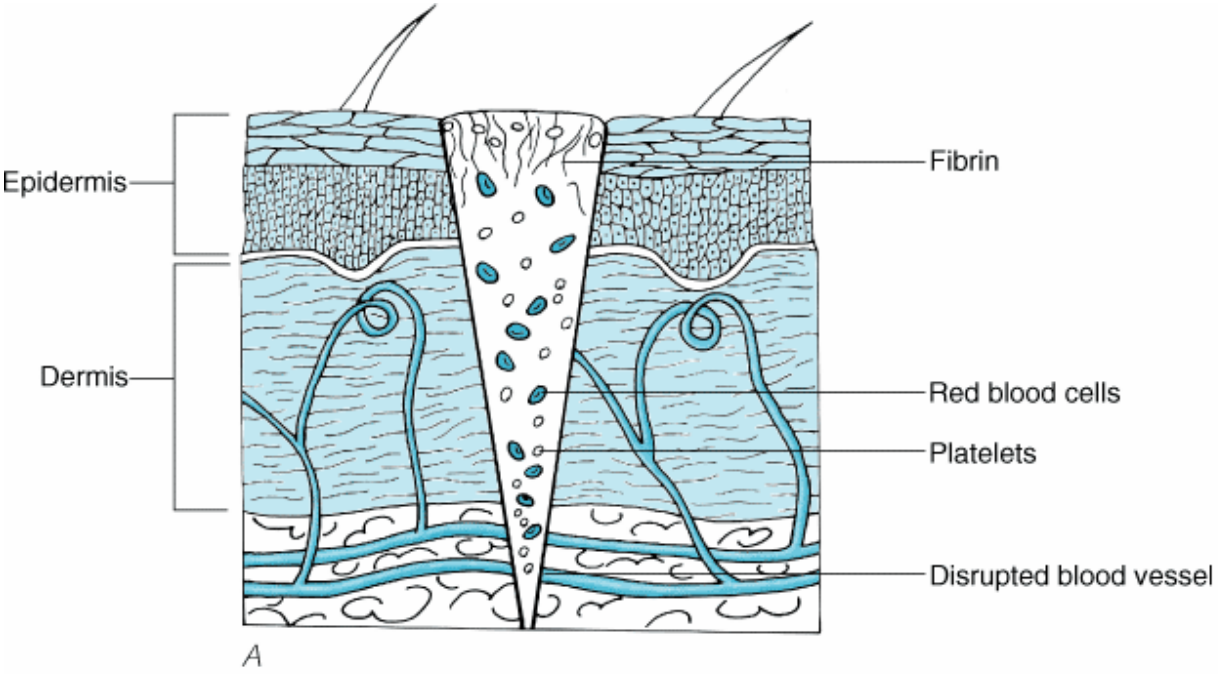
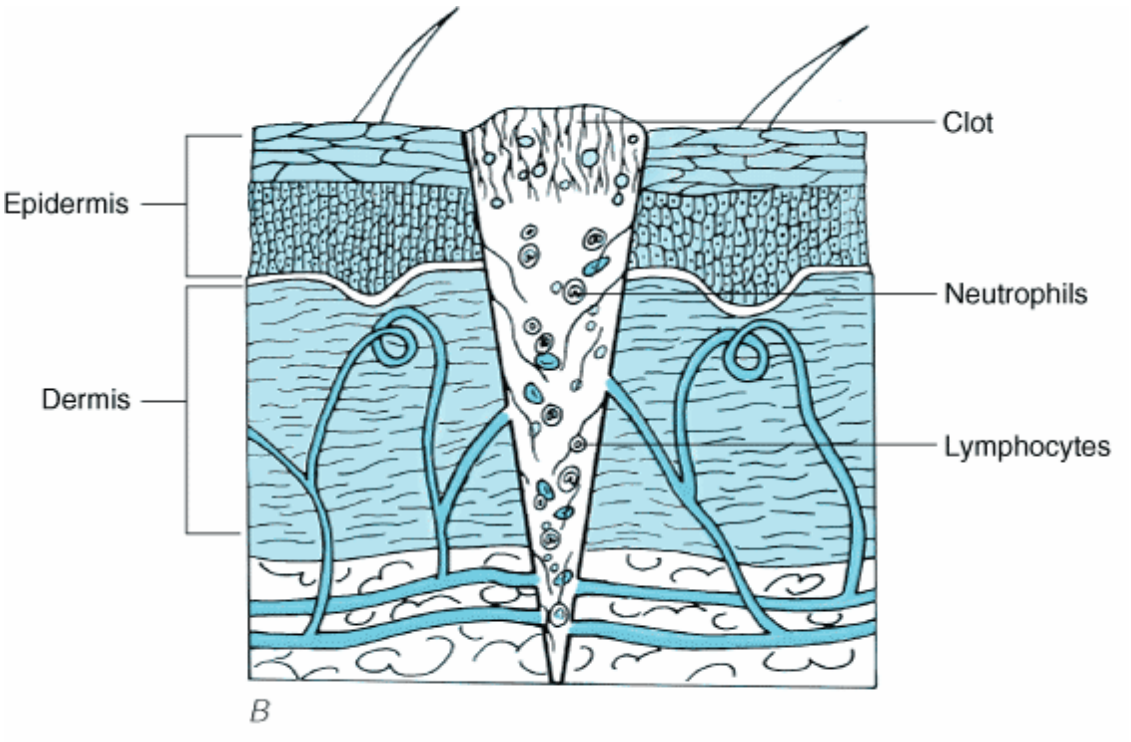


Fig.8:Phases of wound healing.


Epidermis
Dermis
Fibrin
Red blood cells
Platelets
Disrupted blood vessel
A


Epidermis
Dermis
Clot
Neutrophils
Lymphocytes
B

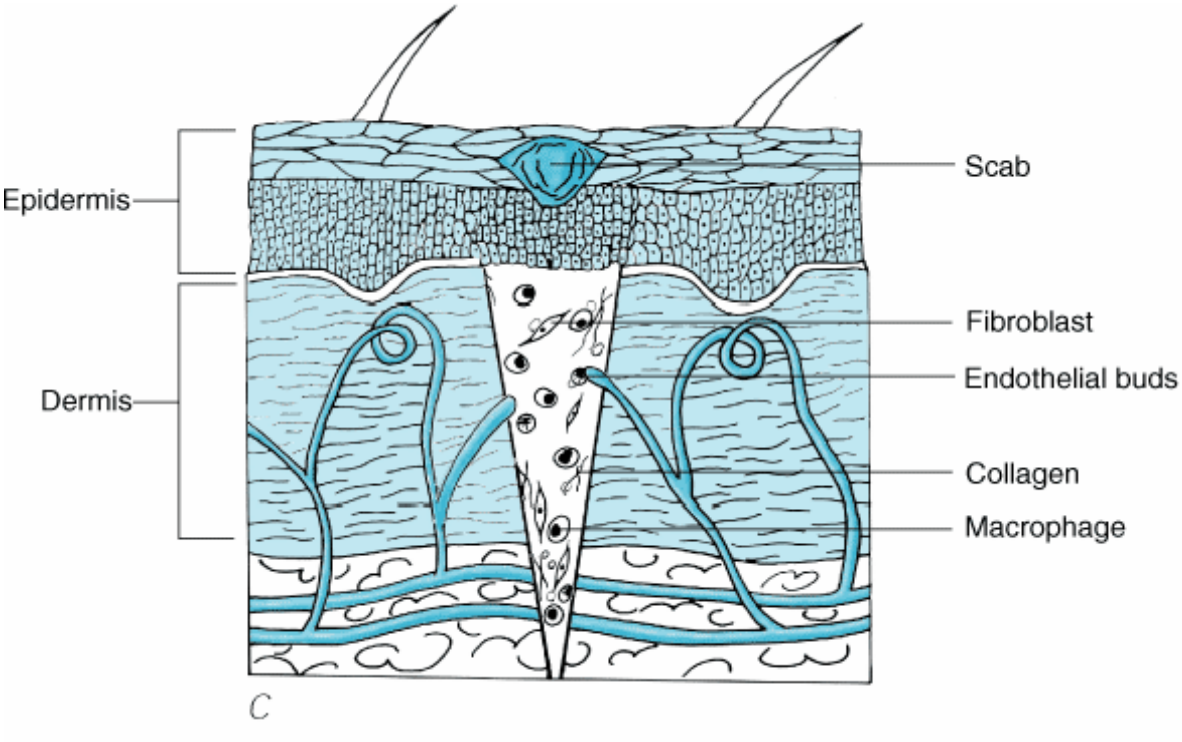


Fig.9: The phases of wound healing viewed histologically

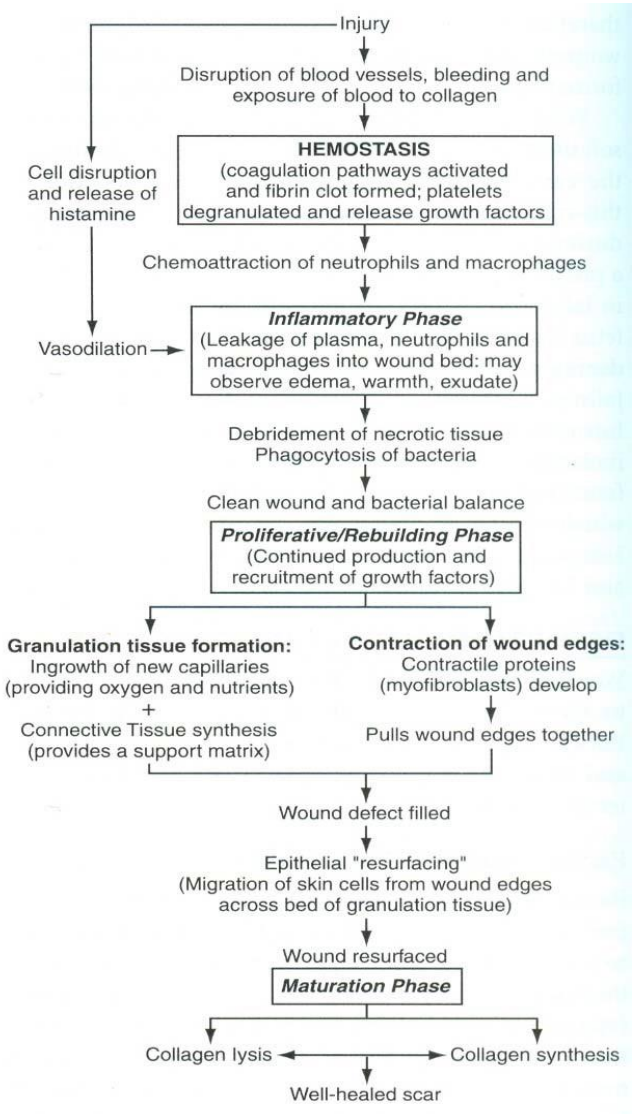


Fig.10:Cascade of events in wound repair process:

BASED ON TIMING OF WOUND HEALING THEY ARE CLASSIFIED INTO:

A. Primary Healing (First intention)

B. Secondary Healing (Second intention)

C. Healing by tertiary intention (Delayed primary closure)

HEALING BY PRIMARY INTENTION:

It occurs in a clean incised wound or surgical wound. The wound is closed by direct approximation by sutures or by placement of a graft or flap. There is more epithelial regeneration than fibrosis. Wound heals rapidly with complete closure and scar will be linear and supple. Wounds that are less than 6 hrs old are considered and are less likely to develop into chronic wounds.

HEALING BY SECONDARY INTENTION:

It occurs in a wound with extensive soft tissue loss like major trauma. It occurs when a wound is left open and heals slowly by fibrosis. It is commonly used in the management of wounds that are treated beyond the initial 6 hrs. It leads to wide scar and contraction, later may lead to disability.

HEALING BY TERTIARY INTENTION:

It is used for wounds that are heavily contaminated, after control of infection wound closed with sutures or covered using skin graft. The wound is vascularised after 4-5 days of open observation so that the cutaneous edges can be approximated at that time.

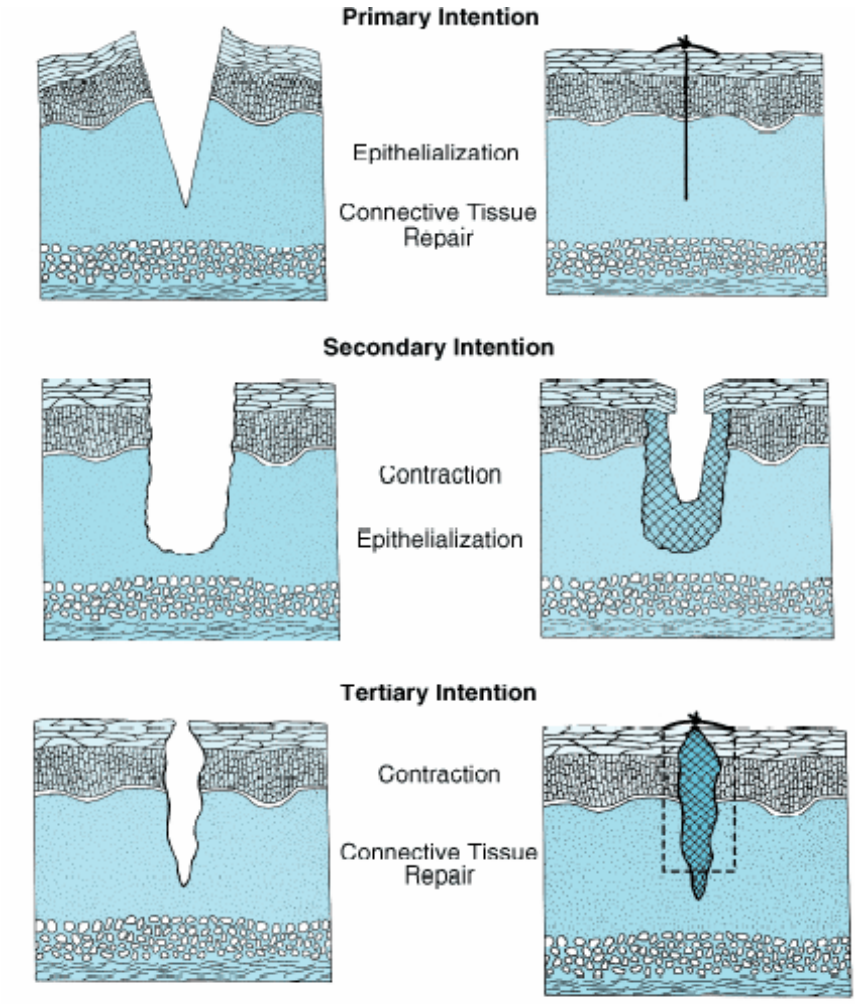


Fig.11:Classification of wound healing.

ABNORMAL WOUND HEALING:

Wound healing is a complex process, series of events occurs during healing, number of factors can impede the outcome of healing. The amount of tissue damaged or lost, the presence of foreign substance or bacterial infection and the duration of exposure to external factors affect the time of recovery. The greater the insult, the longer the healing process and the greater the extent of scar formation. Intrinsic factors such as chemotherapeutic drugs, atherosclerosis of vessels, renal failure, cardiac status and site of wound over the body all affect wound healing. The blood supply in lower extremity is the worst in the body; that on the face and hands is the best.[18]

FACTORS THAT INHIBIT WOUND HEALING:

- AGE - older patients have slow healing process than young.
- MALNUTRIOTION- It affects the normal healing through the indirect and the direct effects of vitamins and mineral deficiency. Proteins, calories, vitamin-C, vitamin-A, zinc, magnesium, and iron are all critical to collagen synthesis and development of normal tensile strength. Adequate nutrition is essential to good immune system of body and helps in infection control. Protein catabolism can result in a delay of wound healing.

NUTRIENT	ROLE IN HEALING
Protein	Fibroplasia
	Angiogenesis
	Collagen formation and wound remodeling
	Immune function
	Precursor nitric oxide
Carbohydrates	Energy supply
	Protein sparing
	By-product of lactate
	Angiogenesis
Fat	Formation and stability of cell walls and intracellular organelles
	Inflammation
Vitamin A*	Epithelialization
	Wound closure
	Inflammatory response
	Angiogenesis
	Collagen formation
B vitamins	Cofactor in enzyme systems
	Immune response
	Synthesis of protein, fat, and carbohydrate
Vitamin C	Collagen synthesis
	Capillary wall integrity
	Fibroblast function
	Immunologic function
	Antioxidant
Vitamin D*	Calcium metabolism for building and maintaining bone
Vitamin E*	Unknown in relation to wound healing
	Antioxidant
Vitamin K*	Coagulation
Copper	Cross-linking of collagen
	Erythropoiesis
Iron	Collagen formation
	Leukocyte function
	Oxygen transport
Magnesium	Protein synthesis
Zinc	Collagen formation
	Protein synthesis
	Cell membrane stability
	Host defenses

*Fat-soluble vitamins.

Fig.12:The role of nutrients in wound healing:

- INFECTION- wound infection prolongs the inflammatory phase, delays collagen synthesis, prevents epithelialization and increases the

production of inflammatory cytokines, which may lead to additional tissue destruction. Presence of more than 10^5 bacteria signifies infection. Critical factors are concentration, virulence, and host resistance.

- DIABETES MELLITUS- impaired wound healing in diabetes patients is well established. The wound repair in patients with DM is characterized by reduced collagen synthesis and deposition and decreased tensile strength. A direct relationship between tensile strength and glycosylated hemoglobin levels have been described. The differences in wound repair may be partially explained by increased levels of proteases, decreased levels of proliferative cytokines and abnormal insulin levels. DM can also result in compromised perfusion, patients with DM are at high risk of microvascular diseases. Consequently the delivery of micronutrients at the capillary level is impaired and there is an increase in vascular permeability. The end result seems to be increased risk of infection with diminished support for healing.
- OBESITY- It is a risk factor for impaired wound healing. Adipose tissue is poorly vascularized. In addition cardiac function is frequently compromised in obese patients, further diminishing tissue perfusion. As a result infection, seroma formation, anastomotic leak and wound dehiscence are all more common among the obese population.

- MEDICATION- medications compromising wound repair are chemotherapeutic drugs and NSAIDS. Former have an impact on rapidly dividing cells. The cells more profoundly affected are fibroblasts

and myofibroblasts, resulting in impaired collagen synthesis and wound contraction.

- STRESS-both psychological and physiological stress has been implicated as a potential cofactor in impaired wound healing. Stress elevates serum corticosteroid levels, which compromises immune function, and sympathetic stimulation which compromises perfusion due to vasoconstriction.
- IMMUNOSUPPRESSION- immunosuppression can retard wound healing and increase susceptibility to infection. This is attributed primarily to impairment of the inflammatory process.
- RADIATION THERAPY- it delays healing due to damage to the keratinocytes and fibroblasts as well as nutrient blood vessels.
- CIGARETTE SMOKING-it affects both perfusion and oxygenation. The three byproducts of cigarette smoking are nicotine, carbon monoxide, and hydrogen cyanide. Nicotine is a potent vasoconstrictor and potentiates platelet aggregation, carbon monoxide lowers oxygen saturation and hydrogen cyanide interferes with cellular transport of oxygen. Therefore a high incidence of wound infection, dehiscence and delayed healing is seen among smokers.
- LOCAL FACTORS-such as wound bed desiccation, ph, hypothermia, excess wound fluid, and/ or heavy bacterial colonization can affect the repair process. Wound healing is best supported by a moist, clean wound surface that is maintained at a temperature of about 30 degree Celsius.

SURGICAL SITE INFECTION

These are the infections present in any location along the surgical tract after a surgical procedure. Centre for disease control and prevention defines SSI as the one that can occur anytime from 0 to 30 days after the operation or up to 1 year after a procedure that has involved the implantation of foreign material (mesh, vascular grafts, prosthetic joint and so on).

Classification—Incisional superficial SSI

Incisional deep SSI

Organ/space related SSI

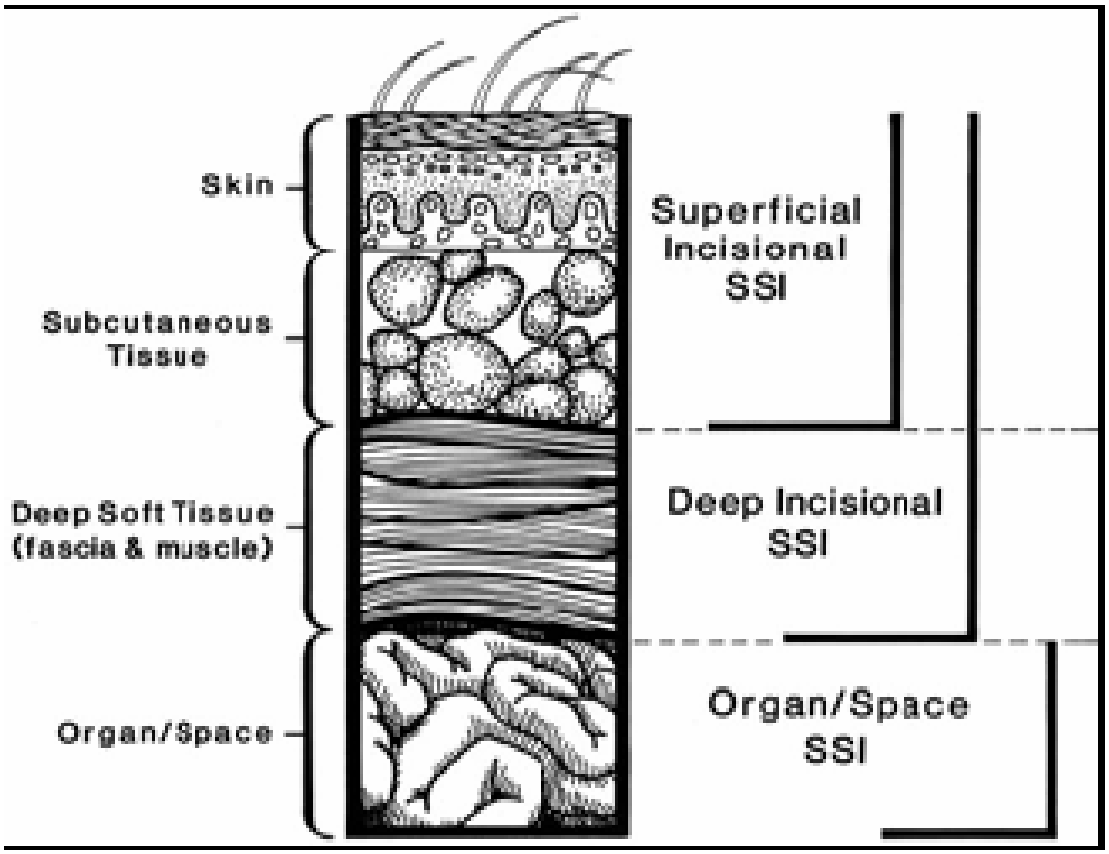


Fig.13:Types of SSI

SUPERFICIAL SSI

Infection which occurs within 30 days of surgery involving only skin and subcutaneous tissue and at least one of the following

a. Purulent discharge
b. Organisms isolated from aseptically cultured fluid and tissue
c. At least one sign of infection : pain or tenderness, localized swelling , redness or heat and incision is deliberately opened by surgeon unless the incision is culture negative
d. Diagnosis of SSI by surgeon or attending physician.

DEEP SSI

Infection occurring within 30 days of surgery or within 1 year of operation, if implants are in place; and infection involving deep soft tissue; and at least one of the following

a. Purulent discharge

b. Deep incision spontaneously dehisces or is deliberately opened by a surgeon when the patient has at least one of the following symptoms; fever more than 38 degree Celsius, localized pain or tenderness unless the site is culture negative

c. Evidence of deep infection on direct examination , during reoperation or on radiological examination

d. Diagnosis of SSI by surgeon.

ORGAN/SPACE SSI:

Infection occurring within 30 days of surgery or within 1 year of operation if implants are in place and infection involving any part of anatomy that was manipulated during an operation, other than the incision and at least one of the following:

a. Purulent discharge that is placed through a stab wound into the organ space.

b. Organisms isolated from and aseptically cultured fluid or tissue. Evidence of deep infection on the direct examination, during reoperation, or on radiological examination.

c. Diagnosis of SSI by surgeon or attending physician.

SSI are the most common nosocomial infection and constitute 38% of all infection in surgical patients. Incisional infections are the most common. They account for 60-80% of all SSI's and have better prognosis than organ/space related SSI's do, with the latter accounts for 93% of SSI related mortality.[14] Staphylococcus aureus remains the most common pathogen in SSI followed by coagulase negative staphylococcus, enterococci , and E.coli. Bacterial contamination > 10^5 organisms frequently causes infection whereas contamination with < 10^5 organisms usually does not.

SURGICAL WOUND CLASSIFICATION ACCORDING TO DEGREE OF CONTAMIMATION

A. **Clean:** uninfected operative wound in which no inflammation is encountered and respiratory, alimentary, genital, or infected urinary tract is not entered. Wounds are closed primarily and if necessary drained with closed drainage. Surgical wounds after blunt trauma should be included in this if they meet the criteria.

B. **Clean contaminated:** an operative wound in which the respiratory, alimentary, genital or urinary tract is entered under controlled conditions and without unusual contamination.

C. **Contaminated:** open, fresh, accidental wounds. In addition operation with major break in the sterile technique or gross spillage from gastrointestinal tract and incision in which acute nonpurulent inflammation is encountered are included in this category.

D. **Dirty:** old traumatic with retained and devitalized tissue and those that involve existing clinical infection or perforated viscera .This definition suggests that the organisms causing postoperative infection were present in the operative field before the operation.

The accepted range of infection rates has been 1-5% for clean, 3%-11% for clean contaminated, 10%-17% for contaminated, and >27% for dirty wounds.

Grade	Appearance
0	Normal healing
1	Normal healing with mild bruising or erythema
1a	Some bruising
1b	Considerable bruising
1c	Mild erythema
2	Erythema + other signs of inflammation
2a	At one point
2b	Around sutures
2c	Along wound
2d	Around wound
3	Clear or haemoserous discharge
3a	At one point only(<2cm)
3b	Along wound(>2cm)
3c	Large volume
3d	Prolonged (>3 days)
Major complications	
4	Pus
4a	At one point only (<2cm)
4b	Along wound (>2cm)
5	Deep or severe wound infection with or without tissue breakdown; haematoma requiring aspiration

Fig.14:Southampton wound grading system:

PULMONARY COMPLICATIONS:

A host of factors contributes to abnormal pulmonary physiology after an operative procedure. First loss of functional residual capacity is present in virtually all patients. This loss may be due to multitude of problems, including abnormal distention, a painful upper abdominal incision , obesity, a strong smoking history with associated COPD, prolonged supine positioning, and fluid over load leading to pulmonary edema.

Two types of respiratory failure are commonly described. Type1 or hypoxic with a low PaO2 and normal PaCO2. Type2 failure is associated with hypercapnia and is characterized by a low PaO2 and high PaCO2.

Patients with a history of heavy smoking, patients maintained on home oxygen, patients who are unable to walk one flight of stairs without severe respiratory compromise, patients with a previous history of major lung resection, and elderly patients who are malnourished all must be carefully screened with PFTs.[18] Several studies have found that a low levels of serum albumin is associated with a high risk of pulmonary complications and a higher overall mortality.[5]

PLASMA PROTEINS:

The concentration of total protein in human plasma is approximately 7.0–7.5 g/dl and comprises the major part of the solids of the plasma. The proteins of the plasma are actually a complex mixture that includes not only simple proteins but also conjugated proteins such as glycoproteins and various types of lipoproteins. Thousands of antibodies are present in human plasma, though the amount of any one antibody is usually quite low under normal circumstances.

One can separate the proteins of the plasma into three major groups— fibrinogen, albumin and globulins by the use of varying concentrations of sodium or ammonium sulfate.

The most common method of analyzing plasma proteins is by electrophoresis.[16]

Function	Plasma Proteins
Antiproteases	Antichymotrypsin α_1-Antitrypsin (α_1-antiproteinase) α_2-Macroglobulin Antithrombin
Blood clotting	Various coagulation factors, fibrinogen
Enzymes	Function in blood, eg, coagulation factors, cholinesterase Leakage from cells or tissues, eg, aminotransferases
Hormones	Erythropoietin[1]
Immune defense	Immunoglobulins, complement proteins, β_2-microglobulin
Involvement in inflammatory responses	Acute phase response proteins (eg, C-reactive protein, α_1-acid glycoprotein [orosomucoid])
Oncofetal	α_1-Fetoprotein (AFP)
Transport or binding proteins	Albumin (various ligands, including bilirubin, free fatty acids, ions [Ca^{2+}], metals [eg, Cu^{2+}, Zn^{2+}], metheme, steroids, other hormones, and a variety of drugs Ceruloplasmin (contains Cu^{2+}; albumin probably more important in physiologic transport of Cu^{2+}) Corticosteroid-binding globulin (transcortin) (binds cortisol) Haptoglobin (binds extracorpuscular hemoglobin) Lipoproteins (chylomicrons, VLDL, LDL, HDL) Hemopexin (binds heme) Retinol-binding protein (binds retinol) Sex hormone-binding globulin (binds testosterone, estradiol) Thyroid-binding globulin (binds T_4, T_3) Transferrin (transport iron) Transthyretin (formerly prealbumin; binds T_4 and forms a complex with retinol-binding protein)

[1]Various other protein hormones circulate in the blood but are not usually designated as plasma proteins. Similarly, ferritin is also found in plasma in small amounts, but it too is not usually characterized as a plasma protein.

Fig.15:Functions of plasma proteins

ALBUMIN:

It is a major protein of human plasma and makes up approximately 60% of the total plasma proteins. The normal serum value is between 3.5-5.5 gm/dl. Molecular weight is 69kda. 40% of total albumin is present in the plasma and the other 60% is present in the extracellular space. The liver produces about 12gm of albumin per day, representing about 25% of total hepatic synthesis and half its secreted protein.[15]

The total exchangeable pool of albumin is 4-5gm/kg body wt; between 6-10% of the exchangeable pool is degraded per day.[17]

Mature human albumin consists of one polypeptide chain of 585 amino acids and consists 17 disulfide bonds. It has ellipsoidal shape which meaning that it does not increase the viscosity of plasma. It is responsible for 75-80% of the osmotic pressure of the human plasma. To maintain adequate colloidal oncotic pressure serum albumin >2.5gm/dl and total protein >5gm/dl is sufficient.[41]

Albumin synthesis is decreased during fasting and in condition of protein malnutrition.[16] Albumin is a acute phase protein. The concentration of which is decreased by at least 25% following injury.[17]

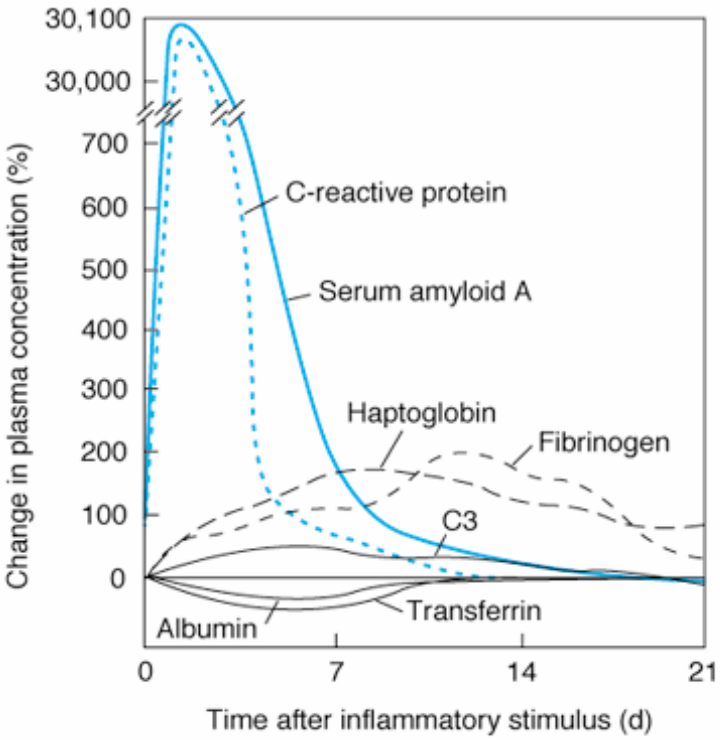


Fig.16:Time course of changes in some major acute phase proteins. C3, C3 component of complement.

EXTENT OF MALNUTRITION

- Normal- 3.5-5.5gm/dl
- Mild – 2.8-3.5gm/dl
- Moderate[1]- 2.1-2.7gm/dl
- Severe[1]- <2.1gm/dl

1- Require nutritional supplement. [9]

CAUSES OF HYPERALBUMINEMIA (>5.5g/dl)

There are no known pathological causes for a raised plasma albumin.

Causes include:

a. Dehydration: this is also reflected in an increase in plasma total protein, a raised hematocrit and appropriate clinical features.

b. Venous stasis

c. Albumin infusion

CAUSES OF HYPOALBUMINEMIA (<3.5g/dl)

a. Increase in plasma H_2O: This occurs as a part of physiological response in pregnancy. Other causes include, excessive infusion of IV fluids, H_2O retention in SIADH, glucocorticoid deficiency.

b. Diminished synthesis- any cause of generalized protein malnutrition will ultimately be reflected in low plasma albumin. Causes for this include: deficient diet in protein nitrogen; protein malabsorption such as coeliac disease, tropical sprue, crohn's disease, cystic fibrosis; decreased synthesis in chronic liver disease; in hereditary analbuminemia there is a marked impairment of albumin biosynthesis with plasma levels which are typically low.

c. Increased catabolism- This is a feature of hypercatabolic state. The important feature of hypercatabolic state is the stress related stimulation of glucocorticoid production. These hormones are known to stimulate protein catabolism. Conditions such as fever, trauma, major surgery, severe sepsis, malignant disease may all be associated with varying degrees of hypoalbuminemia.

d. Losses of albumin from body- sites of excessive loss are GI tract , kidneys and skin.[16]

Hypoalbuminemia is associated with delay in healing process, diminished collagen synthesis in healing wounds or at the anastomosis, and abnormal immune response such as macrophage activation and granuloma formation.

Therefore in hypoalbuminemic patients, local wound infection and other infections such as pneumonia and anastomotic leakage were commonly found.[41]

The precision and reliability of estimates of the association between preoperative serum albumin concentration and surgical outcome. A total of 54,215 major non cardiac cases were studied. A decrease in serum albumin from concentration > 46gm/dl to < 21gm/dl was associated with an exponential increase in mortality rates

from <1% to 29% and in morbidity rates from 10% to 65%. Albumin levels was a better predictor of some type of morbidity, particularly sepsis and major infection than other types. Serum albumin is a better predictor of surgical outcome than many other preoperative patient characteristics. It is relatively low cost test that should be used more frequently as a prognostic tool to detect malnutrition and risk of adverse surgical

outcomes.[16]

In a study of 158 patients on multivariate analysis , preoperative hypoalbuminemia was significantly associated with higher morbidity and longer hospital stay regardless of the type of surgery.[19]

In a study done on 202 patients who were planned for gastrointestinal surgery , several techniques of nutritional assessments were adapted to predict major post operative complications. Subjective global assessment(SGA) and albumin were both of predictive value , and combination of these variables were useful in differentiating low risk from high risk patients. It was concluded in study that SGA and albumin are useful "nutritional assessment techniques" for patients undergoing major gastrointestinal surgeries if the purpose of such an assessment is to predict postoperative nutrition associated complications.[20]

A retrospective study of 526 surgical patients who had preoperative serum albumin levels measured and were undergoing elective esophageal, gastric, pancreaticoduodenal, or colon surgery a serum albumin levels below 3.25gm/dl correlated immensely with complications, length of stay, postoperative stay, and mortality.[21]

A meta-analysis of cohort studies and controlled studies of hypoalbuminemia in acute illness; is there a rationale for intervention by Vincent et al shows that hypoalbuminemia was a potent dose dependant , independent predictor of poor outcome.[24] Each 10gm/dl decline in serum albumin concentration significantly raised the odds of mortality by 137%, morbidity by 89%, prolongs icu and hospital stay by 28% and 71% respectively. A serum albumin level of <2gm/dl in critically ill patients has been shown to be associated with a mortality of nearly 100%. The association between hypoalbuminemia and poor outcome appeared to be independent of both nutritional status and inflammation. The complication rates may be reduced when Serum albumin level attained during albumin administration exceeds 3.0gm/dl. Complications were higher when serum albumin level was lower than <2.5gm/dl in critically ill adult patients. [22] The cholesterol and serum albumin as a risk factor for death in patients undergoing general surgery, multivariate analysis revealed significant negative trends for serum albumin, total cholesterol and HDL-C ; for each variable a lower level was associated with a higher risk of death. Total cholesterol and its fraction were similar in patients with a serum albumin levels below 3.4gm/dl and in those with a higher level.[23]

The results indicate that low levels of serum albumin, total cholesterol and HDL-C are associated with risk of death up to 2years after general surgery.

In a study done on 434 patients who were evaluated for the accuracy of nutritional assessment tools for predicting adverse hospital outcomes, it was concluded that serum albumin level was the strongest predictive parameter for death and hospital infection(<3.5g/dl). A BMI <18.5 kg/m^2 was also associated with death and infection postoperatively and length of hospital stay.[24]

The role of serum albumin concentration on length of postoperative illness , the hypothesis was that patients whose albumin levels dropped below 3.5gm/dl would have a more prolonged postoperative hospital course as a result of delay in return of bowel function.[26]

Serum albumin below 3.5g/dl at the onset of treatment was a predictor of kidney and liver failure, hospital infection, and mortality in 12 patients strata.[27]

Increased incidence of pneumonia, wound infection, septicemia postoperatively was reported by Brown et al in their study in patients with serum albumin levels < 3g/dl.[28]

The preoperative nutritional status assessment in surgical patients and its relation to postoperative outcomes, anthropometry and biochemical indices are included. The more useful parameters were preoperative weight loss and low serum albumin levels.[29]

Patients with hypoalbuminemia (serum albumin levels <3g/dl) upon admission in surgical icu due to vascular insufficiency, hip fractures, gastrointestinal bleeding , cancer, perforated viscus, intra-abdominal infection or bowel obstruction. Complications were higher in patients with hypoalbuminemia (36.9%) and mortality of (5.8%).[30]

While assessing 6 patients submitted to elective urology and gynecology surgical procedures , Anderson et al observed that low albumin had a sensitivity of 22% and a specificity of 91% in predicting hospitalization lasting more than 10 days and a sensitivity of 10% and specificity of 86% for complications.[31]

In patients undergoing major intra-abdominal cancer surgery, obesity(BMI >30) is not a risk factor for postoperative mortality or major complications. Importantly, underweight(BMI <18.5) patients have a five fold increased risk postoperative mortality, perhaps a consequence of their underlying nutritional status.[32]

The effect of serum albumin and clinical outcome in paediatric patients undergoing cardiac surgery shows that, low serum albumin level (<3g/dl) was associated with a poor postoperative outcome which included increased postsurgical infection, increased mortality and longer hospital stays.[33]

In a study done on 183,069 pts subjected to general and vascular surgeries , it was determined through a logistic regression analysis that a serum albumin <3.5g/dl and weight loss >10% was associated with cardiac complication with a significant p value(0.0001).[34]

During the study of importance of BMI and serum albumin on mortality and morbidity after cardiac surgery, Serum albumin levels <2.5g/dl and BMI <20 and >30 was associated with increased risk of infection.[36]

In a study by Varut et al, 244 patients subjected to rectal cancer surgery, an assessment of the role of preoperative serum albumin as a risk factor for postoperative outcome was studied. It was concluded that preoperative hypoalbuminemia (<3.5gm/dl) is an independent risk factor for postoperative complications following rectal cancer surgery including mortality. Complications, time to first bowel movement, time to first

defecation, time to resumption of normal diet and length of hospital stay.[37]

The preoperative evaluation for postoperative pulmonary complications showed that low albumin levels was associated with respiratory failure and higher postoperative mortality and morbidity rates. Moreover morbidity increases exponentially as albumin levels fall below 4g/dl. Patients with >10% weight loss in 6 months prior to surgery are at increased risk for pneumonia and respiratory failure.[38]

The impact of BMI and tobacco smoking on outcome after open appendicectomy, concluded that tobacco smoking and a BMI of 27.5 or more were associated with more postoperative complications after open appendicectomy in patients with non-perforated appendicitis.[40]

In an univariate analysis study , low BMI and low serum albumin increased relative hazard for death and risk of infection. In a multivariate analysis study ,low BMI there is increased relative risk of death and low serum albumin there is increased risk for infection. In cardiac surgery patients with low BMI increased the relative hazard for death and a low S-albumin increased the risk of infection.[42]

Malnutrition is common in the elderly and may adversely affect surgical outcome. Compared to patients with normal albumin, hypoalbuminemic patients had an increased frequency of postoperative confusion, congestive heart failure, low cardiac output, renal dysfunction and gastrointestinal complications.[43]

Patients with low BMI are seen to be at higher risk for postoperative complications following cardiac surgery than normal or even severly obese patients.[44]

There is a high incidence of malnutrition in hospitalized patients undergoing gastrointestinal surgery. The prevalence of malnutrition in hospitalized patients is widespread and has consistently been reported to range from 30% to 50%.[45] Low preoperative serum albumin is associated with an increased risk of complications and mortality after radical cystectomy(RC). Serum albumin can be measured with a simple and inexpensive blood test that may identify patients at high risk of morbidity and mortality after RC. Such information may be useful for postoperative management of at-risk individuals.[46]

Lower BMI preoperatively is associated with a longer length of hospital stay and a trend towards a greater number of postoperative complications following pelvic exenteration for rectal cancer. Also, the nutritional status was not associated with long-term QoL in this patient group. However, the many limitations of this study indicate that further prospective research is essential to determine the nutritional status of patients before and after pelvic exenteration for rectal cancer and its association with postoperative outcomes.[47]

Low BMI and high BMI did not increase mortality after CABG, but patients with a higher BMI are at increased risk of complications after heart surgery and their outcomes are worse. In patients with low serum albumin levels, mortality after CABG is high. These patients also have higher postoperative complications. Values of albumin levels are more prognostic for patient outcome and are of further importance.[48]

MATERIALS

AND

METHODS

METHODOLOGY

SOURCE OF DATA:

Patients admitted to General surgical units in R L JALAPPA HOSPITAL AND RESEARCH CENTRE ATTACHED TO SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR during the period from January 2014 to August 2015.

METHOD OF COLLECTION OF DATA:

Type of study: Observational study

- Calculated sample size n=100

(using formula $n=4pq/l^2$ using prevalence of hypoalbuminemia as 40% and allowable error of 10%[10])

- Details of cases will be recorded including history and clinical examination and follow up till discharge.
- Anthropometry – height and weight recorded.
- Body Mass Index calculated.
- Pre-operative Serum albumin levels assessed.
- Observe for postoperative complications like infections, flap necrosis, pulmonary embolism, lower respiratory tract infection, fistulas and other complications and mortality.
- Correlate pre operative serum albumin and BMI with post operative morbidity and mortality.

INCLUSION CRITERIA :

Patients between age group 18 years to 80 years of both sexes.

EXCLUSION CRITERIA :

Patients who have jaundice , diabetes mellitus, chronic renal disease.

Patients with immunodeficiency states like AIDS, on steroids, radiotherapy and chemotherapy.

STATISTICAL ANALYSIS:

Chi square test (paired data)

Fischer's Exact test

Mean ± SD.

OBSERVATIONS

AND

RESULTS

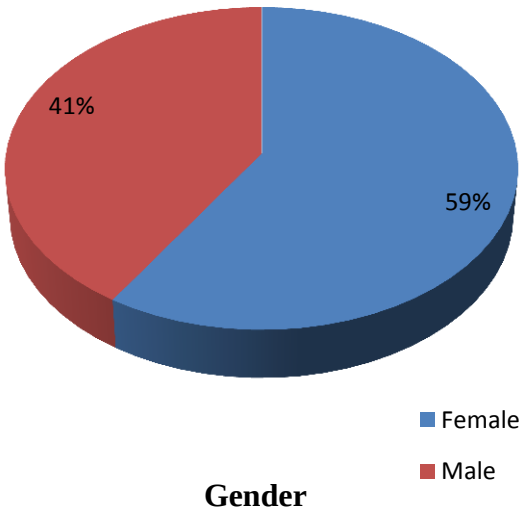
RESULTS:

The study was conducted on 100 patients, aged between 19 -79 yrs, who underwent any major elective surgeries at R L Jalappa Hospital, Kolar from January 2014 to August 2015. Among 100 patients, 46 patients developed complications and 54 had uneventful recovery.

Table.1: Gender wise distribution of patients studied

Gender	No. of patients	Percentage(%)
Female	59	59%
Male	41	41%
Total	100	100%

Graph.1:Gender wise distribution of patients studied



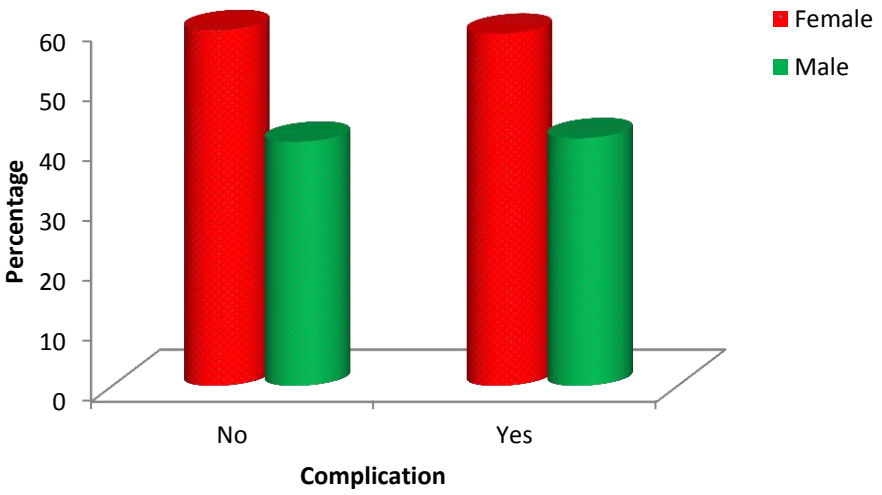
Total of 100 patients studied, in that 41% were male and 59% were female.

Table.2 Gender distribution of patients studied in relation to presence of complications

Gender	Complication		Total
	No	Yes	
Female	32(59.3%)	27(58.7%)	59(59%)
Male	22(40.7%)	19(41.3%)	41(41%)
Total	54(100%)	46(100%)	100(100%)

P=0.954, Not significant, Chi-Square test

Graph.2: Gender distribution of patients studied in relation to presence of complications



Of the 41 male patients 19 (41.3%) had complications and of the 59 female patients 27(58.7%) had complications.The P value found to be 0.954, which is not significant on comparing gender with complication

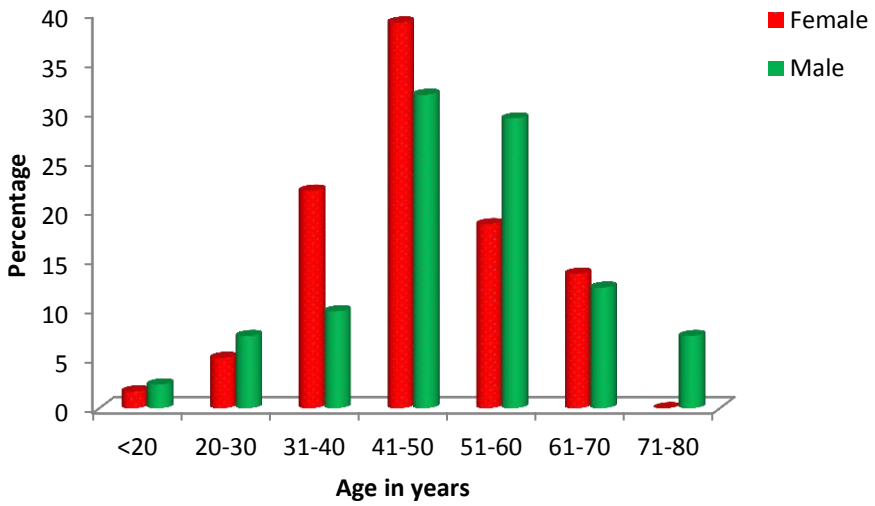
Table.3: Age-wise distribution of patients studied

Age in years	Gender		Total
	Female	Male	
<20	1(1.7%)	1(2.4%)	2(2%)
20-30	3(5.1%)	3(7.3%)	6(6%)
31-40	13(22%)	4(9.8%)	17(17%)
41-50	23(39%)	13(31.7%)	36(36%)
51-60	11(18.6%)	12(29.3%)	23(23%)
61-70	8(13.6%)	5(12.2%)	13(13%)
71-80	0(0%)	3(7.3%)	3(3%)
Total	59(100%)	41(100%)	100(100%)

Mean Age: 48.01±12.80

P=0.216, Not significant, Fisher Exact test

Graph 3 :Age-wise distribution of patients studied



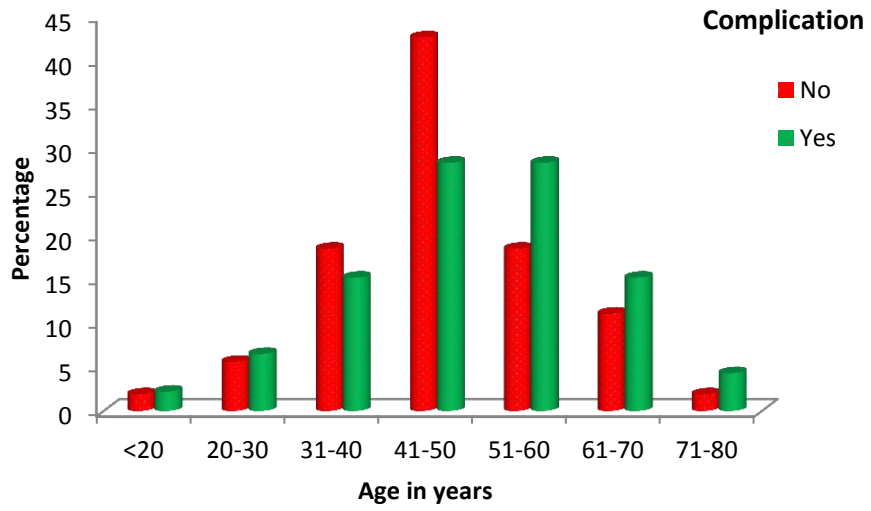
Of the 100 patients, the age varied from 19-79 yrs. The number of patients in the 41 – 60 years group was the highest (59%). The number of pateints >60 years were 16% and patients with <40 years age were 25%. The P value found to be 0.216, which is not significant.

Table.4: Age distribution of patients studied in relation to presence of complications

Age in years	Complication		Total
	No	Yes	
<20	1(1.9%)	1(2.2%)	2(2%)
20-30	3(5.6%)	3(6.5%)	6(6%)
31-40	10(18.5%)	7(15.2%)	17(17%)
41-50	23(42.6%)	13(28.3%)	36(36%)
51-60	10(18.5%)	13(28.3%)	23(23%)
61-70	6(11.1%)	7(15.2%)	13(13%)
71-80	1(1.9%)	2(4.3%)	3(3%)
Total	54(100%)	46(100%)	100(100%)

P=0.745, Not significant, Fisher Exact test

Graph 4: Age distribution of patients studied in relation to presence of complications

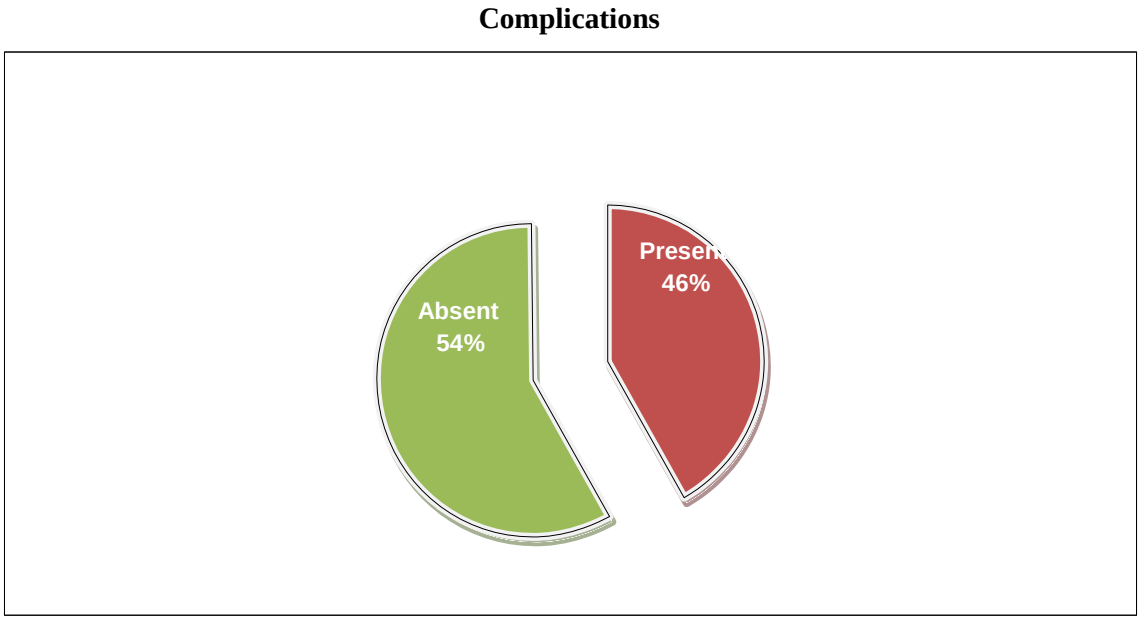


Among all the age group, patients between 41-60 years had the highest complication rate of 56.6%. Patients with age <40 years had 23.9% of complications, patients with age >60 years had 19.5% of complications. The P value found to be 0.745, which is insignificant.

Table.5: Distribution of number of patients based on complication

Complications	Frequency	Percentage (%)
Present	46	46%
Absent	54	54%
Total	100	100%

Graph. 5: Distribution of patients based on complications

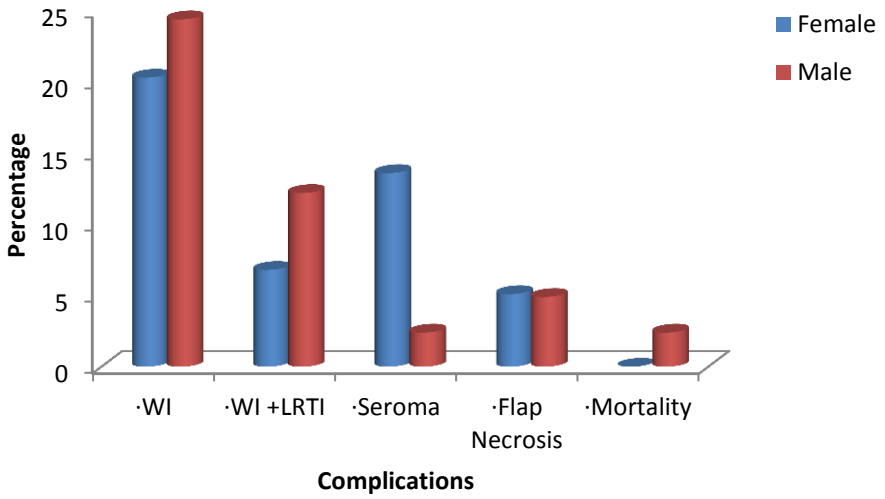


Out of 100 patients studied, 46% patients developed postoperative complications and remaining 54% had no complications.

Table.6:Pattern of complications studied in patients

Complications	Gender		Total (n=100)	Percentage (%)
	Female (n=59)	Male (n=41)		
• WI	12(20.3%)	10(24.4%)	22	47.8%
• WI +LRTI	4(6.8%)	5(12.2%)	9	19.6%
• Seroma	8(13.6%)	1(2.4%)	9	19.6%
• Flap Necrosis	3(5.1%)	2(4.9%)	5	11%
• Mortality	0	1(2.4%)	1	2%

Graph.6: Pattern of complications studied in pateints



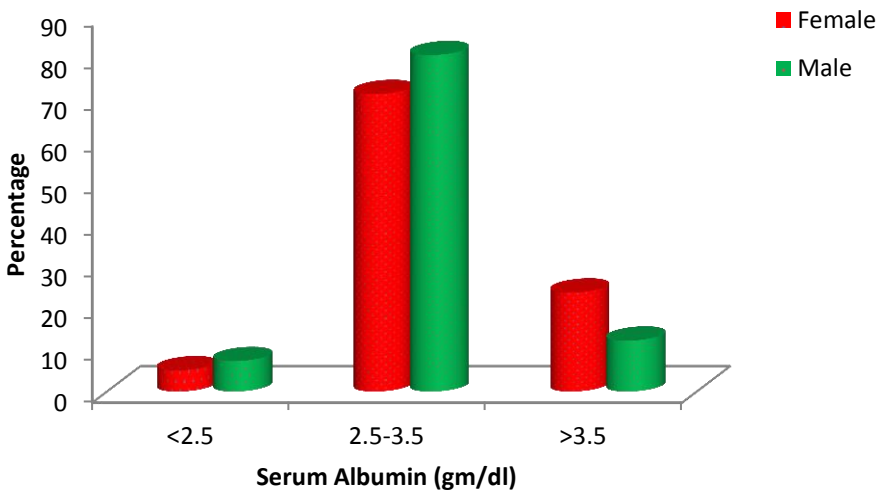
In our study, 46(46%) patients had complication out of 100 patients. Out of them about 22(47.8%) patients had wound infection which is the most commonest complication. Some patients also had associated LRTI with wound infection(19.6%), seroma(19.6%) and Flap necrosis(11%). In our study one mortality(2%) occurred postoperatively.

Table.7: Serum Albumin (gm/dl) distribution in patients studied

Serum Albumin (gm/dl)	Gender		Total
	Female	Male	
<2.5	5(8.5%)	5(12%)	10(10%)
2.5-3.5	40(67.8%)	31(75.6%)	71(71%)
>3.5	14(23.7%)	5(12.2%)	19(19%)
Total	59(100%)	41(100%)	100(100%)

P=0.359, Not significant, Fisher Exact test

Graph.7: Serum Albumin (gm/dl) distribution in patients studied



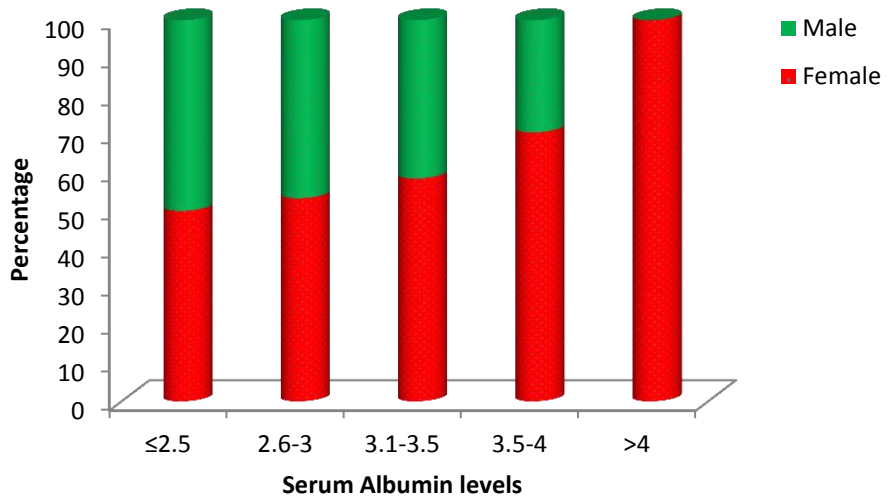
In our study of 100 patients, 71% of patients had serum albumin level between 2.5-3.5g/dl. Remaining 19% pts had serum albumin >3.5g/dl and 10% had serum albumin <2.5g/dl. The P value found to be 0.359, which is not significant for the study.

Table.8: Gender distribution of patients studied in relation to levels of serum albumin

Gender	Serum Albumin levels					Total
	≤2.5	2.6-3	3.1-3.5	3.5-4	>4	
Female	5(50%)	16(53.3%)	24(58.5%)	12(70.6%)	2(100%)	59(59%)
Male	5(50%)	14(46.7%)	17(41.5%)	5(29.4%)	0(0%)	41(41%)
Total	10(100%)	30(100%)	41(100%)	17(100%)	2(100%)	100(100%)

P=0.636, Not significant, Chi-Square test

Graph.8: Gender distribution of patients studied in relation to levels of serum albumin



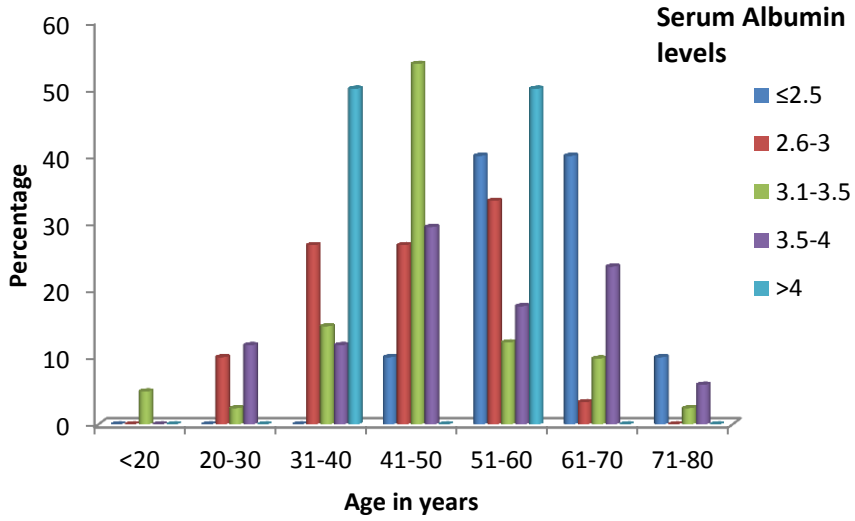
Out of 100 patients of our study, female patients constitutes a larger group with abnormal serum albumin levels(<3.5g/dl) compared to male patients. The P value found to be o.636, which is not significant.

Table.9: Age distribution of patients studied in relation to levels of serum albumin

Age in years	Serum Albumin levels					Total
	≤2.5	2.6-3	3.1-3.5	3.5-4	>4	
<20	0(0%)	0(0%)	2(4.9%)	0(0%)	0(0%)	2(2%)
20-30	0(0%)	3(10%)	1(2.4%)	2(11.8%)	0(0%)	6(6%)
31-40	0(0%)	8(26.7%)	6(14.6%)	2(11.8%)	1(50%)	17(17%)
41-50	1(10%)	8(26.7%)	22(53.7%)	5(29.4%)	0(0%)	36(36%)
51-60	4(40%)	10(33.3%)	5(12.2%)	3(17.6%)	1(50%)	23(23%)
61-70	4(40%)	1(3.3%)	4(9.8%)	4(23.5%)	0(0%)	13(13%)
71-80	1(10%)	0(0%)	1(2.4%)	1(5.9%)	0(0%)	3(3%)
Total	10(100%)	30(100%)	41(100%)	17(100%)	2(100%)	100(100%)

P=0.046*, significant, Fisher Exact test

Graph.9: Age distribution of patients studied in relation to levels of serum albumin



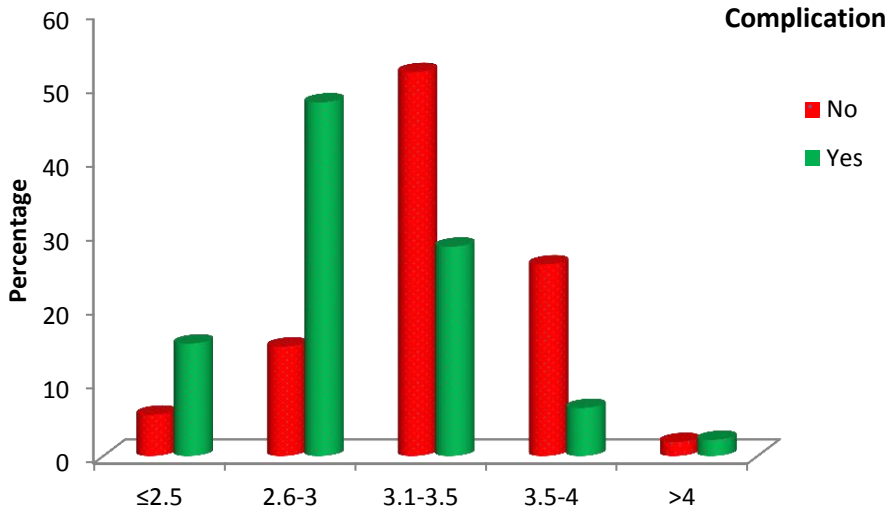
Out of 100 patients studied in our study, patients with age group 41-60 years have more percentage of abnormal serum albumin (<3.5g/dl) compared to other age groups. The P value found to be 0.046, which significant for the study.

Table.10: Serum Albumin of patients studied in relation to presence of complications

Serum Albumin	Complication		Total
	No	Yes	
≤2.5	3(5.6%)	7(15.2%)	10(10%)
2.6-3	8(14.8%)	22(47.8%)	30(30%)
3.1-3.5	28(51.9%)	13(28.3%)	41(41%)
3.5-4	14(25.9%)	3(6.5%)	17(17%)
>4	1(1.9%)	1(2.2%)	2(2%)
Total	54(100%)	46(100%)	100(100%)

P<0.001**, significant, Fisher Exact test

Graph.10: Serum Albumin of patients studied in relation to presence of complications



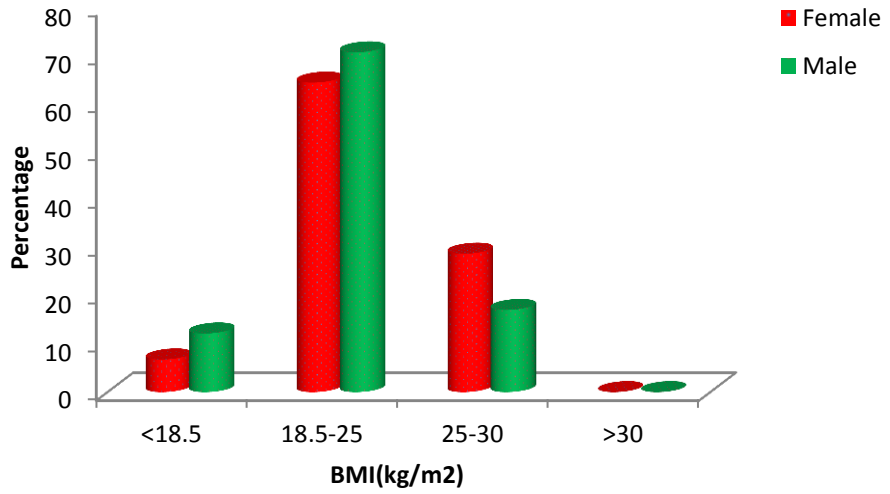
It was observed that the complication rate was more when levels of serum albumin was less than 3.0 gm/dl which is statistically significant(P <0.001). There is significant difference in rate of complications, when serum albumin levels were <3.0, and >3.0gm/dl. Serum albumin level >3.5gm/dl were associated with statistically significant lower complications. It was found that patients with serum albumin <3.0 g/dl had 72.5% complication. Patients with serum albumin 3.1-3.5g/dl had 31.7% and patients with serum albumin >3.5g/dl had 21% complication.

Table.11: BMI (kg/m^2) distribution in patients studied

BMI (kg/m^2)	Gender		Total
	Female	Male	
<18.5	4(6.8%)	5(12.2%)	9(9%)
18.5-25	38(64.4%)	29(70.7%)	67(67%)
25-30	17(28.8%)	7(17.1%)	24(24%)
>30	0(0%)	0(0%)	0(0%)
Total	59(100%)	41(100%)	100(100%)

P=0.326, Not significant, Chi-Square test

Graph.11: BMI (kg/m^2) distribution in patients studied



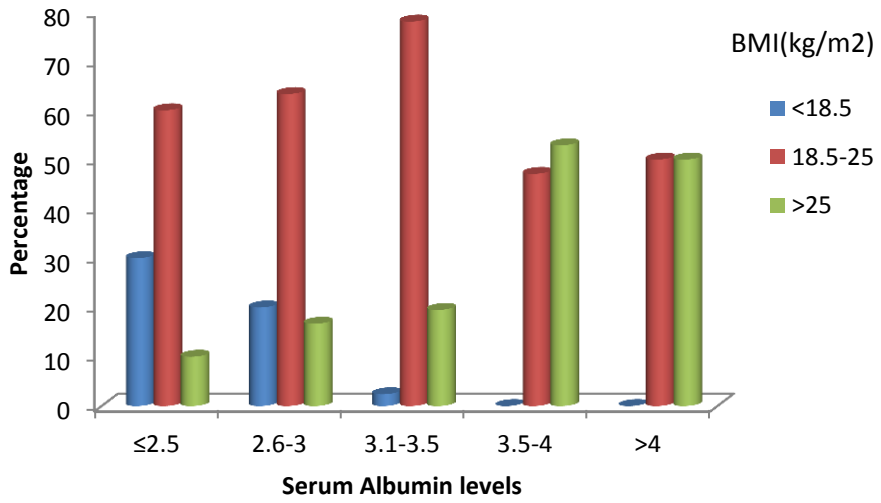
Out of 100 patients studied in our study, maximum number of patients (67%) had BMI if 18.5-25, 24% had BMI of >25 and 9% had BMI of <18.5. The P value found to be 0.326, which is not significant.

Table.12: BMI (kg/m^2) of patients studied in relation to levels of serum albumin

BMI (kg/m^2)	Serum Albumin levels					Total
	≤2.5	2.6-3	3.1-3.5	3.5-4	>4	
<18.5	3(30%)	6(20%)	1(2.4%)	0(0%)	0(0%)	10(10%)
18.5-25	6(60%)	19(63.3%)	32(78%)	8(47.1%)	1(50%)	66(66%)
>25	1(10%)	5(16.7%)	8(19.5%)	9(52.9%)	1(50%)	24(24%)
Total	10(100%)	30(100%)	41(100%)	17(100%)	2(100%)	100(100%)

P=0.005**, significant, Fisher Exact test

Graph.12: BMI (kg/m^2) of patients studied in relation to levels of serum albumin



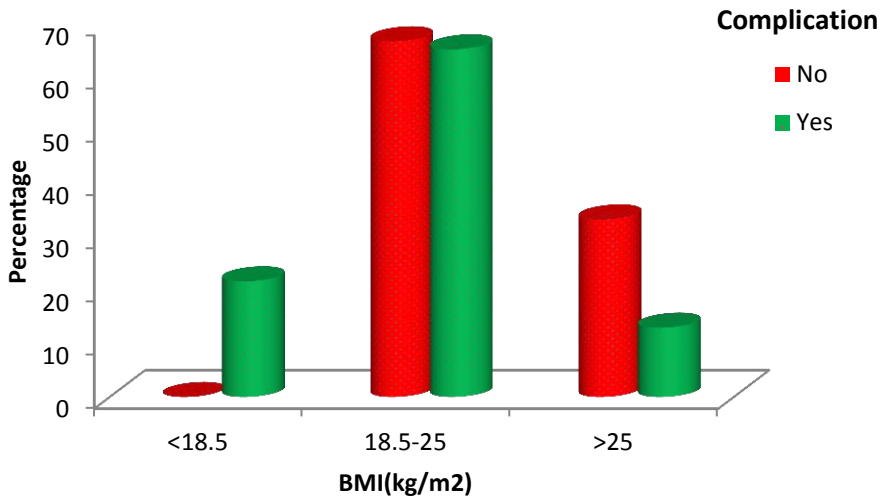
In comparison of BMI with Serum albumin levels, patients with low BMI (<18.5) had more percentage of low Serum albumin <2.5 g/dl, patients with BMI of 18.5 to 25 had more percentage of Serum albumin 2.6-3.5g/dl and patients with BMI >25 had more percentage of Serum albumin >3.5g/dl. The P value found to be <0.005, which is significant.

Table.13: BMI (kg/m^2) of patients studied in relation to presence of complications

BMI (kg/m^2)	Complication		Total
	No	Yes	
<18.5	0(0%)	10(21.7%)	10(10%)
18.5-25	36(66.7%)	30(65.2%)	66(66%)
>25	18(33.3%)	6(13%)	24(24%)
Total	54(100%)	46(100%)	100(100%)

P<0.001**, significant, Fisher Exact test

Graph.13: BMI (kg/m^2) of patients studied in relation to presence of complications



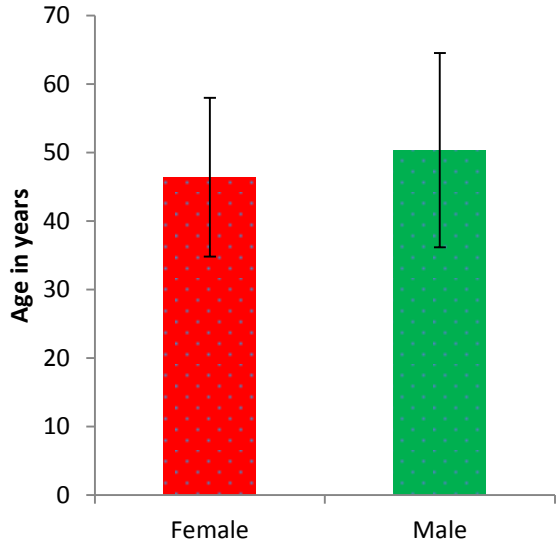
The complication rate was found to be high with BMI<18.5kg/m^2. In our study, the complication rate with BMI < 18.5 was 100%, between 18.5-25 was 45%, >25 was 25% . The P value found to be <0.001, which is significant for the study.

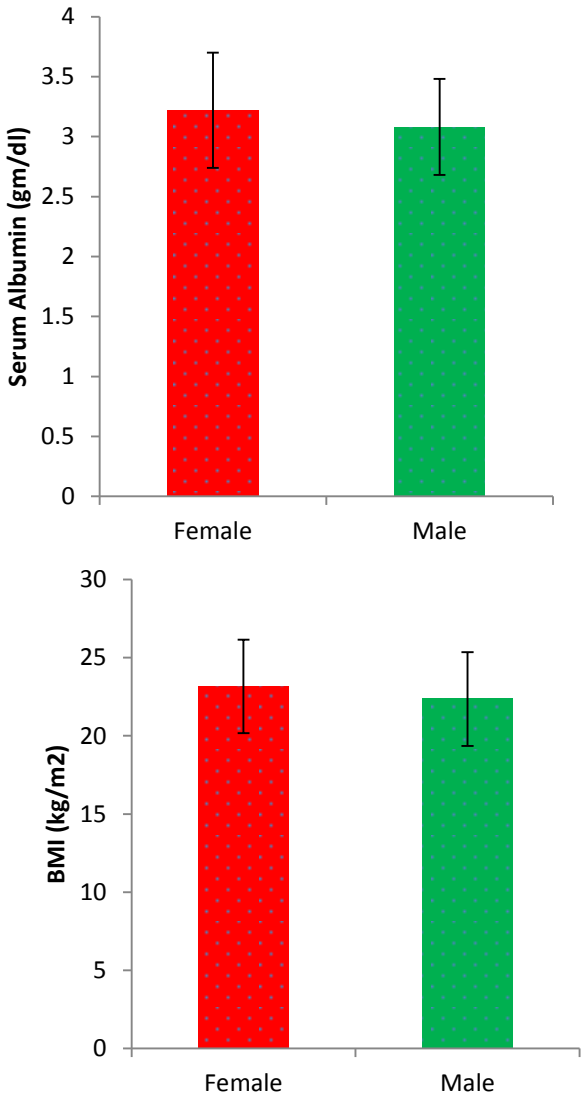
Table.14: Comparison of variables according to gender

variables	Gender		Total	P value
	Female	Male		
Age in years	46.39±11.59	50.34±14.19	48.01±12.80	0.130
BMI (kg/m^2)	23.16±2.99	22.36±3.00	22.83±3.00	0.194
Serum Albumin (gm/dl)	3.22±0.48	3.08±0.40	3.16±0.45	0.114

Student t test

Graph.14:Comparison of variables according to gender





In total of 100 patients studied, on comparing the variables like Age, BMI and Serum albumin with Gender, it was found that the mean Age is 48.01±12.80(P 0.130), mean BMI is 22.83±3.0(P 0.194) and mean Serum albumin is 3.16±0.45(P 0.114).

DISCUSSION

DISCUSSION:

The association between hypoalbuminemia and poor outcome are associated with each other and they are based on both nutritional status and inflammation. The complication rates may be reduced when Serum albumin level attained during albumin administration exceeds 3.0gm/dl. Complications were higher when serum albumin level was lower than <2.5gm/dl in critically ill adult patients.[21]

In a study done on 434 patients who were evaluated for the accuracy of nutritional assessment tools for predicting adverse hospital outcomes, it was concluded that serum albumin level(<3.5g/dl) was the strongest predictive parameter for death and hospital infection. A BMI <18.5 kg/m2 was also associated with death, postoperative infection and length of hospital stay.[23]

Nutritional assessment for patients undergoing surgery is very much helpful for identifying those who are at risk of developing complications post operatively. A variety of nutritional indices have been found to be valuable in predicting patient outcome. Preoperative serum albumin level and BMI were used for nutritional assessment of patients in several studies.

Patients with serum albumin levels <3g/dl upon admission in surgical icu, complications were higher in patients with hypoalbuminemia of about 36.9% and mortality of about 5.8%.[29] While assessing 6 patients subjected to elective urology and gynecology surgical procedures , Anderson et al observed that low albumin had a sensitivity of 22% and a specificity of 91% in predicting hospitalization lasting more than 10 days and a sensitivity of 10% and specificity of 86% for complications.[30]

In patients undergoing major intra-abdominal cancer surgery, obesity(BMI >30) is

not

a risk factor for postoperaive mortality or major complications. Importantly, underweight(BMI <18.5) patients have a five fold increased risk of postoperative mortality, perhaps a consequence of their underlying nutritional status.[31] Obese individuals had more wound complication than thin patients.

The effect of serum albumin and clinical outcome in paediatric patients undergoing cardiac surgery shows that, low serum albumin level (<3g/dl) was associated with a poor postoperative outcome which included increased postsurgical infection, increased mortality and longer hospital stays.[32]

The study conducted by Gibb's et al observed that a decrease in serum albumin from concentrations greater than 4.6 g/l to less than 2.1 g/l was associated with an exponential increase in mortality rates from less than 1% to 29% and in morbidity rates from 10% to 65%. In the regression models, albumin level was the strongest predictor of mortality and morbidity for surgical patients. Albumin level is a good predictor of some types of complications like sepsis and major infections, and it is a good prognostic indicator,

In a study of 244 surgical patients by Varut Lohsiriwat et al in their study evaluates that pre-operative hypoalbuminemia is a major risk factor for post operative omplications following rectal Cancer surgeries. The study suggests that the albuminemia of <3.5gm/dl is an independent risk factor for post operative complications following rectal cancer surgery as well as post operative bowel function and hospital stay.[36]

Table 15: Significance of serum albumin levels in predicting postoperative outcomes.

Study name	Sr. Alb g/dl associated With increased complications	P value
Beghetto et al	<3.5	<0.05
Leite et al	<3	<0.05
Brown et al	<3	<0.05
Engelman et al	<2.5	<0.001
Foley et al	<2.5	<0.001
Present study	<3	<0.001

During the study of importance of BMI and serum albumin on mortality and morbidity after cardiac surgery by Engelman et al, Serum albumin levels <2.5g/dl and BMI <20 and > 30 was associated with increased risk of infection[35]. It is observed that albumin less than 2.5 g/dl ($p<0.001$) and BMI less than 20 kg/m2 ($p<0.005$) and greater than 30 kg/m2 ($p<0.005$) was associated with increase in post operative complications .

The impact of BMI and tobacco smoking on outcome after open appendicectomy, concluded that tobacco smoking and a BMI of 27.5 or more were associated with more postoperative complications after open appendicectomy in patients with non-perforated appendicitis($p<0.001$).[39]

Malnutrition is common in the elderly and may adversely affect surgical outcome. In a study by Michael et al on malnutrition, outcome and nutritional support suggest pre-operative risk indications like BMI < 18.5kg/m2 and serum albumin <2.1gm/dl have an impact on surgical outcome. Such patients are clearly malnourished or catabolic and consequently have longer ICU stays, unable to tolerate full treatment regimens, and experience a 40-60% greater frequency of complications in response to surgical/medical treatments. Compared to patients with normal albumin hypoalbuminemic patients had an increased frequency of postoperative confusion, congestive heart failure, low cardiac output, renal dysfunction and gastrointestinal complications.[43]

The study by Jessica beaton suggested that, lower BMI preoperatively is associated with a longer length of hospital stay and a trend towards a greater number of postoperative complications following pelvic exenteration for rectal cancer. Also, the nutritional status was not associated with long-term QoL in this patient group. This will help to better tailor nutrition-assessment practices and nutrition interventions for these patients.[47]

Present study: The observations in our study shows that patients with serum albumin less than 3g/dl had more postoperative complications compared to patients with serum albumin >3.5g/dl, which was statistically significant($p<0.001$). The study concludes that as the serum albumin level is low, the complication rate is

high. Wound infection being the most common complication (48%) of the overall complications.

During study of BMI in relation to complications, our study shows that the complication rate was high(100%) in BMI group<18.5kg/m^2 compared to patients with normal BMI(45%) and in patients with >25 BMI has 25% complication rate.It shows it is statistically significant($p<0.001$).

CONCLUSION

CONCLUSION:

Our study shows that serum albumin is a good indicator of postoperative complications. An abnormal BMI was associated with more complications.

Maximum percentage patients with complications were noted with serum albumin <2.5g/dl. The complication rate was almost similar with serum albumin range of 2.5-3.0, and 3.0-3.5g/dl. The patients with serum albumin <3.0g/dl had a higher complication rate which was statistically significant ($p<0.001$). Patients with serum albumin >3.5g/dl had less complications which was statistically significant ($p<0.001$).

It is observed that patients with abnormal BMI (<18.5)had more complications compared to patients with normal BMI(18.5 to 25). Patients with BMI <18.5, all the patients had developed complications.

Serum albumin concentration is a better predictor of surgical outcomes than any other preoperative patient characteristics. It is a relatively low cost test that should be used more frequently as a prognostic tool to detect malnutrition and risk of adverse surgical outcomes, particularly in populations in whom co-morbid conditions are relatively common.

BMI is also relatively easy bed side test to measure the nutritional status of surgical patients. It also helps to assess the malnutrition in the patients admitted in hospital.

Thus serum albumin amd BMI are good prognostic indicators because of its ability to detect PEM, and malnutrition in the patients and are associated with significant increased risk of morbidity and mortality.

SUMMARY

SUMMARY:

The above study was conducted with 100 patients who underwent major elective surgeries at R L Jalappa Hospital and Research Centre attached to Sri Devaraj Urs Medical College, Tamaka, Kolar during the perod from January 2014 to August 2015.

Serum albumin and BMI were estimated preoperatively and postoperative complications were observed.

Statistical analysis was done using Chi square test and Fischer exact test, p value < 0.05 was considered as significant value.

Serum albumin values of < 3.0g/dl was associated with increased post operative morbidity and mortality and is statistically significant.

Patients with BMI <18.5 and >25 were more prone to increased complication rate. Both of these were statistically significant.

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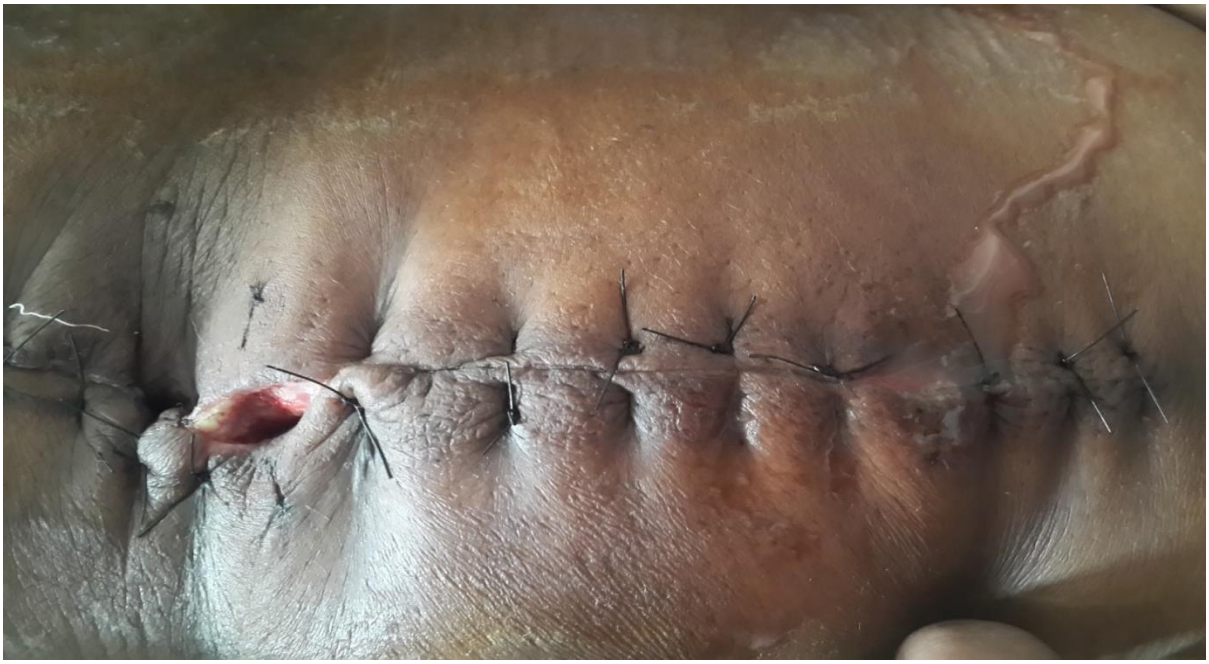
ANNEXURES

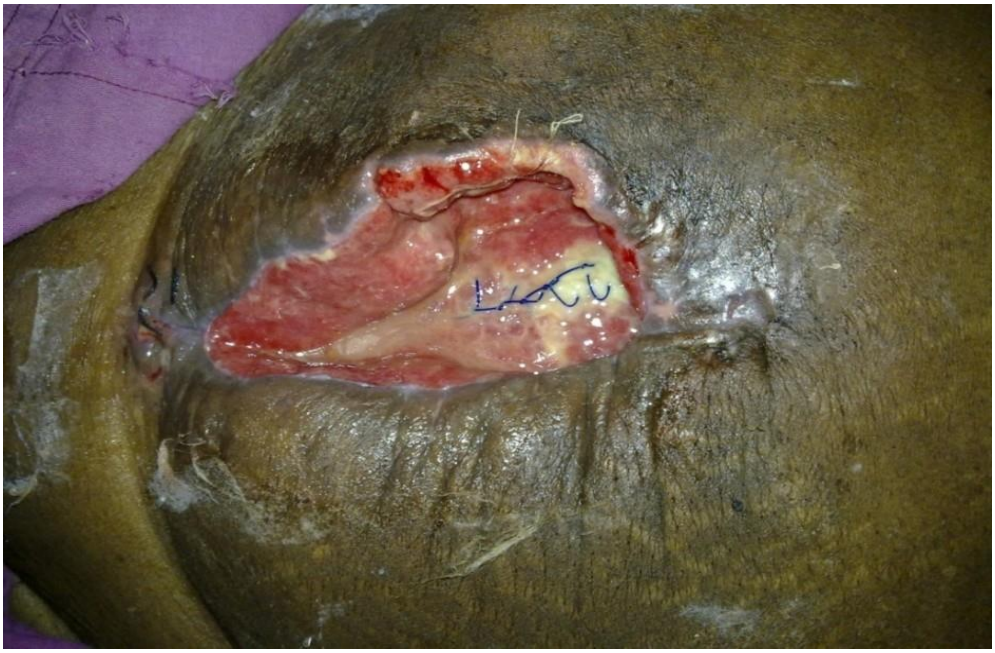
ANNEXURE :

PHOTOS:

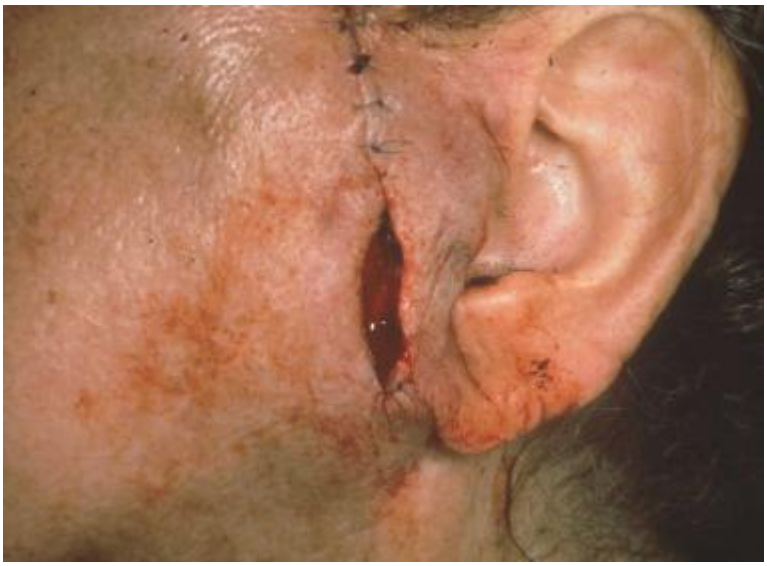
1. INFECTED POSTOPERATIVE WOUND



2: DEEP INCISIONAL SSI



3.FLAP NECROSIS



PROFORMA

Name : **DOA:**

Age : **DOD:**

Sex : **IP/OP NO:**

Religion : **Unit No:**

Education: **Date of surgery:**

Occupation :

Address:

1. CHIEF COMPLAINTS:

2. HOPI

3. PAST HISTORY:

4. PERSONNEL HISTORY
5. MENSTRUAL HISTORY:

6. FAMILY HISTORY :

7. GENERAL PHYSICAL EXAMINATION

Built and nourishment :
Level of Consciousness :

Vital Data
Temperature:
Pulse :
BP :
RR :
Pallor : **YES OR NO**
Icterus : **YES OR NO**
Clubbing :**YES OR NO**
Cyanosis :**YES OR NO**
Lymphadenopathy :**YES OR NO**
Edema :**YES OR NO**

Height: cms Weight: kgs

BMI: <18.5
18.5 to 24.9
25 to 29.9
>30

8. SYSTEMIC EXAMINATION

Per Abdomen:

A Inspection

B. Palpation:

C. Percussion:

D. Auscultation:

Respiratory System:

Cardiaovascular System:

Central Nervous system:

Per Rectum:

Per vagina:

Local Examination:

X. INVESTIGATIONS:

A. BLOOD

Hb %:
TC:
RBS:
Blood Urea:
Serum Creatinine:
Serum albumin:

10. Diagnosis:

MANAGEMENT:

SURGERY PERFORMED :

POSTOPERATIVE PROGRESS :

COMPLICATIONS:
SEROMA:
WOUND INFECTION :
WOUND INFECTION WITH LRTI:
FLAP NECROSIS:
DEHISCENCE :

OTHERS :

MORTALITY :

INFORMED CONSENT FORM

I,_____ have been told about the study in my own understandable language (_____). I have been told that this is for dissertation procedure, that my participation is voluntary and I reserve the full right to withdraw from the study at my own initiative at any time, without having to give any reason, and that decision to participate or withdraw from the study at any stage will not prejudice my rights and welfare. Confidentiality will be maintained and only be shared for academic purposes

I hereby give consent to participate in the above study. I am also aware that I can withdraw this consent at any later date, if I wish to. This consent form being signed voluntarily indicates agreement to participate in the study and the procedures involved, until I decide otherwise.

Signature of the subject:
Date:
Place:
Contact address:

I, Dr. Munireddy.M.V, post graduate student in the Department of General Surgery conducting dissertation work for award of MS Degree in General Surgery

The study Topic is "**Evaluation of preoperative serum albumin and body mass index as predictors of postoperative morbidity and mortality in elective major surgeries"**

I here by state that the study and procedures involved were explained in detail and all questions were fully and clearly answered to the above mentioned participant/her relative.

Investigators Signature:
Date:
Place:

KEY TO MASTER CHART

BMI	:Body Mass Index
Ca	:Carcinoma
CJ	:Cystojejunostomy
Ex lap	:exploratory laparotomy
F	:Female
FND	:functional neck dissection
GJ	:Gastrojejunostomy
Lt	:Left
LRTI	:Lower Respiratory tract infection
M	:Male
MNG	:Multinodular goiter
MRM	:Modified Radical Mastectomy
MRND	:Modified Radical neck dissection
RA	: Resection and anastomosis
RND	:Radical Neck Dissection
Rt	: Right
Sup	:Superficial
Sr.Alb	:Serum albumin
TV	:Truncal Vagotomy
WI	:Wound infection

MASTER CHART

SL NO	IP NO	NAME	AGE(yrs)	SEX	DISEASE	PROCEDURE DONE	BMI(kg/m2)	Sr.Alb(gm/dl)	COMPLICATION
1	352	Ravi babu	55	M	Umbilical hernia	Meshplasty	23	2.8	Yes WI
2	1745	Jay lakshmi	66	F	Umbilical hernia	Meshplasty	28	2.5	Yes WI
3	2183	Suma	35	F	Incisional hernia	Hernioplasty	25	3.2	No
4	2639	Zareen	35	F	Cholelithiasis	Open cholecystectomy	26		Yes, SEROMA
5	3863	Pushpa	40	F	Incisional hernia	Meshplasty	18.0	3.2	Yes,WI
6	4195	Govindappa	55	M	Multinodular goiter	Subtotal thyroidectomy	24	3.1	No
7	9312	Bhagyamma	45	F	Umbilical hernia	Meshplasty	27	3.5	No
8	904915	Vedavathi	26	F	Umbilical hernia	Meshplasty	26	3.9	No
9	914258	Venkatamma	43	F	Solitary hyroid nodule	Hemithyroidectomty	19.0	3.4	No
10	937211	Amaravathi	38	F	Ca left breast	MRM Lt side	22	2.6	Yes,Flap necrosis
11	946257	Rajappa	55	M	Inguinal hernia right side	Lichenstein hernioplsty	25.4	3.6	No
12	946200	Devraj	20	M	Tropical splenomegaly	Splenectomy	20.6	3.5	No
13	948376	Pothappa	65	M	Carcinoma ascending colon	Right hemicolectomy	18.6	2.4	Yes, WI,LRTI
14	959755	Tabsum	32	F	Umbilical hernia	Meshplasty	26	3.4	No
15	964318	Nagaraj	57	M	Chronic duodenal ulcer	Truncal Vagotomy with GJ	20.8	3.0	Yes, WI
16	968715	Girija	35	F	Incisional hernia	Meshplasty	24	3.8	No
17	952780	Kalavathi	52	F	Papillary Ca thyroid	Total thyroidectomty with MRND		2.8	Yes, SEROMA
18	967382	Fathima	60	F	Cholelithiasis	Open cholecystecyomy	28.3	3.9	No
19	962545	Papalamma	65	F	Multinodular goiter	Total thyroidectomy	23	2.3	No
20	970740	Parvathamma	50	F	Recurrent incisional hernia	Hernioplasty	22	2.0	Yes, WI
21	972563	Ramappa	38	M	Inguinl hernia left side	Lichenstein hernioplasty	23	2.6	No
22	967369	Narasamma	45	F	Ca breast right side	MRM Rt side	25.4	3.7	No
23	970720	Sarala	31	F	Cholelicystitis	open cholecystectomy	27	3.0	No
24	972563	Ramakka	55	F	Carcinoma stomach-pylorus	Distal partial gastrectomy	20.4	2.8	Yes, WI
25	965284	Kempamma	32	F	Incisional hernia	Hernioplasty	28	4.2	No
26	972985	Feroz	53	M	Cholelithiasis	Open cholecystectomy	27	2.8	Yes,WI, LRTI
27	9753084	Nagamma	45	F	Ca thyroid	Total thyroidectomy	19.6	3.0	No
28	962581	Mala	42	F	Ventral hernia	Meshplasty	27.2	3.4	No
29	977183	Rudersh	60	F	Veriose veins left lower limb	Flush ligation and stripping	23.4	3.8	No
30	976101	Sarojamma	50	F	Ca breast right side	MRM Rt side	23	3.0	No
31	978251	Amzad kha	45	M	Chronic duodenal ulcer	T V and GJ	23.4	3.1	Yes,WI,LRTI
32	977462	Rajesh	40	M	Divarication of recti with epigastric hernia	Hernioplasty	21.4	2.8	No
33	977738	Rama	47	F	Supraumbilical Hernia	Meshplsty	22.6	3.5	No
34	977374	Sowbhagya	49	F	Ductal Ca breast right side	MRM Rt side	22	3.2	No
35	977702	Beerappa	45	M	Inguinal hernia	Lichenstein hernioplasty	21.2	3.2	No

36	977716	Krishnappa	68	M	Cholelithiasis	Open cholecystectomy	28	3.8	No	
37	977775	Eshwaramma	65	F	Incisional hernia	Meshplasty	24	3.2	Yes, WI	
38	977836	Shivanna	28	M	Acute intestinal obstruction	Laparotomy, resection anastamosis of bowel	18.4	3.0	Yes,WI	
39	977852	Thimmakka	65	F	Multinodular goiter	Total thyroidectomy	25.4	3.6	No	
40	986891	Devalakshmi	32	F	Chronic pancreatitis	Puestow's procedure	21	3.0	Yes, SEROMA	
41	987329	Frazana	45	F	Ca rectum	Abdominoperineal resection	20.5	3.1	YES,WI	
42	989735	Basher	42	M	Umbilica hernia	Meshplasty	25	3.2	No	
43	989821	Rangaraj	45	M	Varicose veins right lower limb	Flush ligation and stripping	26	3.2	No	
44	991829	Rajasri	34	F	Ca brest left side	MRM Lt side	23.4	3.0	Yes, WI,LRTI	
45	992481	Anasuyamma	65	F	Infraumbilical incisional hernia	Hernioplsty	24	4.0	No	
46	994183	Makbul	50	F	Cholelithiais	Open colecystectomy	23.2	3.6	Yes, WI,LRTI	
47	992911	Sarojamma	67	F	Appendicular mass	Adhesiolysis	23.5	3.2	Yes, WI	
48	993872	Gangamma	52	F	Ca stomach	Gastrectomy with esophogojejunostomy	18.0	2.5	Yes,SEROMA	
49	994952	Ramachandrappa	50	M	Umbilical hernia	Meshplasty	24.3	3.4	No	
50	995379	Chowdappa	55	M	Ca sigmoid colon	Lt hemicolectomy	18.2	2.7	Yes,WI	
51	995427	Nareppa	50	M	Benign tumour over back of chest	Excision	26	3.8	No	
52	995632	Narayanappa	53	M	Epigastric hernia	Hernioplasty	23.1	3.2	Yes,WI	
53	995047	Venkatamma	45	F	Bilateral ca breast	MRM	23	3.1	Yes, Flap necrosis	
54	996273	Rajappa	42	M	Pleomorphic adenoma of lt parotid	Sup parotidectomy	23.5	3.4	No	
55	996438	Daniel	68	M	Cholelithiasis	Open cholecystectomy	24.2	3.8	No	
56	996707	Raghavendra	32	M	Paraumbilical hernia	Anatomical repair	26	3.5	No	
57	997329	Laxmipathy	42	M	Chronic duodenal ulcer	T V and posterior GJ	24	3.2	Yes,WI,LRTI	
58	999111	Shylaja hegde	48	F	Incisional hernia	Meshplasty	27	3.4	No	
59	1000458	Srinivas	58	M	Hydatid cyst of liver	Cystopericystectomy	17.4	3.0	Yes,Flap necrosis	
60	1000713	Raju	60	M	Cholelithiasis	Open cholecystectomy	23.5	2.5	No	
61	999337	Aluvelamma	42	F	Ca breast right side	MRM	24.2	3.1	Yes,WI,LRTI	
62	999450	Gantalamma	32	F	Incisional hernia	Anatomical repair	27	3.7	No	
63	1000052	Ramesh	45	M	Right inguinal hernia	Hernioplasty	21.5	3.2	Yes, SEROMA	
64	1001122	Md ali beg	60	M	Umbilical hernia	Meshplasty	28	3.1	No	
65	1001652	Doddareddy	72	M	Ca stomach	Gastrectomy and esophagojejunostomy	16.8	2.4	Mortality	
66	1002193	Neelamma	57	F	Papillary ca thyroid	Total thyroidectomy	22.1	2.7	Yes,WI	
67	1002426	Husnabee	45	F	MNG	Total thyroidectoy	20	3.2	No	
68	1002464	Balaram	52	M	Verocse veins left lower limb	Trendalenburg procedure	22.7	2.4	No	
69	1002480	Gangalamma	45	F	Cholelithiasis	Open cholecystectomy	18.6	2.6	Yes,WI, LRTI	
70	1003261	Veda	42	F	Parotid tumour	Sup parotidectomy	18.6	3.4	No	

71	1004551	Vasavi	40	F	Paraumbilical Hernia	Meshplasty	25	3.2	Yes,WI	
72	1005251	Rizvana	27	F	Incisional hernia	Hernioplasty	26	3.9	Yes, SEROMA	
73	1009575	Mohasagulla	65	M	Bilateral direct inguinal herni	Hernioplasty	18.5	2.5	Yes, WI	
74	1009756	Praveen	20	M	Left indirect inginal hernia	Hernioplasty	24	2.6	No	
75	1014503	Pushpa	62	F	Cholelithiasis	Open cholecystectomy	29	3.0	Yes,Flap necrosisI	
76	1015402	Asmat	62	F	Ca breast right side	MRM Rt side	19.2	3.5	Yes,SEROMA	
77	1007175	Anwarkhan	65	M	Cholelithiasis	Open cholecystectomy	25	3.3	No	
78	1019774	Devendra	47	M	Paraumbilical hernia	Open meshplsty	19.2	2.9	Yes,WI	
79	92261	Thulasi	19	F	Intramuscular cysticercosis left calf muscle	Excision	23.4	3.4	No	
80	89472	Sriramappa	60	M	Monomorphic adeoma left parotid	Sup parotidectomy	19.8	3.4	No	
81	116205	anilkumar	19	M	Pleomorphic adenoma right parotid	Sup parotidectomy	23	3.2	Yes,Flap necrosis	
82	119580	Eshwaramma	47	F	Umbilical hernia	Meshplasty	25.6	3.4	No	
83	166182	Lakshmidevamma	45	F	MNG	Subtotal thyroidectomy	19.6	3.5	No	
84	92242	Sujathamani	45	F	Ca stomach	Partial gastrectomy with GJ	17.8	2.7	Yes,WI	
85	119175	Subramani	47	M	Bilateral hydrocele	Jaboulays procedure	18.8	2.8	No	
86	122180	Jayalakshmi	40	F	Fibroadenoma right breast	Excision	21.9	3.5	No	
87	127392	Ramesh	48	M	Chronic duodenal ulcer	T V and GJ	18.9	3.0	Yes, WI, LRTI	
88	119320	Nalini	48	F	Cholelithiasis	Open cholecystectomy	24.2	3.6	No	
89	124421	Usha	54	F	Ca breast right side	MRM Rt side	23.1	2.8	No	
90	127273	Zainaba	54	F	Bilateral varicose veins	Flush ligation and stripping	23.2	4.1	Yes,SEROMA	
91	129292	Deepa	22	F	Solitary thyroid nodule	Hemithyroidectomy	25.3	2.6	Yes,WI	
92	1021061	Muniyappa	75	M	Inguinal hernia right side	Hernioplasty	22.6	3.7	Yes WI	
93	148469	Venkatarathnamma	45	F	Ca breast right side	MRM Rt side	24	3.2	No	
94	1021160	Narayanappa	50	M	Ca stomach	Distal gastrectomy with GJ	17.6	2.7	Yes,WI	
95	155605	Krishnappa	38	M	Ileocaecal tuberculosis	Rt hemicilectomy with primary RA	22.1	3.0	Yes,WI	
96	162203	Gopal gowda	79	M	Inguinal hernia left side	Herniorraphy	22.4	3.4	No	
97	167964	Venkatamma	50	F	Ca brest right side	MRM Rt side	20.6	3.8	No	
98	174594	Jayamma	58	F	Femoral hernia right side	Herniorraphy	17.2.	2.7	Yes,SEROMA	
99	176625	Vijayamma	55	F	Varicose veins right lower limb	Trendalenburg operation	22.4	3.5	No	
100	188297	Eshwaramma	55	F	Ca stomach	Distal partial gastrectomy with GJ	20	2.3	Yes,WI	