“PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES”

Dissertation Submitted to

THE TAMILNADU Dr. MGR MEDICAL UNIVERSITY

Chennai-600 032

In partial fulfillment of the regulations for the Award of the degree

of

M.S. (General Surgery)

Branch – I

MADRAS MEDICAL COLLEGE

CHENNAI

May 2020

CERTIFICATE

This is to certify that, the dissertation entitled **"PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES"** Is the bonafide work done by **DR. V. NAVEEN KUMAR,** during his **M.S. (General Surgery)** course 2017 - 2020, done under my supervision and is submitted in partial fulfillment of the requirement for the M.S.(BRANCH-I)- General Surgery of The Tamilnadu Dr. MGR Medical University, May 2020 examination.

GUIDE

Prof. Dr. M. ALLI M.S., DGO

Professor
Institute of General Surgery
Madras Medical College
Chennai – 03.

HOD

Prof. DR. R. KANNAN

Professor & Director
Institute of General Surgery
Madras Medical College
Chennai – 03.

DEAN

DR. R. JAYANTHI M.D., FRCP,
THE DEAN
Madras Medical College & Rajiv Gandhi Government
General Hospital
Chennai-03

DECLARATION

I solemnly declare that this dissertation **"PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES"** was prepared by me at Institute of General surgery, madras medical college and RAJIV GANDHI GOVERNMENT GENERAL HOSPITAL, CHENNAI under the guidance and supervision of **PROF. DR. M. ALLI. M.S., D.G.O,** professor of general surgery, institute of general surgery, madras medical college, Chennai. This dissertation is submitted to the Tamil Nadu DR.MGR Medical University, Chennai in fulfillment of the university regulation for the award of the degree M.S. General Surgery (branch 1).

DR. V. NAVEEN KUMAR, M.B.B.S,

Post Graduate in General Surgery

Madras Medical College

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First, I would like to extend my sincere thanks and appreciation towards all our **patients** for their willingness to co-operate with the study.

My inexpressible gratitude to my mentor, **PROF. DR. M. ALLI M.S**, **D.G.O,** Professor and Unit Chief, institute of General Surgery, Madras Medical College, Chennai, for her constant encouragement and skillful guidance at each step of the preparation of this work. Her enthusiasm, zeal for perfection and eagerness for exploring the depth of learning helped me a lot to understand various aspects of the subject. It was only due to her constant inspiration, efforts and suggestions that this study was possible.

I would like to express my heartfelt thanks to my HOD, **PROF. R. KANNAN M.S** whose constant motivation and encouragement kept me strive harder and better to complete the thesis work.

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LIST OF ABBREVATIONS

SSI – Surgical Site Infection

Ab - Antibiotics

CI – Confidence Interval

DM – Diabetes Mellitus

CHF – Congestive Heart Failure

SHT – Systemic Hypertension

IV – Intravenous

CDC – Center for Disease Control and prevention

Hb - Hemoglobin

MCV – Mean Corpuscular Volume

WBC – White Blood Cell

Alb - Albumin

C/S – Culture and Sensitivity

MRSA – Methicillin Resistant Staphylococcus aureus

MBP – Mechanical Bowel Preparation

E.coli – Escherichia coli

P.aeruginosa – Pseudomonas aeruginosa

CBD – Common Bile Duct

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LIST OF TABLES

PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES

QUESTIONAIRE

Name: Age: Sex:

IP No:

Chief complaints:

DIAGNOSIS :

PROCEDURE PLANNED :

INVESTIGATIONS :

- Hemoglobin
- MCV
- WBC count
- Serum albumin
- RBS
- Serum urea
- Serum creatinine
- Pus culture and sensitivity

COMPLICATIONS	**OCCURRED OR NOT**	**DAY OF OCCURENCE**
Seroma		
Sinus or fistula formation		
Wound dehiscence		
Incisional hernia (Follow up)		
Pus c/s		

INFORMATION SHEET

TITLE: **"**PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES**"**

Name of Investigator: Dr. V. NAVEEN KUMAR

Name of Participant:

Study Procedure: For the study, patient demographic details, results of investigations and details of surgical procedures were documented. Patients were followed up during postoperative period and at monthly interval for 6 months. Occurrence of complications like wound discharge, sinus, fistula, wound dehiscence, incisional hernia was noted. These were analyzed to see for any correlation and conclusion will be derived.

Possible Risks: No risks to the patient

Possible benefits

To patient:

Early preventive measures that permits early treatment which in turn improves survival rates, mortality and morbidity.

To doctor & to other people:

This study will give an idea about predictors of surgical wound infection and a protocol can be devised accordingly. This can also give a rough estimate of wound infection in a tertiary teaching center.

Confidentiality of the information obtained from you:

The privacy of the patients in the research will be maintained throughout the study. In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared. Taking part in this study is voluntary. You are free to decide whether to participate in this study or to withdraw at any time. Your decision will not result in any loss of benefits to which you are otherwise entitled.

Signature of Investigator Signature of Participant

Date:

Place:

PATIENT CONSENT FORM

Study Title : **"PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES"**

Study Centre : Rajiv Gandhi Government General Hospital, Chennai.

Patient's Name :

Patient's Age :

In Patient Number :

Patient may check (☑) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction.	❑
I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected.	❑
I understand that sponsor of the clinical study, others working on the sponsor's behalf, the Ethics committee and the regulatory authorities will not need my permission to look at my health records, both in respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study.	❑

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well being or any unexpected or unusual symptoms.	❑
I hereby consent to participate in this study	❑
I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests and to undergo treatment	❑

Signature/thumb impression

Patient's Name and Address:

Signature of Investigator

Study Investigator's Name:

CERTIFICATE – II

This is to certify that this dissertation work titled **"PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES"** of the candidate **Dr. V. NAVEEN KUMAR** with registration Number for the award of **M.S degree** in the branch of **General Surgery**. I personally verified the urkund.com website for the purpose of plagiarism Check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 7% percentage of plagiarism in the dissertation.

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EC Reg. No. ECR/270/Inst./TN/2013
Telephone No.044 25305301
Fax: 011 25363970

CERTIFICATE OF APPROVAL

To
Dr. Naveen kumar. V
Postgraduate in M.S General Surgery
Institute of General Surgery
Madras Medical College
Chennai
Dear Dr. Naveen kumar. V.

The Institutional Ethics Committee has considered your request and approved your study titled **"PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES" – NO. 12012018**

The following members of Ethics Committee were present in the meeting hold on **09.01.2018** conducted at Madras Medical College, Chennai 3

1. Prof. P.V.Jayashankar :Chairperson
2. Prof.R.Narayana Babu,MD., DCH., Dean, MMC,Ch-3 : Deputy Chairperson
3. Prof. Sudha Seshayyan,MD.,Vice Principal,MMC, Ch-3 :Member Secretary
4. Prof. N. Gopalakrishnan,MD.,Director, Institute of Nephrology,MMC,Ch :Member
5. Prof.S.Mayilvahanan,MD,Director,Inst. Of Int.Med,MMC,Ch-3 :Member
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12. Thiru. S. Govindasamy, BA., BL, High Court, Chennai :Lawyer
13. Tmt. Arnold Saulina, M., MSW., :Social scientist
14. Thiru K. Ranjith, Ch-91 : Lay Person

We approve the proposal to be conducted in its presented form.

The Institutional Ethical Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

9/1/2018

Member Secretary – Ethics Committee

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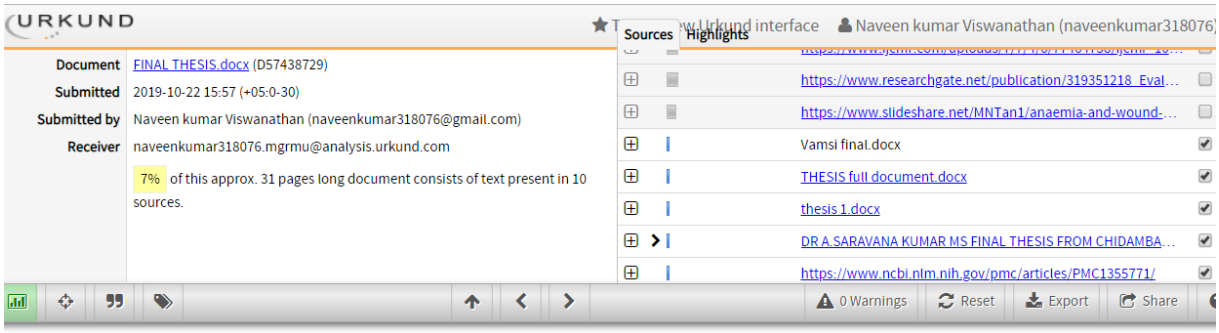

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In partial fulfillment of the regulations for the Award of the degree of M.S. (General Surgery) Branch – I

MADRAS MEDICAL COLLEGE CHENNAI May 2020

CERTIFICATE

This is to certify that, the dissertation

entitled "PREDICTORS AND EVALUATION OF SURGICAL WOUND COMPLICATIONS IN ELECTIVE ABDOMINAL SURGERIES" Is the bonafide work done by DR. V. NAVEEN KUMAR, during his M.S. (General Surgery) course 2017 - 2020, done under my supervision and is submitted in partial fulfilment of the requirement for the M.S.(BRANCH-I)- General Surgery of The Tamilnadu Dr. MGR Medical University, May 2020 examination.

GUIDE HOD

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In partial fulfillment of the regulations for The award of the Degree of M.S DEGREE BRANCH -1 GENERAL SURGERY

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INTRODUCTION

Postoperative wound infections, also known as surgical site infections (SSIs), complicate the recovery course of many patients. As defined by the Centers for Disease Control and Prevention (CDC), these infections typically occur within 30 days of an operation at the site or part of the body where the surgery took place, or within a year if an implant is left in place and the infection is thought to be secondary to surgery.[1–3] Bacterial colonization on the patient's skin and alimentary and genital tract are the principal contributing sources that lead to SSIs.[4] The organism most often isolated is *Staphylococcus aureus*.[5] Exogenous sources, such as breaches in sterile technique and operating room equipment may contribute, albeit much less frequently than endogenous flora.[6] Bacteria within the tissue or organ space hinder the postoperative healing processes, and can lead to anastomotic leaks, wound dehiscence, and superficial incisional infections.

SSIs may be classified as superficial/incisional if limited to the skin and subcutaneous tissue, deep incisional when involving the fascia and muscle, or organ space when involving a body cavity (eg, abdominal cavity following gastrointestinal surgery).[2,3] Deep tissue and organ space

SSIs are less frequently encountered than superficial SSIs, but are associated with greater morbidity/mortality, readmission rates, longer hospital stay, and increased overall hospital-associated costs when compared with superficial SSIs.[7–9] Although the majority of SSIs are uncomplicated, others may be severe and more challenging to manage, such as necrotizing deep soft tissue infections.[2,8] The latter often require extensive surgical debridement, multiple reoperations, and may even be life-threatening.[10,11] The location and extent of the infection, as well as the patient's clinical condition, guide the management approach.[2,10] For instance, in the setting of an implant, as in the case for a synthetic mesh in an infected wound, often times explanation of the implant is required, which may add to the postoperative morbidity. Furthermore, appropriate antibiotic therapy is often necessary to achieve source control in such patients.

With the rising incidence and associated morbidity of SSIs, various studies have looked at ways to better optimize patients prior to surgery or improve surgical technique and management of patients during the recovery period in order to prevent SSIs.[12,13] Data regarding a hospital's rate of SSIs are becoming increasingly used as outcome measures for assessing the quality of their surgical services.[14,15] Employing methods

that could reduce the incidence of SSI would significantly reduce patient morbidity and mortality while lessening the associated economic burden; this has become central to quality improvement initiatives.[16] Herein, the authors provide an update on the epidemiology, risk factors, identification, and management of wound infections following abdominal surgery.

The goal of an operative procedure is an early and complication-free recovery. Post-operative pain, nausea, vomiting are common but some patients develop short and long term complications like fever, wound infection, wound dehiscence, anastomosis disruption, adhesive bowel obstruction, incisional hernia, etc. Such complications are more frequently seen after emergency surgeries, but they do occur in elective procedures also, which is a matter of concern.[18]

Wound infection, wound dehiscence and incisional hernia remain challenging problems. Preoperative antibiotic prophylaxis, effective and persistent skin antisepsis, avoidance of contamination and better surgical skills are most effective methods to reduce complications. Depending on operative conditions, wound infection rates vary from 2.8% to 40%.[19, 20]

Factors like site of surgery, size and depth of incision, antibiotic prophylaxis, instruments and suture material being used, wound closure technique, patient related factors like comorbidities and life style habits like smoking, have significant effect on occurrence of such events. Other factors responsible for complications to occur are anemia, hypoproteinemia, diabetes, jaundice, uremia, COPD, steroids use, obesity, advanced malignancy and advanced age.[21]

AIMS AND OBJECTIVES

- To analyze various factors that may predict the surgical wound complication following elective abdominal surgeries and to evaluate the occurrence of various surgical wound complications.

REVIEW OF LITERATURE

Postoperative wounds are those wounds acquired during surgical procedures. Postoperative wound healing occurs after surgery and normally follows distinct bodily reactions; the inflammatory response and it can be effectively managed well before.

There is significant prolonged stay of patients due to wound dehiscence. The variables associated are hypoalbuminemia, anemia, malnutrition, chronic lung disease and emergency procedure. Other additional factors are increased coughing, vomiting, prolonged intestinal paralysis, repeated urinary retention. Obesity, chronic heart disease, diabetes, alcoholism, preoperative intestinal obstruction, jaundice, systemic and local infections were found to be nonsignificant.[23]Surgical site infections (SSIs) are defined as infections occurring up to 30 days after surgery (or up to one year after surgery in patients receiving implants) and affecting either the incision or deep tissue at the operation site. Despite improvements in prevention, SSIs remain a significant clinical problem as they are associated with substantial mortality and morbidity and impose severe demands on healthcare resources.

The incidence of SSIs may be as high as 20%, depending on the surgical procedure, the surveillance criteria used, and the quality of data collection. In many SSIs, the responsible pathogens originate from the patient's endogenous flora. The causative pathogens depend on the type of surgery; the most commonly isolated organisms are *Staphylococcus aureus*, coagulase negative staphylococci, *Enterococcus* spp. and *Escherichia coli*. Numerous patient-related and procedure-related factors influence the risk of SSI, and hence prevention requires a 'bundle' approach, with systematic attention to multiple risk factors, in order to reduce the risk of bacterial contamination and improve the patient's defenses.

The Centers for Disease Control and Prevention guidelines for the prevention of SSIs emphasize the importance of good patient preparation, aseptic practice, and attention to surgical technique; antimicrobial prophylaxis is also indicated in specific circumstances. Emerging technologies, such as microbial sealants, offer the ability to seal and immobilize skin flora for the duration of a surgical procedure; a strong case therefore exists for evaluating such technologies and implementing them into routine clinical practice as appropriate[24].

Surgical site infections complicate the postoperative course of a significant proportion of general abdominal surgical patients and are associated with excessive health care costs. SSIs increase postoperative morbidity and mortality and may require hospital admission, intravenous antibiotic and even surgical reintervention.[26]

Despite modern surgical and sterilization techniques and prophylactic use of good antibiotics, postoperative wound infection remains a major contributory factor of patient's morbidity. The overall postoperative wound infection rate was 11% in their study. It can reduced by taking proper measures to improve our operation theatres and wars environment, and methods of sterilization.[27]

Sahu et al study reveals a superficial incisional surgical site infection incidence rate of 4.3%. Length of stay, duration of surgery and diabetes mellitus were found to be major risk factors responsible for causing surgical site infection. Minimizing the incidence of postoperative wound infection relies on adequate on adequate asepsis and antisepsis and preservation of the local host defences.[28]

SURGICAL SITE INFECTION CRITERIA:

Superficial incisional SSI

Must meet the following criteria:

Date of event occurs within 30 days after any NHSN operative procedure (where day 1 = the procedure date)

AND

involves only skin and subcutaneous tissue of the incision

AND

patient has at least ***one*** of the following:

- purulent drainage from the superficial incision.

- organism(s) identified from an aseptically-obtained specimen from the superficial incision or subcutaneous tissue by a culture or non-culture based microbiologic testing method which is performed for purposes of clinical diagnosis or treatment (for example, not Active Surveillance Culture/Testing (ASC/AST)).

- superficial incision that is deliberately opened by a surgeon, attending physician or other designee and culture or non-culture based testing of the superficial incision or subcutaneous tissue is not performed **AND** patient has at least one of the following signs or symptoms: localized pain or tenderness; localized swelling; erythema; or heat.

- diagnosis of a superficial incisional SSI by the surgeon, attending physician or other designee.

Superficial Incisional SSI

There are two specific types of superficial incisional SSIs:

1. Superficial Incisional Primary (SIP) – a superficial incisional SSI that is identified in the primary incision in a patient that has had an operation with one or more incisions (for example, C-section incision or chest incision for CBGB)

2. Superficial Incisional Secondary (SIS) – a superficial incisional SSI that is identified in the secondary incision in a patient that has had an operation with more than one incision (for example, donor site incision for CBGB)

Organ/Space SSI

Must meet the following criteria:

Date of event occurs within 30 or 90 days after the NHSN operative procedure (where day 1 = the procedure date) **AND** involves any part of the body deeper than the fascial/muscle layers that is opened or

manipulated during the operative procedure **AND** patient has at least ***one*** of the following:

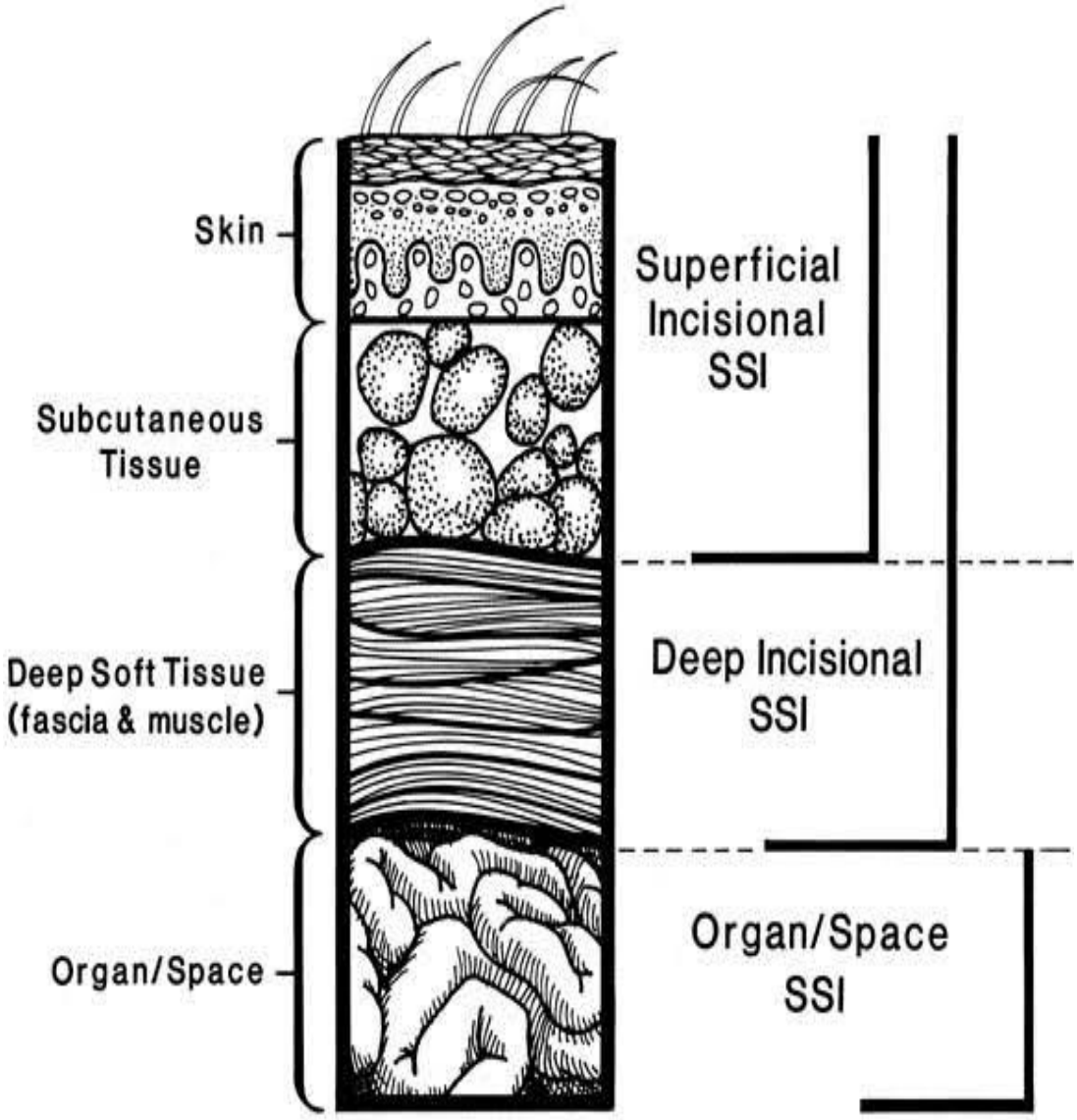
- purulent drainage from a drain that is placed into the organ/space(for example, closed suction drainage system, open drain, T-tube drain, CT-guided drainage).

- organism(s) identified from fluid or tissue in the organ/space by a culture or non-culture based microbiologic testing method which is performed for purposes of clinical diagnosis or treatment (for example, not Active Surveillance Culture/Testing (ASC/AST)).

- an abscess or other evidence of infection involving the organ/space that is detected on gross anatomical or histopathologic exam, or imaging test evidence suggestive of infection.

AND

meets at least ***one*** criterion for a specific organ/space infection[25]



WOUND HEALING PROCESS:

Wound healing is a dynamic process consisting of four continuous, overlapping, and precisely programmed phases. The events of each phase must happen in a precise and regulated manner. Interruptions, aberrancies, or prolongation in the process can lead to delayed wound healing or a non-healing chronic wound.

In adult humans, optimal wound healing involves the following the events: (1) rapid hemostasis; (2) appropriate inflammation; (3) mesenchymal cell differentiation, proliferation, and migration to the wound site; (4) suitable angiogenesis; (5) prompt re-epithelialization (re-growth of epithelial tissue over the wound surface); and (6) proper synthesis, cross-linking, and alignment of collagen to provide strength to the healing tissue.[29] The first phase of hemostasis begins immediately after wounding, with vascular constriction and fibrin clot formation.

The clot and surrounding wound tissue release pro-inflammatory cytokines and growth factors such as transforming growth factor (TGF)-β, platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), and epidermal growth factor (EGF). Once bleeding is controlled, inflammatory cells migrate into the wound (chemotaxis) and promote the inflammatory phase, which is characterized by the sequential infiltration of neutrophils, macrophages, and lymphocytes.[29] A critical function of neutrophils is the clearance of invading microbes and cellular debris in the wound area, although these cells also produce substances such as proteases and reactive oxygen species (ROS), which cause some additional bystander damage.

Macrophages play multiple roles in wound healing. In the early wound, macrophages release cytokines that promote the inflammatory response by recruiting and activating additional leukocytes. Macrophages are also responsible for inducing and clearing apoptotic cells (including neutrophils), thus paving the way for the resolution of inflammation. As macrophages clear these apoptotic cells, they undergo a phenotypic transition to a reparative state that stimulates keratinocytes, fibroblasts, and angiogenesis to promote tissue regeneration.[30] In this way, macrophages promote the transition to the proliferative phase of healing.

T-lymphocytes migrate into wounds following the inflammatory cells and macrophages, and peak during the late-proliferative/ early-remodeling phase. The role of T-lymphocytes is not completely understood and is a current area of intensive investigation. Several studies suggest that delayed T-cell infiltration along with decreased T-cell concentration in the wound site is associated with impaired wound healing, while others have reported that CD 4+ cells (T-helper cells) have a positive role in wound healing and CD8+ cells (T-suppressor-cytotoxic cells) play an inhibitory role in wound healing.[31]

Interestingly, recent studies in mice deficient in both T- and B-cells have shown that scar formation is diminished in the absence of lymphocytes.[32] In addition, skin gamma-delta T-cells regulate many aspects of wound healing, including maintaining tissue integrity, defending against pathogens, and regulating inflammation. These cells are also called dendritic epidermal T-cells (DETC), due to their unique dendritic morphology. DETC are activated by stressed, damaged, or transformed keratinocytes and produce fibroblast growth factor 7 (FGF-7), keratinocyte growth factors, and insulin-like growth factor-1, to support keratinocyte proliferation and cell survival. DETC also generate chemokines and cytokines that contribute to the initiation and regulation of the inflammatory response during wound healing. While cross-talk between skin gamma-delta T-cells and keratinocytes contributes to the maintenance of normal skin and wound healing, mice lacking or defective in skin gamma-delta T-cells show a delay in wound closure and a decrease in the proliferation of keratinocytes at the wound site.[33]

The proliferative phase generally follows and overlaps with the inflammatory phase, and is characterized by epithelial proliferation and migration over the provisional matrix within the wound (re-epithelialization). In the reparative dermis, fibroblasts and endothelial

cells are the most prominent cell types present and support capillary growth, collagen formation, and the formation of granulation tissue at the site of injury. Within the wound bed, fibroblasts produce collagen as well as glycosaminoglycans and proteoglycans, which are major components of the extracellular matrix (ECM). Following robust proliferation and ECM synthesis, wound healing enters the final remodeling phase, which can last for years.

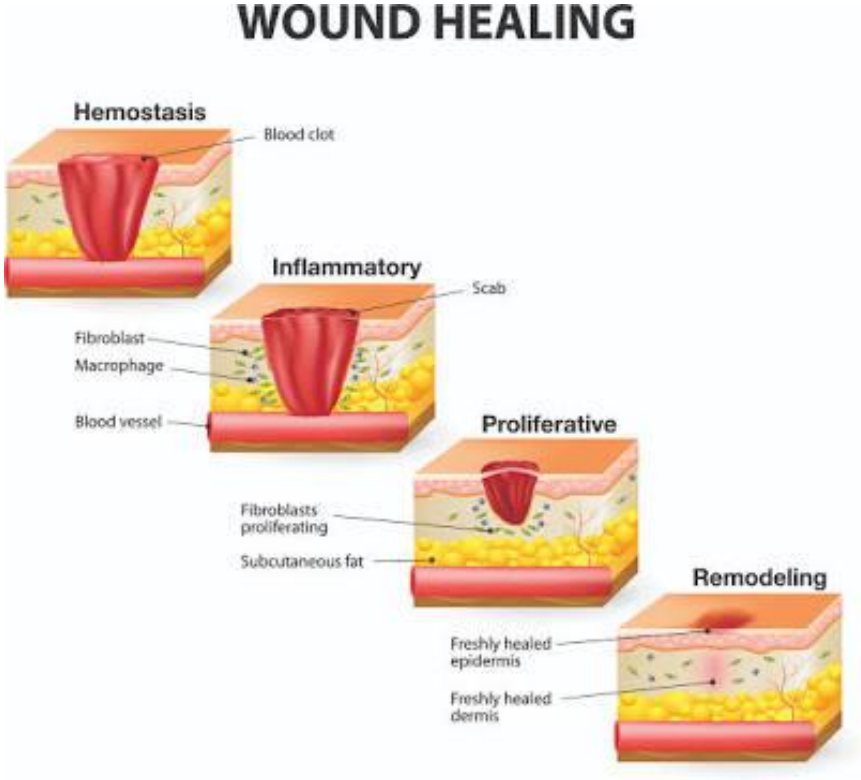
In this phase, regression of many of the newly formed capillaries occurs, so that vascular density of the wound returns to normal. One critical feature of the remodeling phase is ECM remodeling to an architecture that approaches that of the normal tissue. The wound also undergoes physical contraction throughout the entire wound healing process, which is believed to be mediated by contractile fibroblasts (myofibroblasts) that appear in the wound.[29]

The role of stem cells (SC) in cutaneous wound healing and tissue regeneration is a topic of increasing research attention, with a focus on the role of adult stem cells such as epidermal stem cells and bone-marrow (BM)-derived cells (BMDCs). Epidermal stem cells reside in the bulge area of hair follicles and in the basal layer of the epidermis and give rise

to the keratinocytes that migrate and re-epithelialize wounds. Normal skin is also a target organ for BMDCs. Two main stem cell populations are present in the bone marrow: hematopoietic SC (HSC) and mesenchymal SC (MSC).

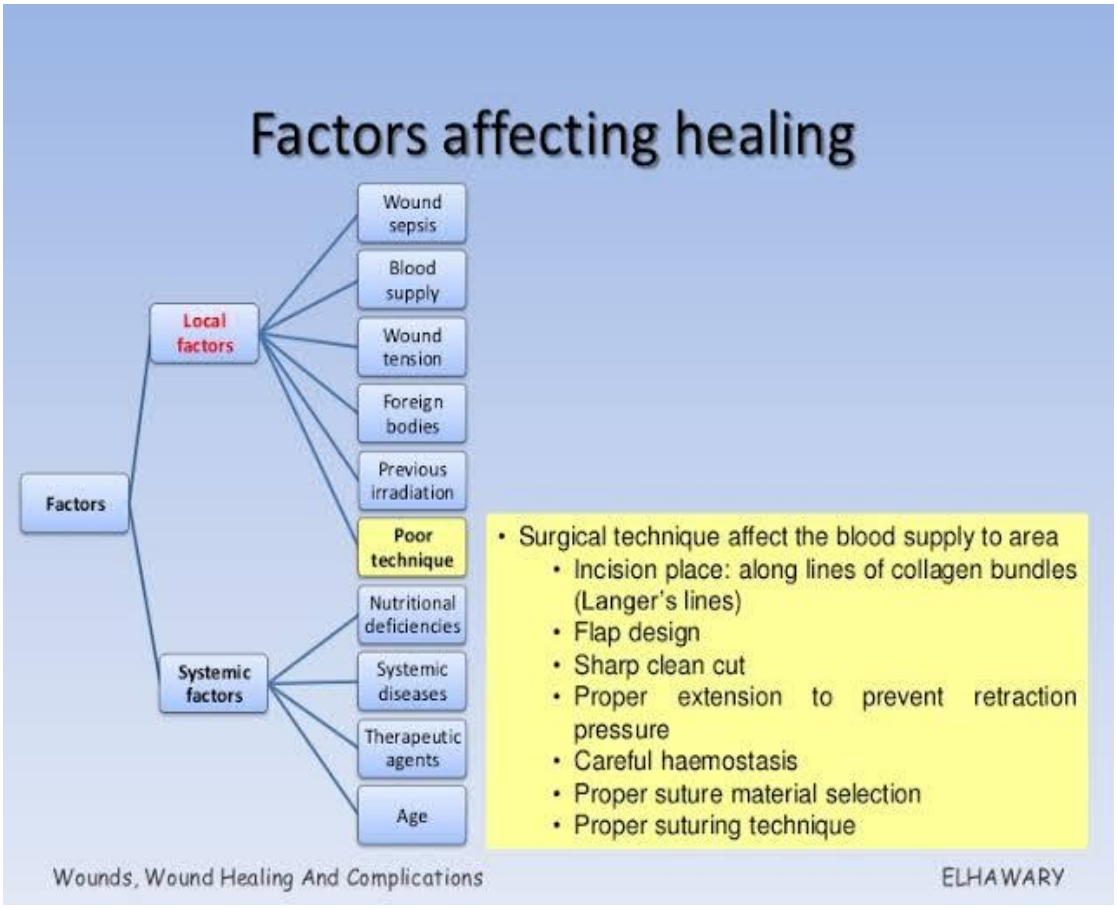
BM-MSCs are able to differentiate into a variety of cell types, including adipocytes, osteoblasts, chondrocytes, fibroblasts, and keratinocytes.[34] Endothelial progenitor cells (EPCs) derived from the HSC lineage are key cells that contribute to neovascularization. Both BM-MSCs and EPCs are involved in the cutaneous wound-healing process. Wound-induced hypoxia triggers the mobilization of bone marrow EPCs to the circulation, playing a significant role in the process of neovascularization.[35]

Several different cell types are involved in the wound healing process, and, as described above, the cellular activities of any particular cell type may also vary during different stages of repair. The complexity and coordination of the healing process are major hurdles to therapeutic approaches, since any therapeutic must effectively be sequenced to the appropriate stage.



FACTORS AFFECTING WOUND HEALING:

Multiple factors can lead to impaired wound healing. In general terms, the factors that influence repair can be categorized into local and systemic. Local factors are those that directly influence the characteristics of the wound itself, while systemic factors are the overall health or disease state of the individual that affect his or her ability to heal. Many of these factors are related, and the systemic factors act through the local effects affecting wound healing.



LOCAL FACTORS THAT INFLUENCE WOUND HEALING:

1. OXYGENATION:

Oxygen is important for cell metabolism, especially energy production by means of ATP, and is critical for nearly all wound healing processes. It prevents wounds from infection, induces angiogenesis, increases keratinocyte differentiation, migration, and re-epithelialization, enhances fibroblast proliferation and collagen synthesis, and promotes wound contraction.[36] In addition, the level of superoxide production (a key factor for oxidative killing

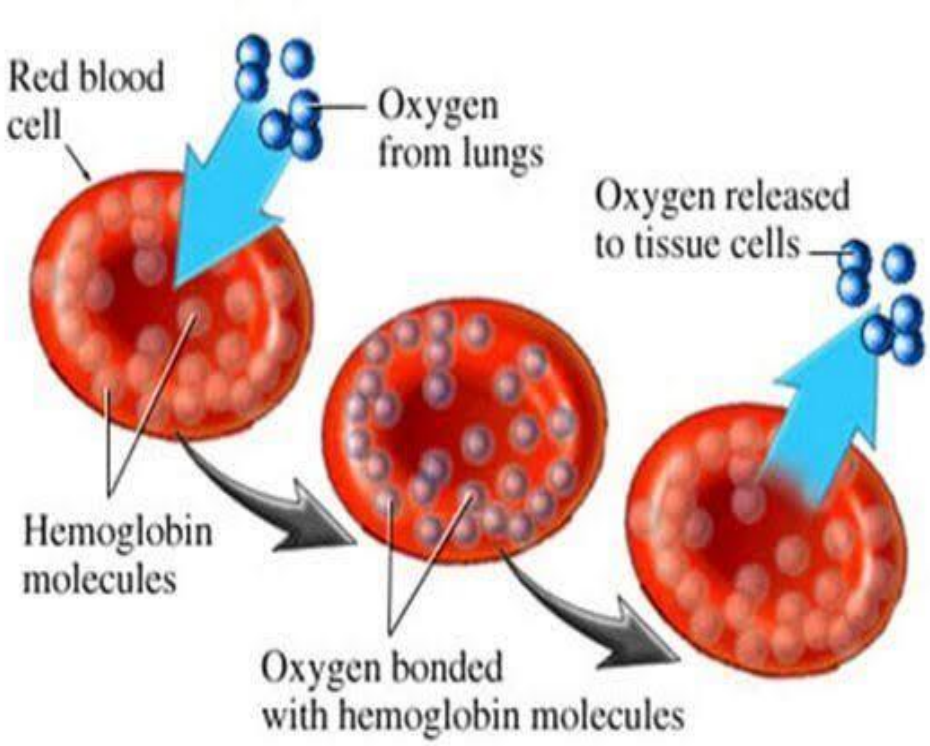
pathogens) by polymorphonuclear leukocytes is critically dependent on oxygen levels.

Due to vascular disruption and high oxygen consumption by metabolically active cells, the microenvironment of the early wound is depleted of oxygen and is quite hypoxic. Several systemic conditions, including advancing age and diabetes, can create impaired vascular flow, thus setting the stage for poor tissue oxygenation. In the context of healing, this overlay of poor perfusion creates a hypoxic wound. Chronic wounds are notably hypoxic; tissue oxygen tensions have been measured transcutaneously in chronic wounds from 5 to 20 mm Hg, in contrast to control tissue values of 30 to 50 mm Hg.[37]

In wounds where oxygenation is not restored, healing is impaired. Temporary hypoxia after injury triggers wound healing, but prolonged or chronic hypoxia delays wound healing.[36] In acute wounds, hypoxia serves as a signal that stimulates many aspects of the wound-healing process. Hypoxia can induce cytokine and growth factor production from macrophages, keratinocytes, and fibroblasts. Cytokines that are produced in response to hypoxia

include PDGF, TGF-β, VEGF, tumor necrosis factor-α (TNF-α), and endothelin-1, and are crucial promoters of cell proliferation, migration and chemotaxis, and angiogenesis in wound healing.[38]

In normally healing wounds, ROS such as hydrogen peroxide (H2O2) and superoxide (O2) are thought to act as cellular messengers to stimulate key processes associated with wound healing, including cell motility, cytokine action (including PDGF signal transduction), and angiogenesis. Both hypoxia and hyperoxia increase ROS production, but an increased level of ROS transcends the beneficial effect and causes additional tissue damage.[38]



In summary, the proper oxygen level is crucial for optimum wound healing. Hypoxia stimulates wound healing such as the release of growth factors and angiogenesis, while oxygen is needed to sustain the healing process.[36] One therapeutic option that can sometimes overcome the influence of tissue hypoxia is hyperbaric oxygen therapy (HBOT).[38] While HBOT can be an effective treatment for hypoxic wounds, its availability is limited.

Three of 4 clinical surveys of patients with abdominal wound disruption have failed to implicate anemia as a major factor. Marsh,[74] Mann,[75] Alexander[76] and their co- workers found that anemia, uncomplicated by other deficiencies, did not contribute significantly to wound dehiscence. Guiney and his colleagues,[77] by contrast, found that 50% of their patients who disrupted their abdominal wounds were anemic as opposed to 20% of the control group who healed normally. However, their criterion of anemia, hemoglobin of less than 12 gm/100 ml, was somewhat strict.

The results of experimental studies, without close analysis, appear conflicting. Besser and Ehrenhaftb[78] found no decrease in the bursting

strength of stomach wounds in dogs made anemic by bleeding and retransfusing pooled plasma. Trueblood and his co-workers[79] found no decrease in the bursting strength of colonic anastomoses in rats made anemic by iron deficiency or bleeding and retransfusion of plasma. Adamson' showed no difference in skin bursting strength with bleeding and retransfusion with plasma expander.

Results obtained by other investigators prove that nutritional deficiency incidental to the production of anemia accounts for some of the apparently conflicting results. Waterman, et al.[80] confirmed that anemia had no effect on either the contraction of open wounds or on the bursting strength of laparotomy incisions in young rats. They also showed that control as well as anemia rats fed powdered milk (a diet often used to produce iron-deficiency anemia) with and without added iron, showed slower contraction and reduced breaking strength when compared to animals fed normal chow.

The chowfed animals gained twice as much weight in the course of the experiment. Macon and Poriesl[81] also found that iron deficiency anemia had no effect on wound breaking strength and young rats fed on

regular chow gained both body weight and wound tensile strength more rapidly than animals which were fed powdered milk with or without iron. Nutritional depletion, therefore, can explain the findings of Jacobson and Van Prohaskal[82] whose chow-fed control mice showed a significantly higher breaking strength than the anemic group which was fed on an iron-free powdered milk diet. Bains, Crawford and Ketcham[83] reported decreased breaking strength in young rats (145-165 gram) made anemic by a combination of iron deficient diet and bleeding. However, iron deficiency in the young has a potent effect on growth rate and hence on repair.

Hugo and his colleagues[84] found a decrease in breaking strength in rabbits made anemic by hemolysis with intraperitoneal phenylhydrazine after 6 days, but there was no difference in strength at 9 or 12 days. The toxic potential of phenylhydrazine on wound metabolism is not known. Abnormalties of blood volume or viscosity, both of which severely impair healing, may also co-exist with anemia.

Sandblom[85] found decreased breaking strength in wounds of rabbits made anemic by bleeding. These animals were often dehydrated and hypovolemic. He later showed that dehydration can decrease wound

strength. Sandberg and Zederfeldt[86] repeated these experiments but replaced blood volume with dextran and restored healing towards normal. When they denervated the wounded area, healing became entirely normal. Hunt, et al. demonstrated that hypovolemia severely decreases oxygen supply to the wound,[87] and wound hypoxia delays repair.[88,89]

Most of these studies relied upon measurement of breaking strength as the index of repair. Collagen accounts for the strength of wounds, and therefore any factor which affects the strength of early wounds must do so by interfering with collagen biosynthesis, or by accelerating collagen lysis. Collagen synthesis requires oxygen for several of its steps. Assembly of the amino acid chains on the ribosome is energy dependent. Fibroblasts contain the enzymes for glycolysis, but most of their energy is ultimately derived from oxidative metabolism. Hydroxylation of some of the proline and lysine molecules, which is essential for structure and function of the collagen molecule, requires vitamin C, ferrous iron, a-ketoglutarate and molecular oxygen as essential cofactors for the hydroxylating enzyme.[88]

Under normal conditions the rate-limiting factor in collagen biosynthesis is the local availability of molecular oxygen. Niinikoski[90]

and Hunt and Pai[91] have shown that the rate of gain in strength and collagen content in experimental wounds was greater when the animals breathed 45% to 60% oxygen and was less in an atmosphere of 12% oxygen as compared to control animals breathing air.

Obviously, oxygen is vital to healing, but the accumulated evidence heavily favors the conclusion that uncomplicated or moderated anemia does not affect wound healing. Nevertheless, surgeons continue to believe and teach that it does. Presumably the reason for this is the fundamental belief that red cell deficiencies must inevitably decrease oxygen delivery to the wound. Apparently this belief will persist until it is shown conclusively that anemia does not decrease wound oxygen supply.

Hemoglobin cutoff is taken as 11g/dl and patients who have hemoglobin less than 11g/dl are considered to be anemic. Several studies have shown a significant association between anemia and development of complications. Hence our study aims to find an association between them so that in future intervention can be done.

INFECTION:

Once skin is injured, micro-organisms that are normally sequestered at the skin surface obtain access to the underlying tissues.

The state of infection and replication status of the microorganisms determines whether the wound is classified as having contamination, colonization, local infection/critical colonization, and/or spreading invasive infection.

Contamination is the presence of non-replicating organisms on a wound, while colonization is defined as the presence of replicating microorganisms on the wound without tissue damage. Local infection/ critical colonization is an intermediate stage, with microorganism replication and the beginning of local tissue responses. Invasive infection is defined as the presence of replicating organisms within a wound with subsequent host injury.[39]

Inflammation is a normal part of the wound-healing process, and is important to the removal of contaminating micro-organisms. In the absence of effective decontamination, however, inflammation may be prolonged, since microbial clearance is incomplete. Both bacteria and endotoxins can lead to the prolonged elevation of pro-inflammatory cytokines such as interleukin-1 (IL-1) and TNF-α and elongate the inflammatory phase. If this continues, the wound may enter a chronic state and fail to heal.

This prolonged inflammation also leads to an increased level of matrix metalloproteases (MMPs), a family of proteases that can degrade the ECM. In tandem with the increased protease content, a decreased level of the naturally occurring protease inhibitors occurs. This shift in protease balance can cause growth factors that appears in chronic wounds to be rapidly degraded.[39] Similar to other infective processes, the bacteria in infected wounds occur in the form of biofilms, which are complex communities of aggregated bacteria embedded in a self-secreted extracellular polysaccharide matrix.[39] Mature biofilms develop protected microenvironments and are more resistant to conventional antibiotic treatment. *Staphylococcus aureus* (*S. aureus*), *Pseudomonas aeruginosa* (*P. aeruginosa*), and β-hemolytic *streptococci* are common bacteria in infected and clinically non-infected wounds.[39]

P. aeruginosa and *Staphylococcus* appear to play an important role in bacterial infection in wounds. Many chronic ulcers probably do not heal because of the presence of biofilms containing *P. aeruginosa*, thus shielding the bacteria from the phagocytic activity of invading polymorphonuclear neutrophils (PMNs). This mechanism may explain the failure of antibiotics as a remedy for chronic wounds.[40]

SYSTEMIC FACTORS THAT INFLUENCE WOUND HEALING:

AGE:

The elderly population (people over 60 years of age) is growing faster than any other age group (World Health Organization [WHO, www.who.int/topics/ageing]), and increased age is a major risk factor for impaired wound healing. Many clinical and animal studies at the cellular and molecular level have examined age-related changes and delays in wound healing. It is commonly recognized that, in healthy older adults, the effect of aging causes a temporal delay in wound healing, but not an actual impairment in terms of the quality of healing.[29]

Delayed wound healing in the aged is associated with an altered inflammatory response, such as delayed T-cell infiltration into the wound area with alterations in chemokine production and reduced macrophage phagocytic capacity.[31] Delayed re-epithelialization, collagen synthesis, and angiogenesis have also been observed in aged mice as compared with young mice.[31] Overall, there are global differences in wound healing between young and aged individuals.

A review of the age-related changes in healing capacity demonstrates that every phase of healing undergoes characteristic age-related changes, including enhanced platelet aggregation, increased secretion of inflammatory mediators, delayed infiltration of macrophages and lymphocytes, impaired macrophage function, decreased secretion of growth factors, delayed re-epithelialization, delayed angiogenesis and collagen deposition, reduced collagen turnover and remodeling, and decreased wound strength.[29]

Several treatments to reduce the age-related impairment of healing have been studied. Interestingly, exercise has been reported to improve cutaneous wound healing in older adults as well as aged mice, and the improvement is associated with decreased levels of pro-inflammatory cytokines in the wound tissue. The improved healing response may be due to an exercise-induced anti-inflammatory response in the wound.[41]

NUTRITION:

For more than 100 years, nutrition has been recognized as a very important factor that affects wound healing. Most obvious is that malnutrition or specific nutrient deficiencies can have a profound impact on wound healing after trauma and surgery.

Patients with chronic or non-healing wounds and experiencing nutrition deficiency often require special nutrients. Energy, carbohydrate, protein, fat, vitamin, and mineral metabolism all can affect the healing process.[43]

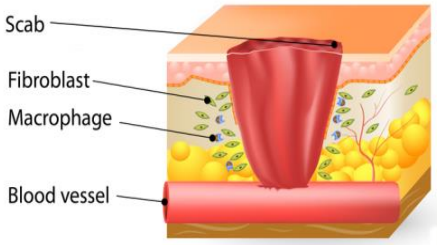
Carbohydrates, Protein, and Amino Acids

Together with fats, carbohydrates are the primary source of energy in the wound-healing process. Glucose is the major source of fuel used to create the cellular ATP that provides energy for angiogenesis and deposition of the new tissues.[43] The use of glucose as a source for ATP synthesis is essential in preventing the depletion of other amino acid and protein substrates.[43]

NUTRITIONAL SUPPORT FOR WOUND HEALING

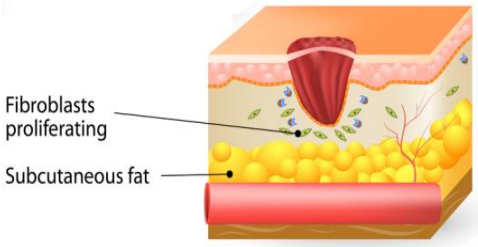
INFLAMMATORY PHASE

VITAMIN A | 25000IU per day
Enhances early immune response.
BROMELAIN | 500-1000mg per day
Prevents prolonged inflammatory phase.
PROTEIN | At least 0.8g/kg of body weight
Prevents prolonging inflammatory phase.
VITAMIN C | 1-2g per day
Optimizes immune response.



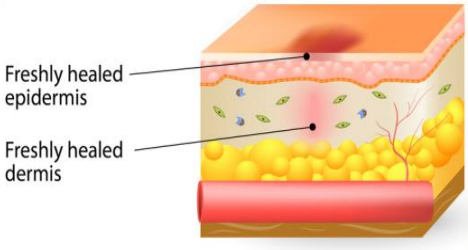
PROLIFERATIVE PHASE

VITAMIN C | 1-2g per day
Necessary for collagen synthesis.
GLUCOSAMINE | 1500mg per day
Enhances hyaluronic acid production.
VITAMIN A | 25000IU per day
Supports epithelial cell differentiation.
ZINC | 15-30mg per day
Helps cells proliferate and protein synthesis.



REMODELING PHASE

PROTEIN | At least 0.8g/kg of body weight
Inadequate protein intake can prolong inflammation and increase susceptibility to infection.



Protein is one of the most important nutrient factors affecting wound healing. A deficiency of protein can impair capillary formation, fibroblast proliferation, proteoglycan synthesis, collagen synthesis, and wound remodeling. A deficiency of protein also affects the immune system, with resultant decreased leukocyte phagocytosis and increased susceptibility to infection.[44]

Collagen is the major protein component of connective tissue and is composed primarily of glycine, proline, and hydroxyproline. Collagen synthesis requires hydroxylation of lysine and proline, and co-factors such as ferrous iron and vitamin C. Impaired wound healing results from deficiencies in any of these co-factors.[45]

The adverse effects of malnutrition on the morbidity and mortality of patients was first recognised by Hippocrates many centuries ago. Nutrition plays a vital role in the care of patients on a surgical service. Between 30% and 50% of hospitalized patients are malnourished and malnutrition is clearly associated with increased morbidity and mortality. In the presence of malnutrition, surgical wounds and anastomoses are less likely to heal, resulting in an increased risk of wound complication and anastomotic dehiscence [52].

Nutritional assessment is essential for identifying patients who are at risk of developing complications. Although a variety of nutritional indices have been found to be valuable in predicting patient outcome when used alone, there is no consensus on the best method for assessing the nutritional status. The serum albumin level is the most readily available and clinically useful parameter. A serum albumin level greater than 3.5 g/dl suggests adequate protein stores and it confers a protective effect through several biological mechanisms.

Serum albumin is a better prognostic indicator than anthropometric markers of nutritional status because its ability to detect protein-energy malnutrition, which is not necessarily accompanied by lower body weight and may not be clinically recognizable, but is associated with significantly increased morbidity and mortality [53]. Protein energy malnutrition results from increased protein or energy requirements associated with the stress of illness, injury or infections. If the increased needs are not met from dietary or therapeutic sources, visceral protein stores are depleted, leading to abnormal function in organ systems, including gastrointestinal malabsorption, impaired immunologic response, impaired production of albumin and other proteins in the liver [54].

Despite compelling evidence that malnutrition increases postoperative morbidity and mortality after major elective surgery, preoperative nutrition is often completely ignored, and postoperative nutrition is not instituted until after the onset of complications. In comparison with trauma patients whose risks of complications can be quantified by the Abdominal Trauma Index (ATI) or Injury Severity Score (ISS),[55] simple scoring system for general surgery populations have not been adopted.

This problem of inadequate attention to nutritional status is compounded by the apparently contradictory clinical data concerning the value of nutrition support in general surgical patients. For example, 2 studies[56,57] noted more risk than benefit when parenteral feeding was provided perioperatively to borderline or mildly malnourished patients. Similarly, whereas 2 studies[58,59] of patients undergoing gastrectomy pancreatectomy, or esophagectomy showed benefits with a specialty enteral diet compared with a standard diet, another study[60] showed no difference between patients given that same specialty diet and unfed patients. Clinicians are often left unconvinced of the benefits of both preoperative or postoperative nutrition and institute therapy only after complications have developed.

Both clinical and laboratory data have been used to determine nutritional status and correlated with outcome. Depressed total lymphocyte count, protein depletion, low serum albumin or transferrin, and a history of significant preoperative weight loss are associated with increased postoperative complications.[61–72] Body mass index, anthropometrics, and percent body weight loss can be used to evaluate muscle protein and fat stores.[73]

For our study, serum albumin level cutoff is placed at 3.5g/dl and serum albumin level below 3.5g/dl is considered as hypoalbuminemia. Based on the above criteria, patients are evaluated to know whether complications are more in hypoalbuminemic patients. Several studies have shown significant association between serum albumin and the development of complications.

Arginine is a semi-essential amino acid that is required during periods of maximal growth, severe stress, and injury. Arginine has many effects in the body, including modulation of immune function, wound healing, hormone secretion, vascular tone, and endothelial function. Arginine is also a precursor to proline, and, as such, sufficient arginine levels are needed to support collagen deposition, angiogenesis, and

wound contraction.[42] Arginine improves immune function, and stimulates wound healing in healthy and ill individuals.[46] Under psychological stress situations, the metabolic demand of arginine increases, and its supplementation has been shown to be an effective adjuvant therapy in wound healing.[45]

Glutamine is the most abundant amino acid in plasma and is a major source of metabolic energy for rapidly proliferating cells such as fibroblasts, lymphocytes, epithelial cells, and macrophages.[43] The serum concentration of glutamine is reduced after major surgery, trauma, and sepsis, and supplementation of this amino acid improves nitrogen balance and diminishes immunosuppression.[45]

Glutamine has a crucial role in stimulating the inflammatory immune response occurring early in wound healing.[43] Oral glutamine supplementation has been shown to improve wound breaking strength and to increase levels of mature collagen.[47]

Fatty Acids

Lipids are used as nutritional support for surgical or critically ill patients to help meet energy demands and provide essential building blocks for wound healing and tissue repair. Polyunsaturated fatty acids (PUFAs), which cannot be synthesized *de novo* by mammals, consist mainly of two families, n-6 (omega-6, found in soybean oil) and n-3 (omega-3, found in fish oil). Fish oil has been widely touted for the health benefits of omega-3 fatty acids such as eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA).

The effects of omega-3 fatty acids on wound healing are not conclusive. They have been reported to affect pro-inflammatory cytokine production, cell metabolism, gene expression, and angiogenesis in wound sites.[48] The true benefit of omega-3 fatty acids may be in their ability to improve the systemic immune function of the host, thus reducing infectious complications and improving survival.[43]

Vitamins, Micronutrients, and Trace Elements

Vitamins C (L-ascorbic acid), A (retinol), and E (tocopherol) show potent anti-oxidant and anti-inflammatory effects. Vitamin C has many roles in wound healing, and a deficiency in this vitamin has multiple

effects on tissue repair. Vitamin C deficiencies result in impaired healing, and have been linked to decreased collagen synthesis and fibroblast proliferation, decreased angiogenesis, and increased capillary fragility. Also, vitamin C deficiency leads to an impaired immune response and increased susceptibility to wound infection.[43]

Similarly, vitamin A deficiency leads to impaired wound healing. The biological properties of vitamin A include anti-oxidant activity, increased fibroblast proliferation, modulation of cellular differentiation and proliferation, increased collagen and hyaluronate synthesis, and decreased MMP mediated extracellular matrix degradation.[49]

Vitamin E, an anti-oxidant, maintains and stabilizes cellular membrane integrity by providing protection against destruction by oxidation. Vitamin E also has anti-inflammatory properties and has been suggested to have a role in decreasing excess scar formation in chronic wounds. Animal experiments have indicated that vitamin E supplementation is beneficial to wound healing[43], and topical vitamin E has been widely promoted as an anti-scarring agent. However, clinical studies have not yet proved a role for topical vitamin E treatment in improving healing outcomes.[50]

Several micronutrients have been shown to be important for optimal repair. Magnesium functions as a co-factor for many enzymes involved in protein and collagen synthesis, while copper is a required co-factor for cytochrome oxidase, for cytosolic anti-oxidant superoxide dismutase, and for the optimal cross-linking of collagen. Zinc is a co-factor for both RNA and DNA polymerase, and a zinc deficiency causes a significant impairment in wound healing. Iron is required for the hydroxylation of proline and lysine, and, as a result, severe iron deficiency can result in impaired collagen production.[42]

As indicated above, the nutritional needs of the wound are complex, suggesting that composite nutrition support would benefit both acute and chronic wound healing. A recent clinical research study examined the effects of a high-energy, protein-enriched supplement containing arginine, vitamin C, vitamin E, and zinc on chronic pressure ulcers and indicated that this high-energy and nutrition-enriched supplement improved overall healing of the pressure ulcer.[51]

In summary, proteins, carbohydrates, arginine, glutamine, polyunsaturated fatty acids, vitamin A, vitamin C, vitamin E, magnesium, copper, zinc, and iron play a significant role in wound healing, and their deficiencies affect wound healing. Additional studies will be needed to fully understand how nutrition affects the healing response.

METHODOLOGY

Title	Predictors and evaluation of surgical wound complications in elective abdominal surgeries
Aims and Objectives	To Analyze various factors that may predict the surgical wound complication following elective abdominal surgeries and to evaluate the occurrence of various surgical wound complications.
Study Centre	Institute of General Surgery, Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai
Duration of Study	January 2018 to May 2019
Study Design	Observational study (Prospective)
Sample Size	120
Inclusion Criteria	1. Patients undergoing elective abdominal surgeries in the age group of 15 to 60 years of age.
Exclusion Criteria	Age: <15 years. - >60years. Patients undergoing emergency abdominal surgery. Patients affected with HIV, Hepatitis–B, Hepatitis–C, Diabetes mellitus. Patient presenting with pre-existing skin infections were excluded.

Antibiotic selection	Third generation Cephalosporins and Metronidazole were used for all the patients and were changed to CDC guidelines.
Pre-operative preparation	Shaving was one on previous day. All patients were advised to take shower on the day of surgery with soap. All patients received Inj. Ceftriaxone 1 gram and Inj. Metronidazole 500mg IV one hour before the incision. CDC guidelines were followed if changes were required. Aseptic precautions in the operation theatre All routine aseptic precautions were taken like using autoclaved gowns, drapes, sterile gloves and instruments. Standard surgical scrub followed by the protocol adopted by our Hospital was followed before performing the operation.
Operative protocol	The operative area was cleaned with spirit and painted with 5% povidone iodine. The principles of surgery were followed in all cases such as minimum tissue handling and maintenance of adequate hemostasis. Drains were used whenever necessary. Skin closure with suture material or skin staples was done.
Post operative care	Injectable Cephalosporins (3rd generation) and Metronidazole were continued in the post-operative period for 48 hours. Then the patient received oral antibiotics till stitch removal. For the patients with surgical site infection, the plan of antibiotic coverage changed according to culture and sensitivity report. The wound was inspected for any evidence of infection starting from 48 hours after surgery day till 8th post-operative day. Patients were followed up till discharge.

Ethics Clearance	Applied
Methodology	• For the study, demographic details of patients, results of investigations and details of surgical procedures were documented. • Patients were followed up during postoperative period and at monthly interval for 6 months. Occurrence of complications like wound discharge, sinus, fistula, wound dehiscence and incisional hernia was noted. • If pus was present, it is subjected to culture and sensitivity. • All the above collected data will be analyzed and conclusions will be derived through statistical analysis using Mann-Whitney U test for continuous variables and Chi-square test for categoric variables. Patients will be called up one month after discharge and follow up will be done.
Sponsorship(Yes/ No) If Yes details	No
Conflict of Interest	No

SAMPLE SIZE CALCULATION:

Based on the study titled "Evaluation of wound complications in elective abdominal surgery[17] by Sharma A.C., Singla Mamta, Shuaib Mohammad, Kumar Spandan, the sample size was calculated.

$$n = \frac{Z^2 pq}{d^2}$$

(where N is required sample size, Z is reliability coefficient at 95% confidence interval, p is proportion of population with characteristics of interest is 23% [22] , d is the absolute error and ε is margin of error at minimum sample size)

When Z = 1.96 for α 0.05; P= 23; q = 77, d is fixed as 8%

$$n = \frac{1.96 \times 1.96 \times 22.4 \times 77.59}{8 \times 8} = 104$$

Assuming a non-response rate of 10%, sample size is fixed at 104+11=115. Hence sample size is fixed at 120.

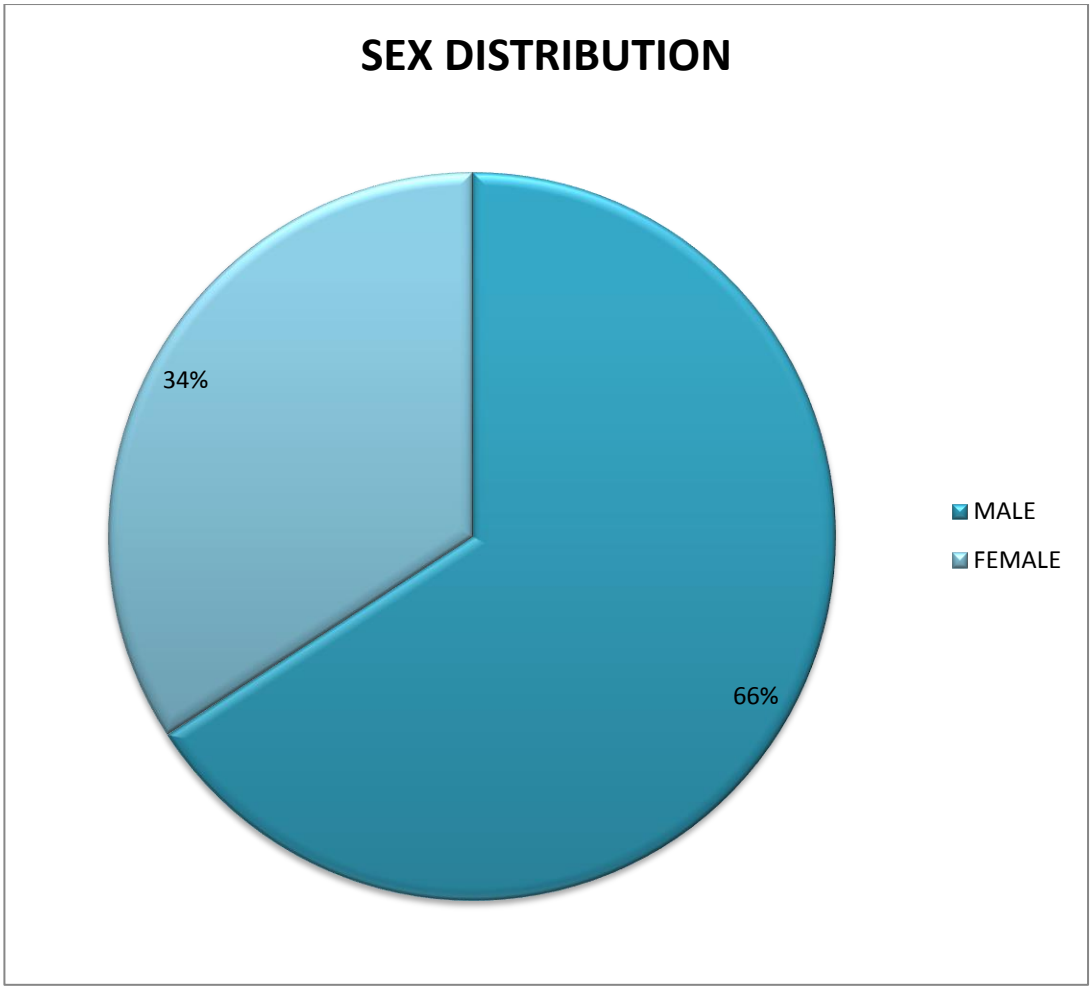
The collected data were analysed with IBM.SPSS statistics software 23.0 Version. To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables.

RESULTS

The study included 120 patients who were selected with inclusion and exclusion criteria. All patients were followed up and the following results were obtained.

AGE AND SEX DISTRIBUTION:

Among the 120 patients, 79(34%) were males and 41(66%) were females. 4(3.33%) were between10 and 20 years, 10(8.33%) were between 20 and 30 years, 34(28.33%) were between 30 and 40 years, about 27(22.5%) were between 40 and 50 years and only 45(37.5%) were between 50 and 60 years. Thus most of the patients in my study were between 50 to 60 years.

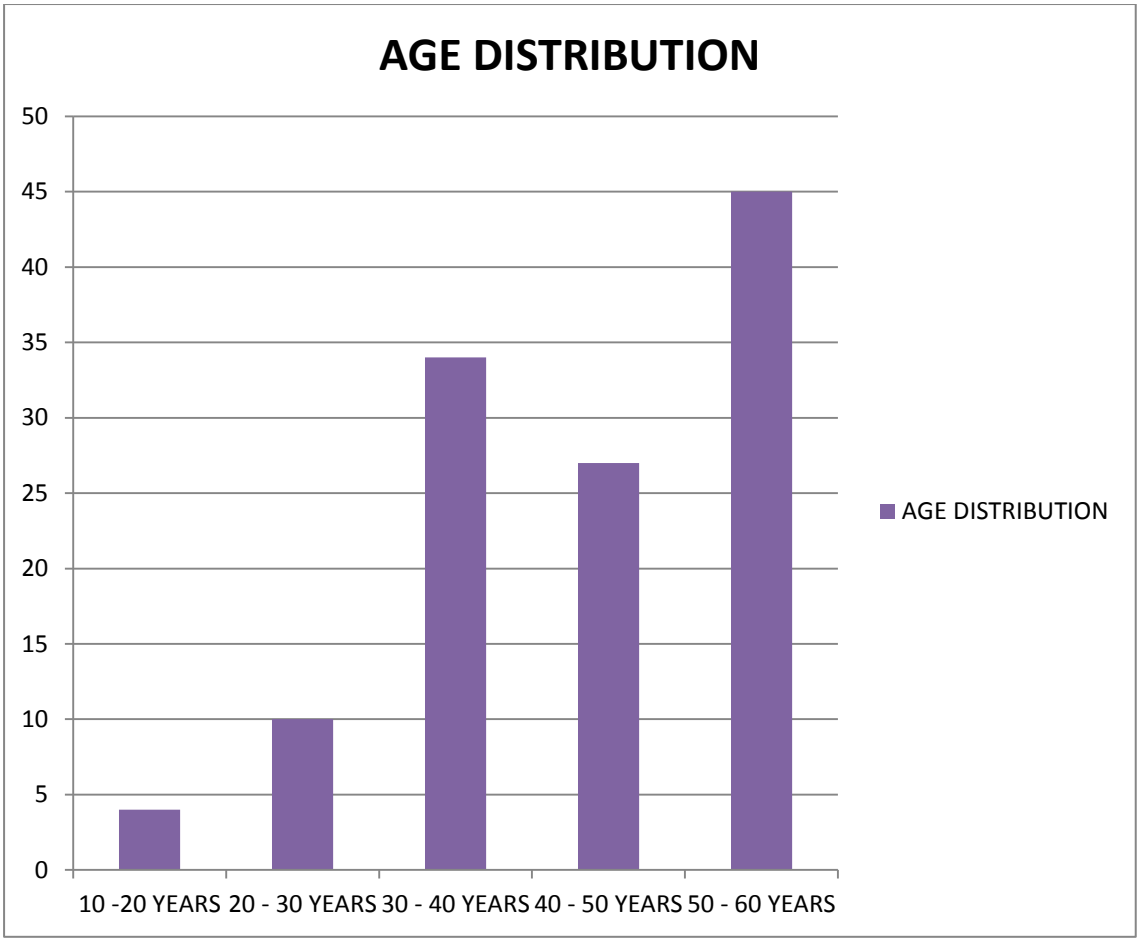


GRAPH 1: SEX DISTRIBUTION

TABLE NO. 1

AGE DISTRIBUTION

AGE	Frequency	Percent
10 - 20 yrs.	4	3.33
21 - 30 yrs.	10	8.33
31 - 40 yrs.	34	28.33
41 - 50 yrs.	27	22.50
51 - 60 yrs.	45	37.50
Total	120	100.0



GRAPH 2: AGE DISTRIBUTION

Mean age of the patients in my study is 44.49 years of age.

PROCEDURE:

S.NO.	PROCEDURE	FREQUENCY	PERCENTAGE
1	HERNIOPLASTY	47	39.2%
2	MESH REPAIR	27	22.5%
3	LAPAROTOMY	16	13.3%
4	APPENDICECTOMY	17	14.1%
5	CHOLECYSTECTOMY	12	10%
6	CBD EXPLORATION	1	0.8%

TABLE 2: TYPE OF SURGICAL PROCEDURES

Thus majority of the procedure is hernioplasty which constitute 39.2%.

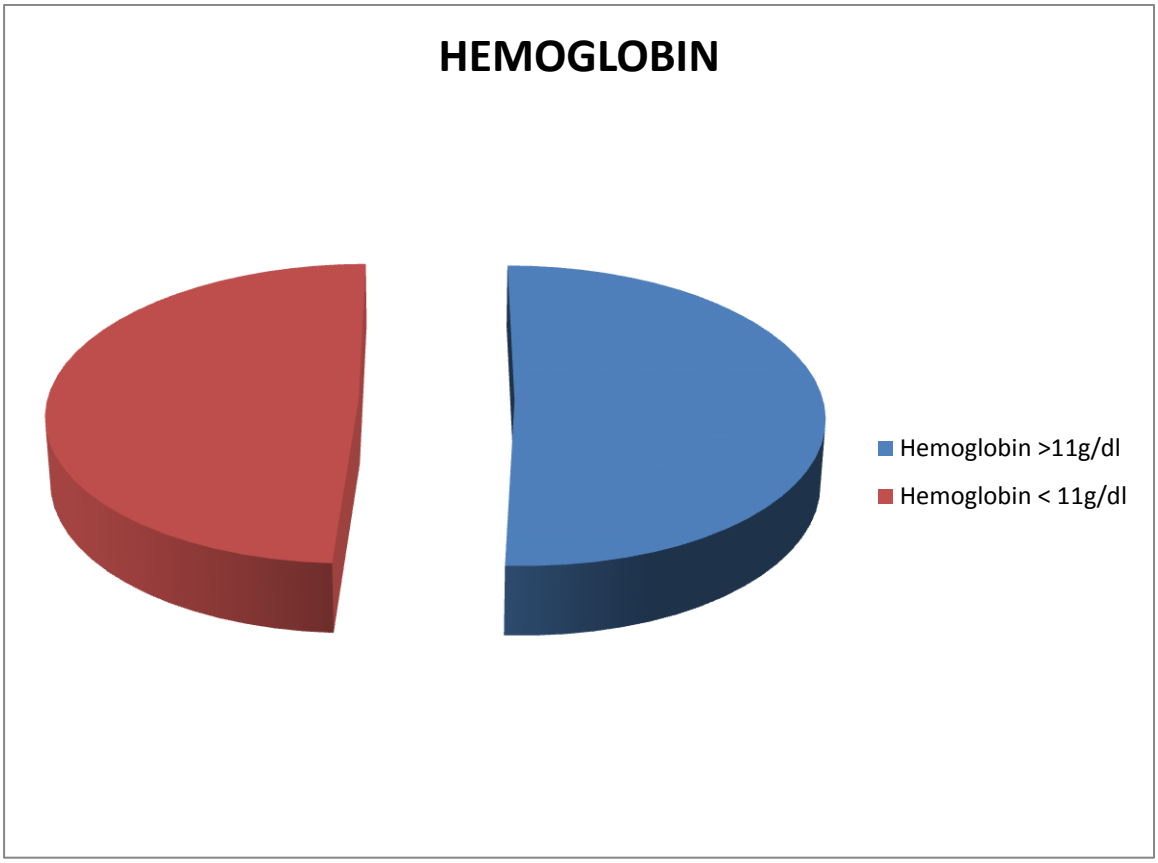
HEMOGLOBIN:

Out of 120 patients in the study, 59 had hemoglobin less than 11g/dl which contributes to 49.16%. Hemoglobin cutoff of 11g/dl was taken according to WHO classification and hence 49.16% were anemic.

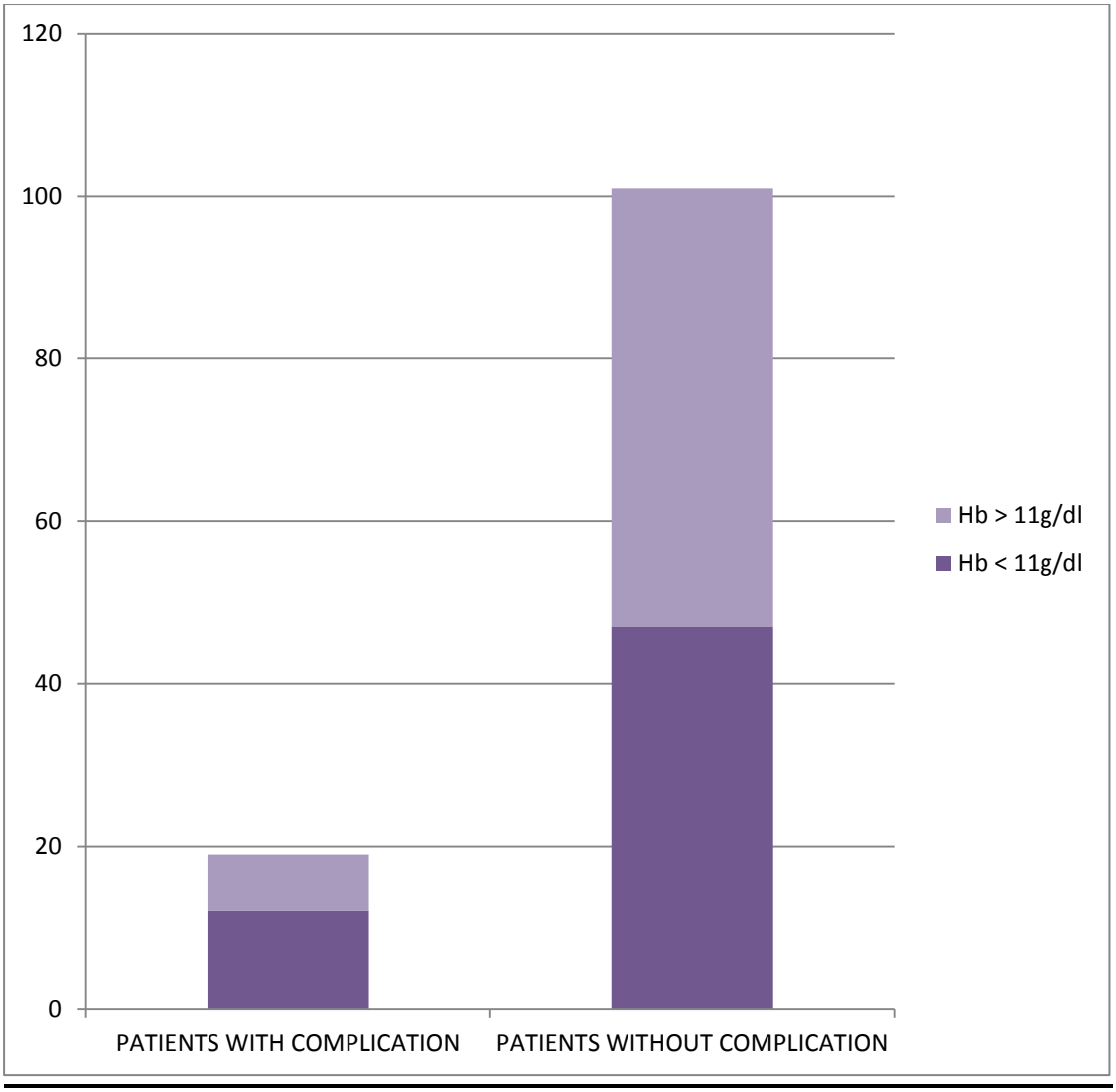
Among the patients who had wound complications following surgery, 63% were anemic. Thus anemic patient are 1.36 times more likely to develop complication following surgery than the patient with normal hemoglobin levels.

TABLE 3: DESCRIPTIVE ANALYSIS OF HEMOGLOBIN

	HEMOGLOBIN
MEAN	10.919
STANDARD DEVIATION	0.5662
MINIMUM	9.0
MAXIMUM	13.0



GRAPH 3: HEMOGLOBIN



GRAPH 4: ANEMIA IN PATIENTS WITH AND WITHOUT COMPLICATIONS

MEAN CORPUSCULAR VOLUME:

TABLE 4: DESCRIPTIVE ANALYSIS OF MCV

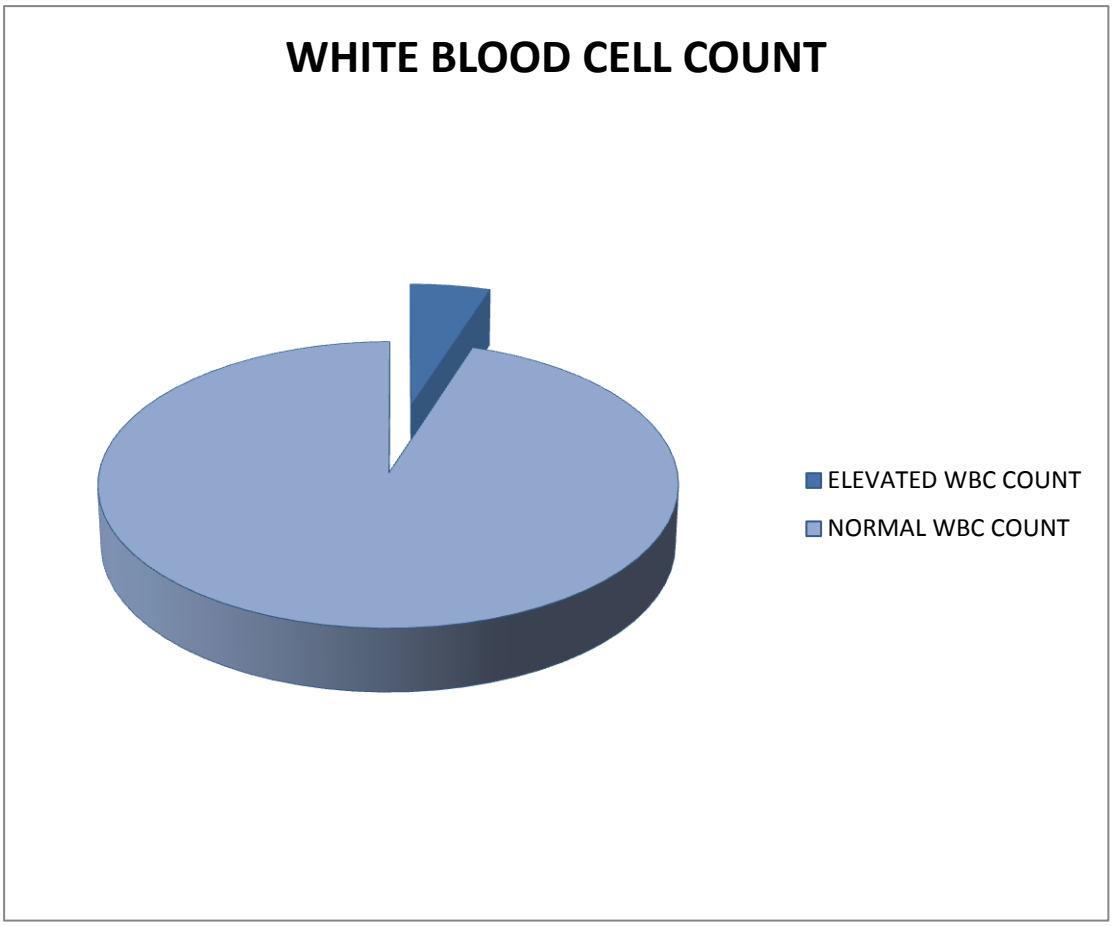
	MCV
MEAN	88.16
STANDARD DEVIATION	3.119
MINIMUM	78
MAXIMUM	95

MCV was found to be a poor predictor of wound complication as there was no significant variation between the patients with complications and without complication.

WHITE BLOOD CELL COUNT:

Cutoff of 10,000/mm^3 was used and count more than 10,000/mm^3 was taken as elevated WBC count Out of 120 patients, 6(5%) patients have elevated white blood cell count where remaining 114(95%) have normal white blood cell count.

GRAPH 5: WHITE BLOOD CELL COUNT



In patients who had complications following surgery the preoperative WBC count was elevated in 15.8%. Thus patients who have elevated WBC count are 5.26 times more likely to end up in complication than the patients with normal count.

ALBUMIN:

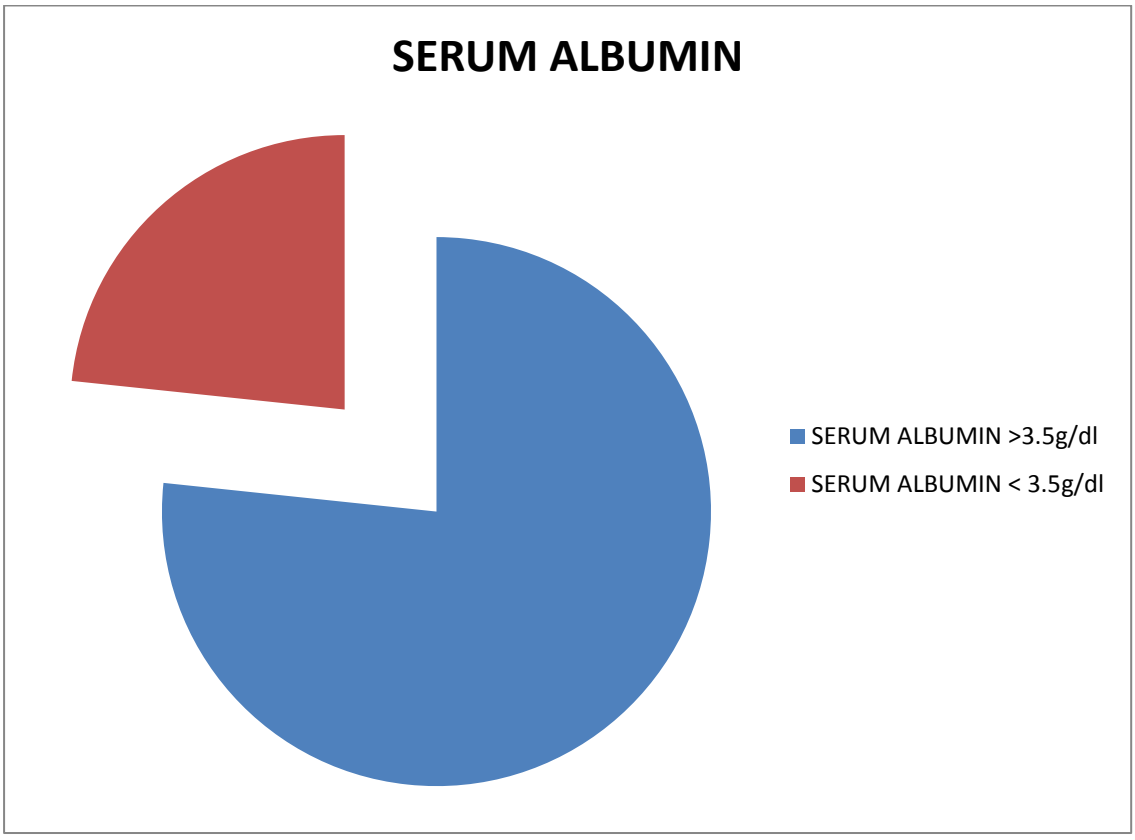
Serum albumin level of 3.5g/dl was taken as the cutoff. In this study of 120 patients, 28(23.33%) have hypoalbuminemia and the rest have normal preoperative serum albumin levels.

Hypoalbuminemia in patients with complication is 52.8% and in patients without complication is 13.8%.

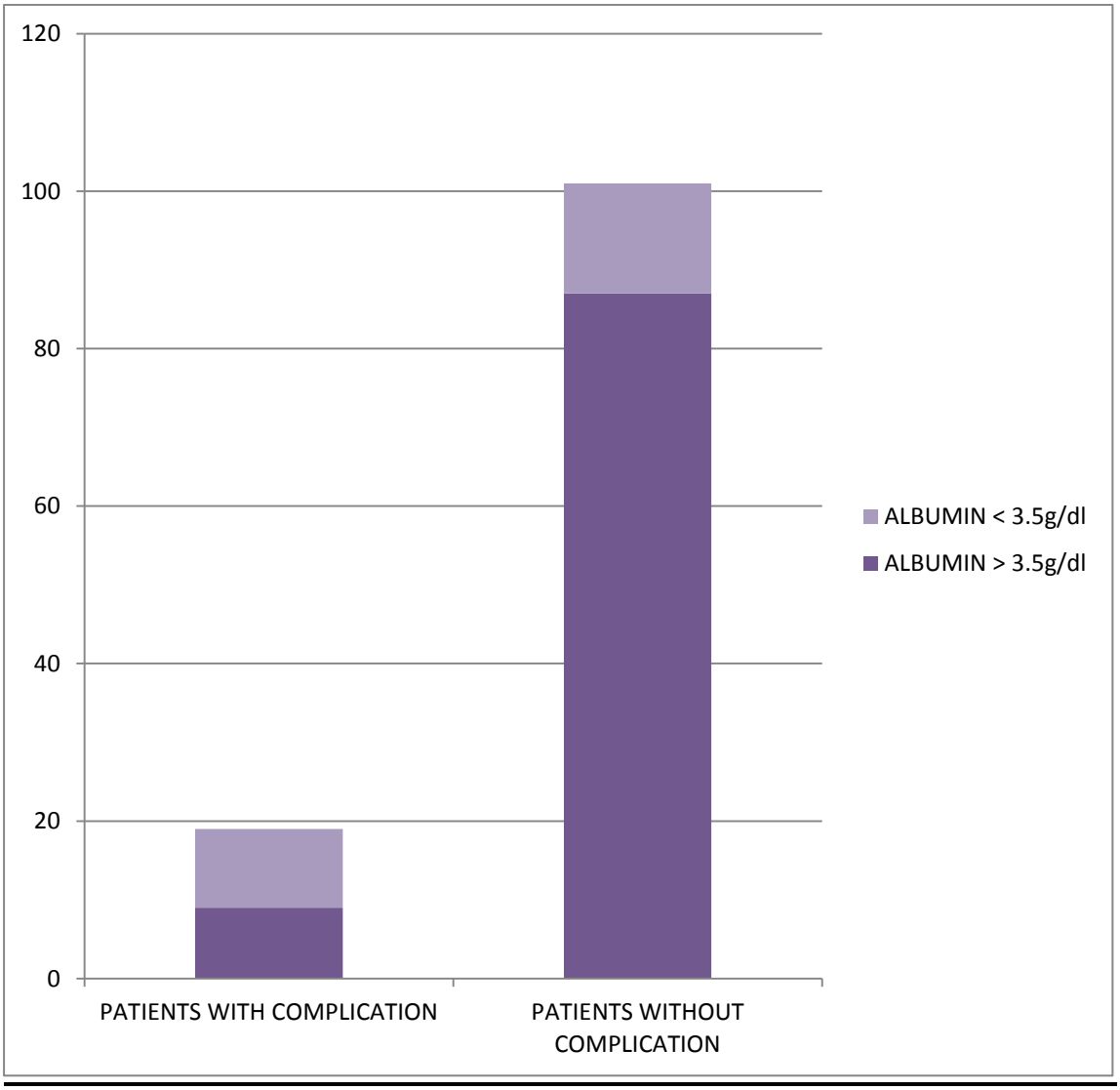
Thus patients with hypoalbuminemia are 3.81 times more likely to end up with complication than patients who had normal serum albumin levels preoperatively.

TABLE 5: DESCRIPTIVE ANALYSIS OF SERUM ALBUMIN

	ALBUMIN
MEAN	3.66
STANDARD DEVIATION	0.372
MINIMUM	2.4
MAXIMUM	4.7



GRAPH 6: SERUM ALBUMIN



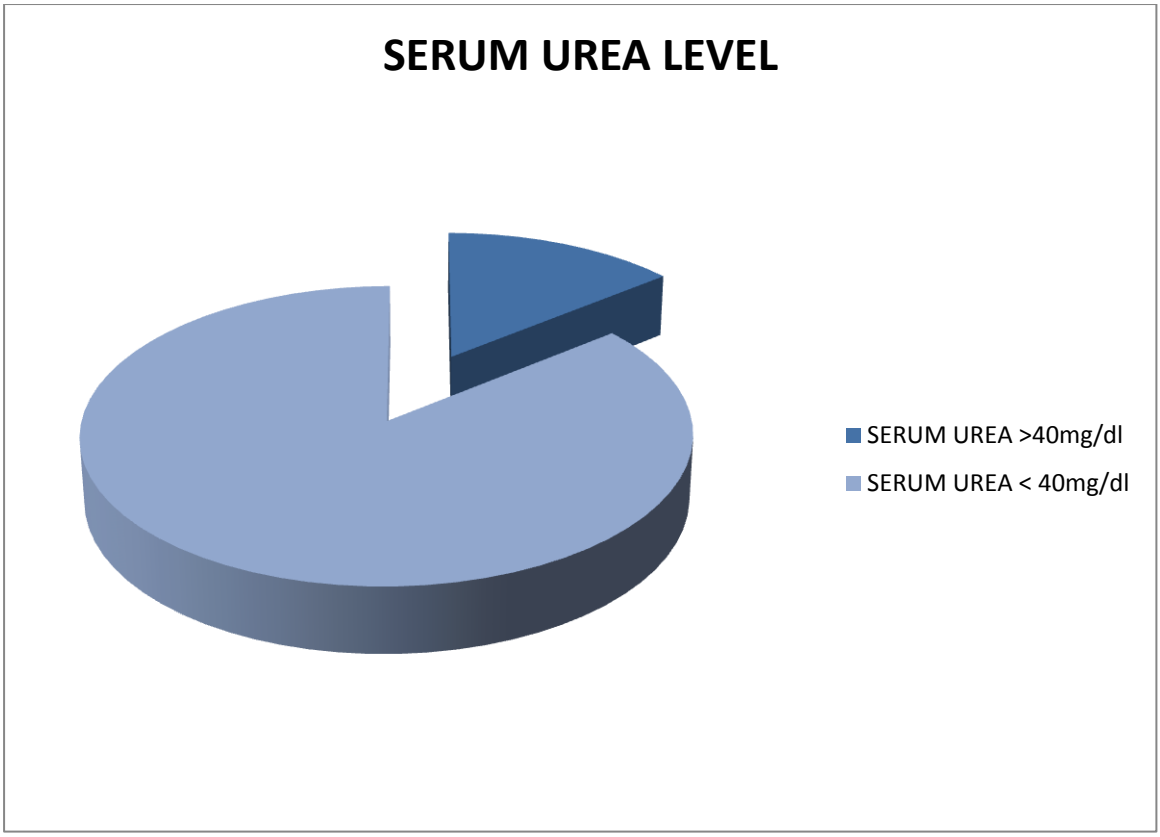
GRAPH 7: HYPOALBUMINEMIA IN PATIENTS WITH AND WITHOUT COMPLICATIONS

UREA:

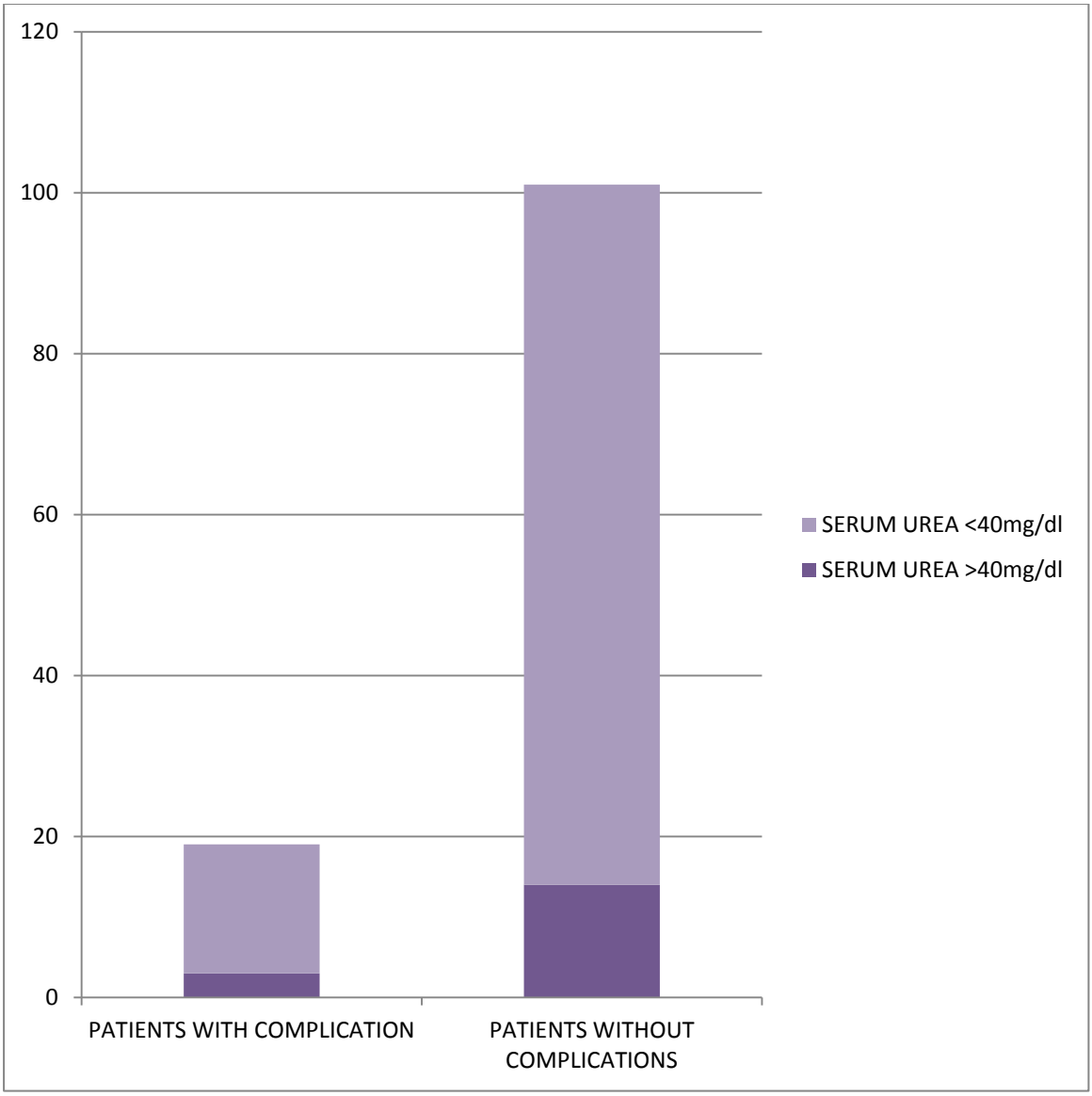
Serum urea level of 40mg/dl is taken as the cutoff. In this study, 17(14.16%) patients had elevated preoperative serum urea level. In patients with complication, urea was elevated in 15.8% cases and in patients without any complication, the urea was elevated in 13.86%. There was no significant impact of preoperative serum urea level predicting the wound complication following surgery.

TABLE 6: DESCRIPTIVE ANALYSIS OF SERUM UREA

	UREA
MEAN	35.74
STANDARD DEVIATION	8.54
MINIMUM	18
MAXIMUM	63



GRAPH 8: SERUM UREA LEVEL



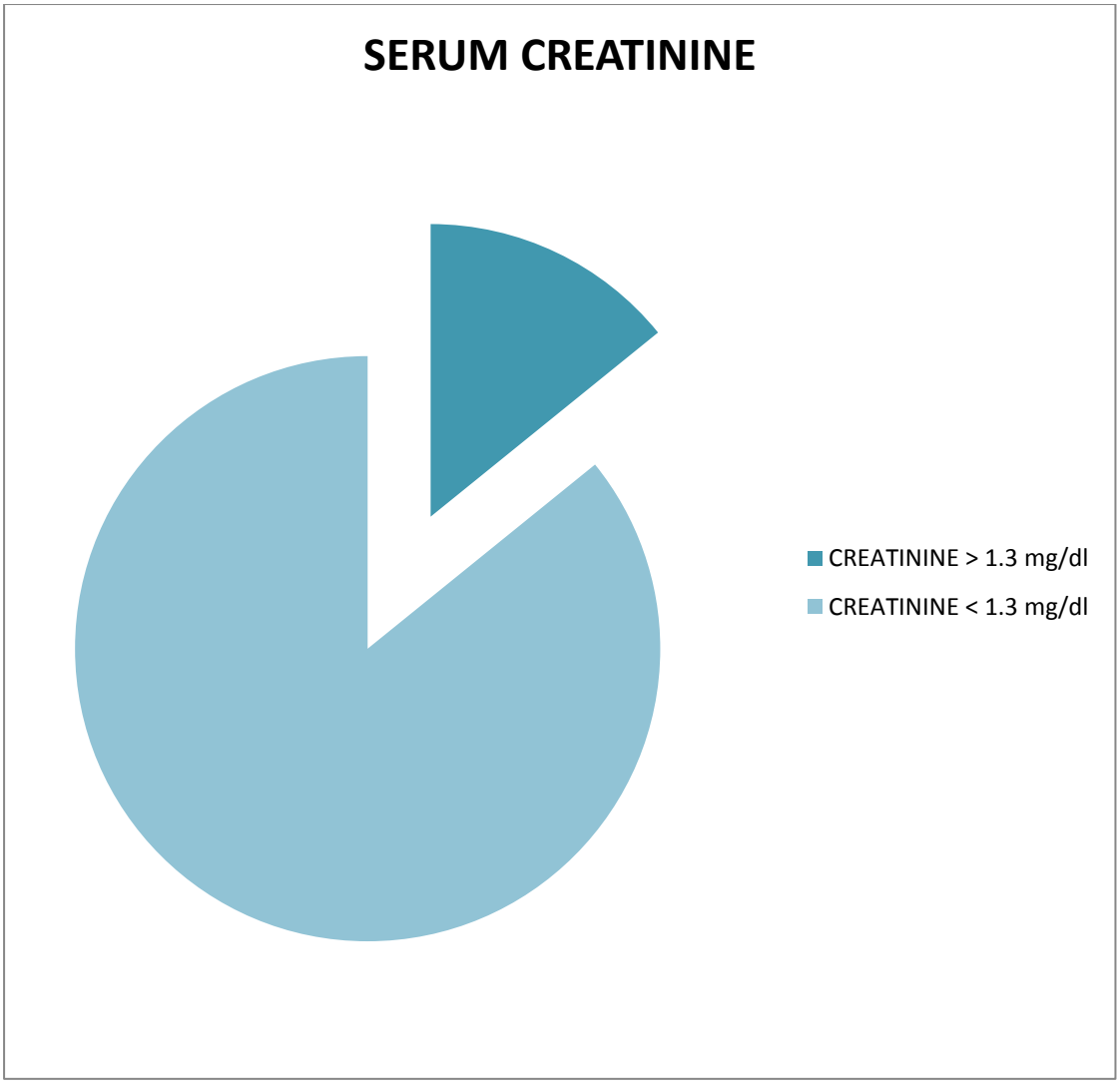
GRAPH 9: ELEVATED SERUM UREA IN PATIENTS WITH AND WITHOUT COMPLICATION

SERUM CREATININE:

In this study, out of 120 patients, 17(14.16%) had elevated serum creatinine levels and the cutoff was 1.3mg/dl. 3(15.8%) of patients with complication had elevated serum creatinine preoperatively and 14(17.82%) of patients without complication had elevated serum creatinine preoperatively. There was no significant impact of serum creatinine in predicting the wound complication following surgery.

TABLE 7: DESCRIPTIVE ANALYSIS OF SERUM CREATININE

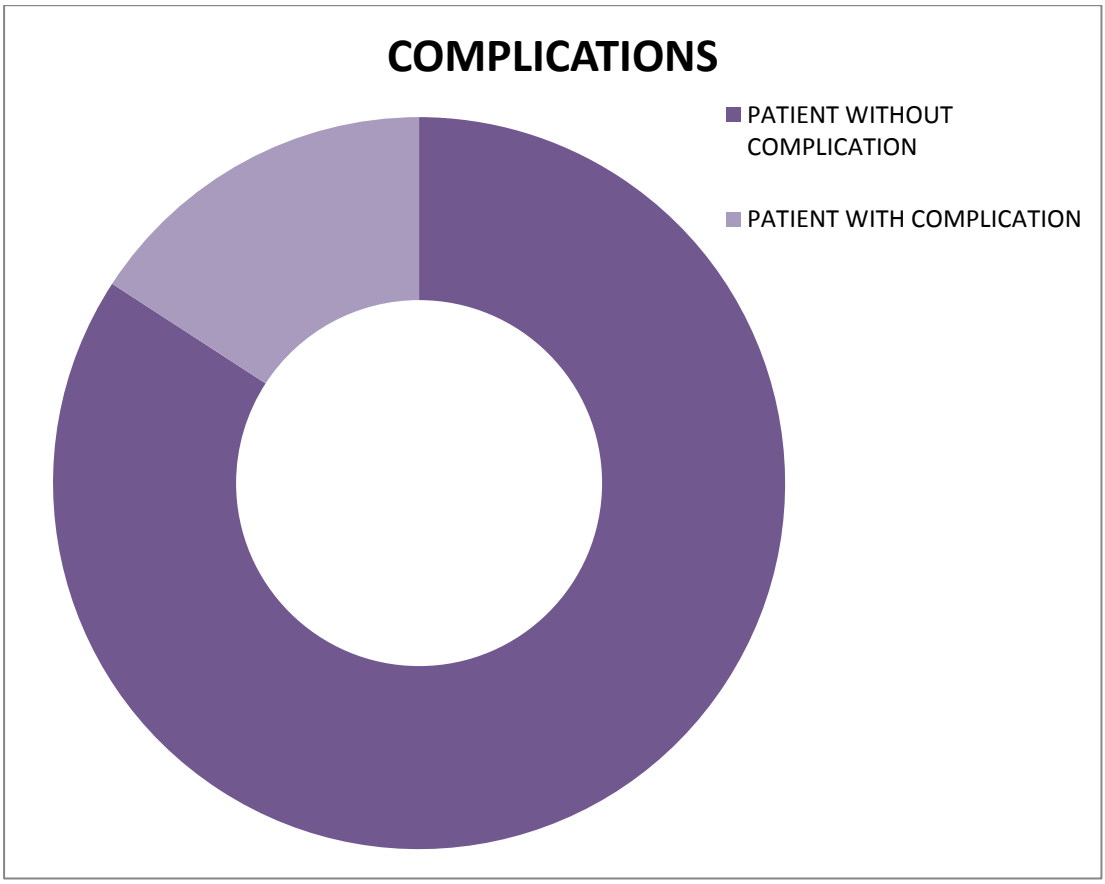
	CREATININE
MEAN	1.065
STANDARD DEVIATION	0.294
MINIMUM	0.4
MAXIMUM	1.6



GRAPH 10: SERUM CREATININE

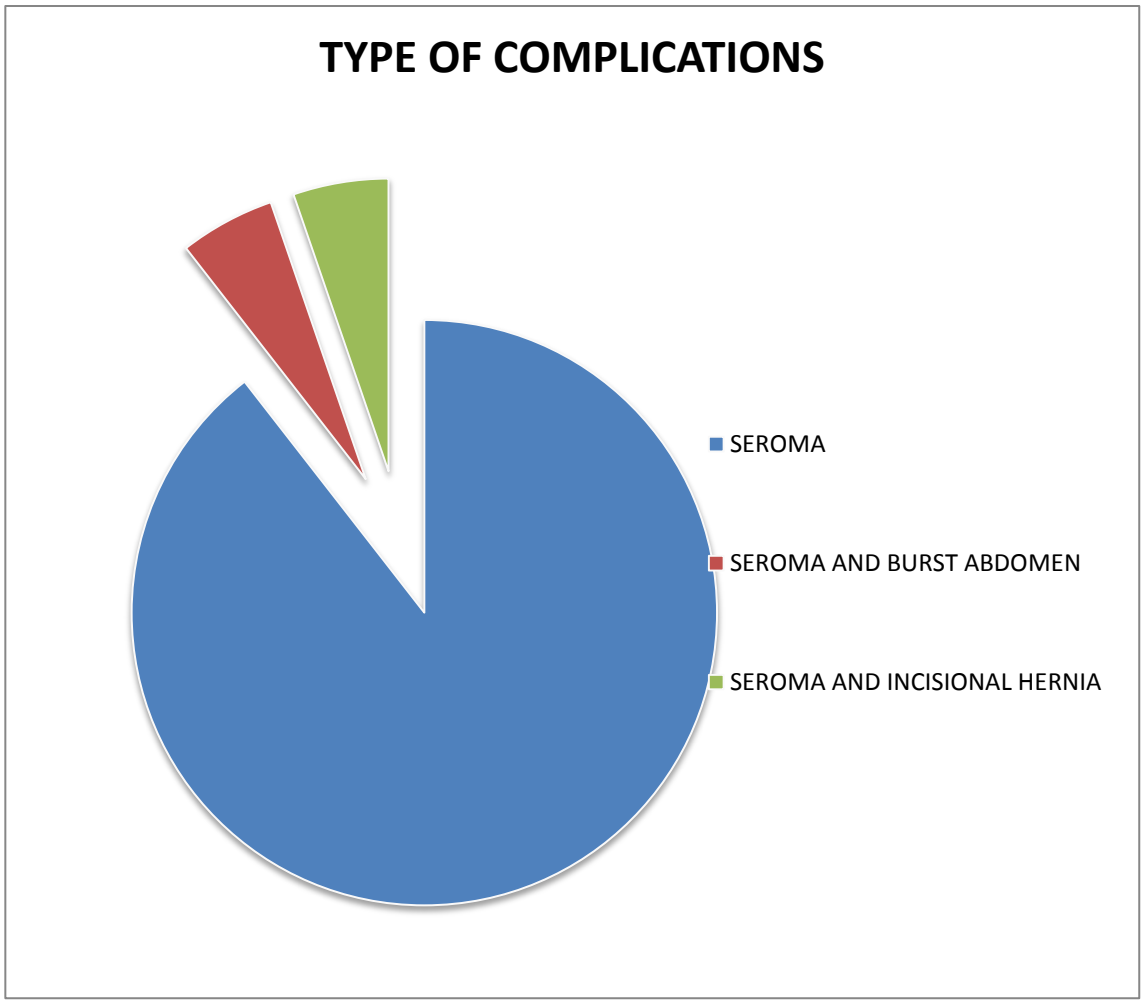
COMPLICATIONS:

Overall the complications occurred in 19 patients out of 120 patients who were followed for 6 months. This constituted a rate of 15.83% and all complications were infection of wound site and were superficial SSIs.



GRAPH 11: COMPLICATIONS

The most common complication was seroma formation postoperatively which was sent for pus culture and sensitivity and results were evaluated. All 19 patients developed seroma from day 2 and it was more on day 3. One patient developed incisional hernia on POD 30 and one more developed burst abdomen on POD 10.



GRAPH 12: TYPE OF COMPLICATIONS

No mortality was noted in the study.

Out of 47 patients who underwent hernioplasty, 8 patients developed complications which constituted 17% more than the average infection rate.

Out of 16 patients who underwent laparotomy, 3 patients developed infection which constituted 18.75%. All laparotomy cases were clean and clean contaminated wounds. No dirty wounds were taken for the study.

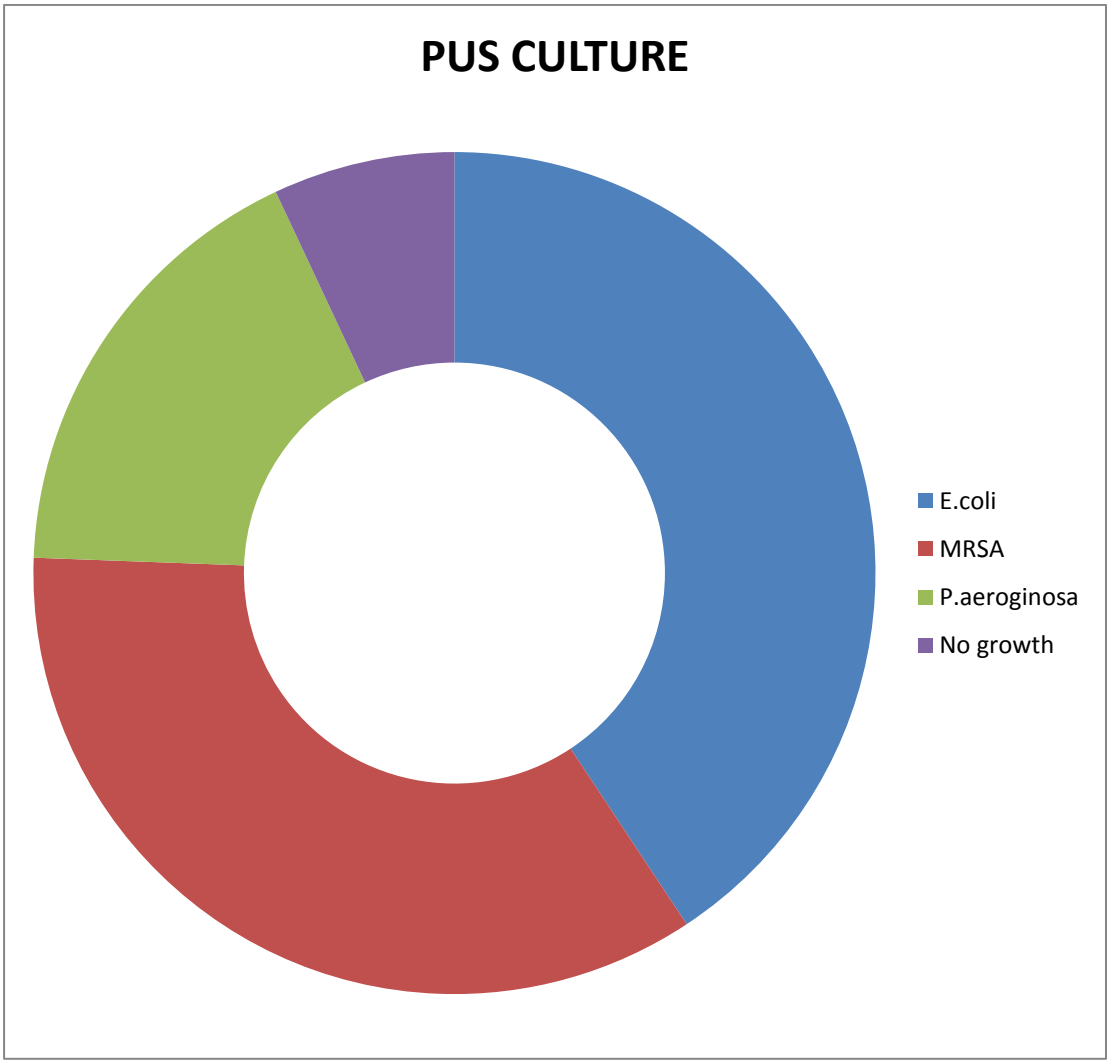
Appendectomy had infection rate of 11.76% and ventral hernia mesh repair had infection rate of 14.81%. The infection rate in CBD exploration cannot be assessed as there is only one case in the study.

TABLE 8: SURGICAL PROCEDURES AND COMPLICATIONS

S.NO.	SURGICAL PROCEDURE	TOTAL NO. OF CASES	CASES COMPLICATED - FREQUENCY	CASES COMPLICATED – PERCENTAGE
1	Hernioplasty	47	8	17.02%
2	Ventral hernia mesh repair	27	4	14.81%
3	Appendectomy	17	2	11.76%
4	Laparotomy	16	3	18.75%
5	Cholecystectomy	12	2	16.66%
6	CBD exploration	1	0	0%

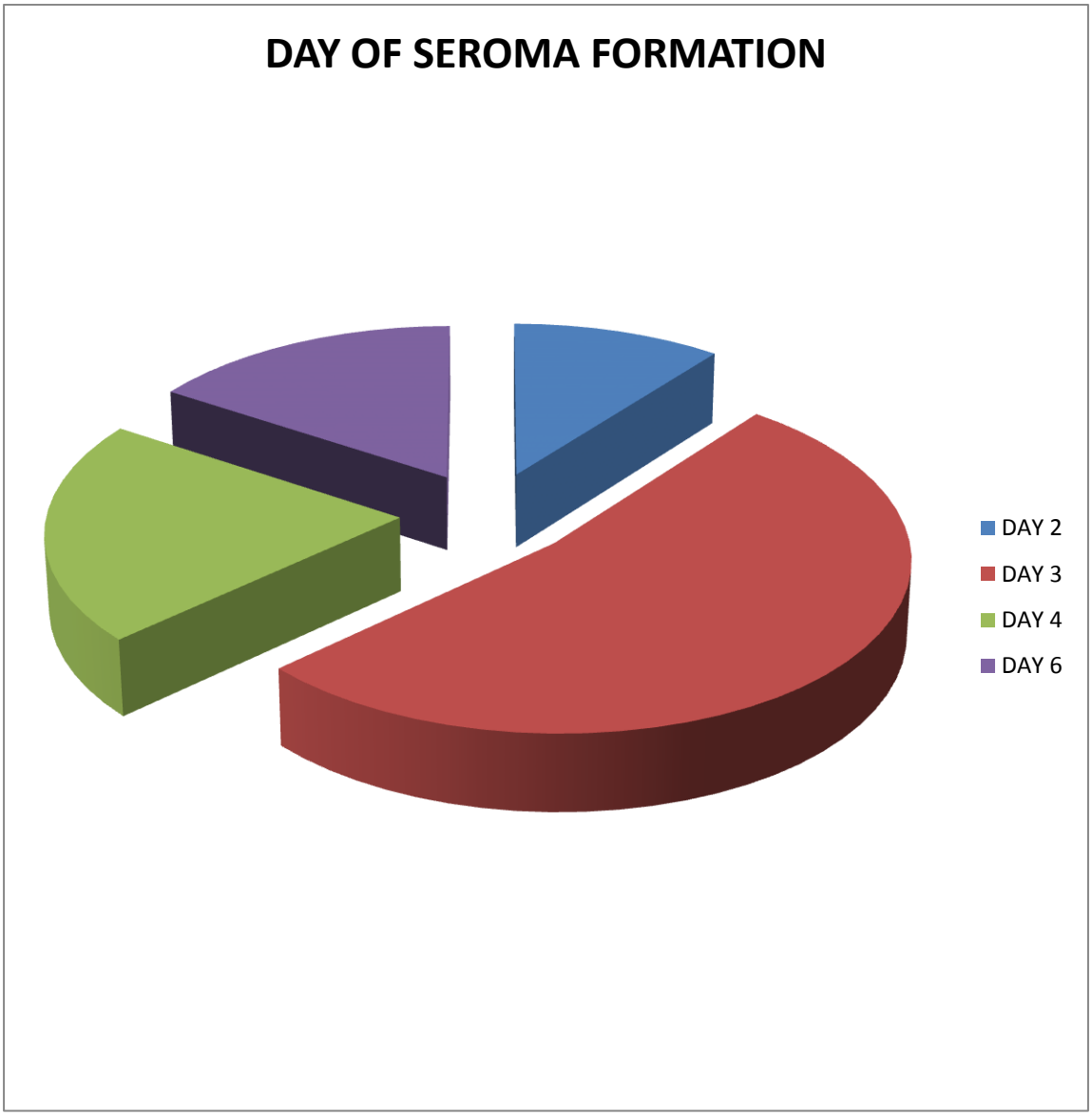
Burst abdomen was noted in a patient who underwent laparotomy and it was a clean contaminated wound. Patient underwent right hemicolectomy for carcinoma caecum.

16(84.21%) of 19 patients who developed seroma tested positive for organism in pus culture and sensitivity. E.coli was positive in 7 patients followed by MRSA in 6 patients. These patients were adequately treated with specific antibiotics.



GRAPH 13: PUS CULTURE

About 10 patients developed seroma on day 3 post operatively. Rest of the patients developing seroma on day 2, 4, 6. These patients had increased length of hospital stay by 3 days when comparing to patients who didn't have any complication.



GRAPH 14: DAY OF SEROMA FORMATION

DISCUSSION

Postoperative wound complications alter the outcome of surgery, hence they are of great importance to a surgeon. They complicate the postoperative course of a significant proportion of abdominal surgical patients, are associated with excessive health-care costs, increased morbidity and mortality, and may require further hospital admissions, IV antibiotics and even surgical re-intervention.

Despite great progress made in the perioperative care, wound complications occur in the range of 2-40% depending on several factors. These rates vary widely from one country to another and it also depends on health infrastructure availability.

Unlike emergency surgeries, elective surgeries have much more scope for flexibility allowing us to assess various factors that can be controlled and predicted. It is difficult to control each and every variable and hence there is possibility of wound complications in spite of best care taken.

In our observational study, we wanted to study the association of investigations that can predict the development of complication on early basis.

In this study, patients are more of male constituting about 66% and about 45 patients out of 120 are in the age group between 50 and 60 years of age constituting 37.50%. mean age of the population is 44.49 years.

About 39% procedures are hernioplasty followed by mesh repair for ventral hernia (22.5%). 16 out of 120 cases underwent laparotomy and included only class I and class II wounds and contaminated and dirty wounds are not taken into the study.

Hemoglobin of 11g/dl is taken as cutoff and anemic patients were identified. Mean hemoglobin is 10.92 with standard deviation of 0.57. Overall 49.16% patients are anemic with hemoglobin less than 11g/dl. 63% of patients who developed complication are anemic with mean of 10.37g/dl and this is higher when comparing to the patients who didn’t develop complications which is around 46.53% who had a mean of 11.04g/dl. Thus anemic patients are 1.36 times more likely to develop

complication than patients who have normal hemoglobin values. There was significant finding noted in MCV values and majority of the patients had normal MCV. Hence it is not possible to find any value of predicting wound complications.

White cell count is taken as an indicator to predict the patients who wound develop complications. The cutoff was taken as 10,000/mm^3. About 15.8% patients who developed complications have elevated white cell count when comparing to patients who didn't have any complication postoperatively which was 5%. Thus if the white cell count is more than 10,000/mm^3, these patients are 5.26 times more likely to end up with complications postoperatively than whose counts are normal.

Based on several studies, cutoff for albumin was taken as 3.5g/dl and patients who have values less than 3.5g/dl are considered hypoalbuminemic. Mean albumin level of patients in this study is 3.66 with a standard deviation of 0.37. Overall, 23.33% patients (28 out of 120 patients) had hypoalbuminemia. Hypoalbuminemia in patients who have complication is 52.6% which is much higher than hypoalbuminemia in patients who had no complications which was 13.8%. Thus it can be well established that if patient have hypoalbuminemia preoperatively, those

patients are 3.81 times more likely to end up with complications postoperatively. Thus malnutrition significantly contributes to impaired wound healing.

There was no significant finding relating the elevated urea and creatinine to the development of complications postoperatively.

Overall 19 patients out of 120 developed some complication postoperatively. This constituted about 15.83%. Sharma et al[17] study had wound complication rate of 10.9%. This result was little higher when comparing to other Indian studies who has a rate of 13.7%. However, they are much higher than the bench mark set by Cruse and Ford[92] who reported 1-2% infection rate in elective surgeries in Canada which is way less than the infection rate in our study which is 13.33%. Various studies has reported infection rate of 5 to 19%. Ahmed et al[93] reported overall infection rate of 11%. Sahu et al[28] reported overall infection rate of 5%, while Satyanarayana et al (2011)[93] reported infection rate of 7.6% in elective surgeries. Saxena et al (2013)[94] observed a infection rate of 14.33%. Our study has infection rate of 13.33%.

Seroma is the most common complication in all patients. Out of 19 patients, 16 patients is culture proven (84.21%) and only 3 patients have no growth. E.coli is isolated in 7 patients (43.75%) followed by MRSA in 6 patients (37.50%). Sahu et al[28] reported E.coli in 50% of infected wounds. Studies by Classen et al (1992)[95] and Giacometti et al (2000)[96] reported monomicrobial and polymicrobial wound infections respectively. We found monomicrobial infections in this study. Seroma develop more on day 3. 5 patients reported wound dehiscence out of 19 patients who developed complications. On POD 10, one patient developed burst abdomen and one more patient developed incisional hernia on POD 30.

Wound dehiscence occurred in 4% patients and all had wound infection. 20% patients of wound dehiscence developed incisional hernia and the occurrence rate of incisional hernia is 0.83%. These observations are in concordance with observations of Wilson and Clark (2003)[97], Ashraf et al (2009)[98] and Murtaza et al (2010)[18] who concluded that the wound infection is the most important single factor in the development of incisional hernia. Studies on wound dehiscence reported similar rates at Indian centers and include those by Srivastava et al (2004)[99] reporting 9% rate, Parmar et al (2009)[100] – 5.6% and Rana et al (2013)[101] – 9% rate.

CONCLUSION

Results of the present study showed a wound complication rate of 15.83% in the form of wound discharge. Purulent discharge was the most common complication (13.33%) with most cases testing positive for E.coli and MRSA as causative pathogen. Wound dehiscence occurred in 4% patients and all had wound infection. 20% patients of wound dehiscence developed incisional hernia and the occurrence rate of incisional hernia is 0.83%.

Mean age of the patient who developed complication is 53.52 years and mean hemoglobin is 10.37. High age and low hemoglobin was observed in patients who developed wound complications. More patients developed complications following hernioplasty possibly due to the mesh and laparotomy as there are clean contaminated cases in which up to 19% wound complication is observed worldwide.

These findings suggest that wound complications do occur in elective abdominal surgeries. These can be reduced to a certain extent by

- careful case selection,
- improving hemoglobin,

- improving nutritional status,
- adopting better aseptic measures,
- better surgical practices,
- using adequate prophylaxis

CDC guidelines are recommended for adoption following this study and this can bring down the wound complications on a larger scale. The infection rate is 15.83% in our study which is acceptable when comparing to other Indian studies further decrease in rates can be achieved by improving the hemoglobin and nutritional status of these patients and by adopting strict aseptic precautions.

LIMITATIONS

- Our study was done involved those patients who were admitted in our surgical department. Hence it does not reflect that of a community as a whole.

- Our study does not include the patients who have comorbidity which can greatly influence wound healing and development of complications.

- This study was done in a tertiary hospital in Chennai and the data cannot be applied to whole of Tamilnadu.

- As the study population is small, it needs large scale study in future to understand better.

BIBLIOGRAPHY

1. Horan TC, Gaynes RP, Martone WJ, Jarvis WR, Emori TG. CDC definitions of nosocomial surgical site infections, 1992: a modification of CDC definitions of surgical wound infections. *Infect Control Hosp Epidemiol*. 1992;13(10):606–608.
2. Barie PS, Wilson SE. Impact of evolving epidemiology on treatments for complicated skin and skin structure infections: the surgical perspective. *J Am Coll Surg*. 2015;220(1):105–106.
3. Horan TC, Andrus M, Dudeck MA. CDC/NHSN surveillance definition of health care-associated infection and criteria for specific types of infections in the acute care setting. *Am J Infect Control*. 2008;36(5): 309–332.
4. Altemeier WA, Culbertson WR, Hummel RP. Surgical considerations of endogenous infections – sources, types, and methods of control. *Surg Clin North Am*. 1968;48(1):227–240.
5. Cheadle WG. Risk factors for surgical site infection. *Surg Infect (Larchmt)*. 2006;7 Suppl 1:S7–S11.
6. Anderson DJ. Surgical site infections. *Infect Dis Clin North Am*. 2011; 25(1):135–153.

7. Anderson DJ, Chen LF, Sexton DJ, Kaye KS. Complex surgical site infections and the devilish details of risk adjustment: important implications for public reporting. *Infect Control Hosp Epidemiol*. 2008; 29(10):941–946.
8. Segal CG, Waller DK, Tilley B, Piller L, Bilimoria K. An evaluation of differences in risk factors for individual types of surgical site infections after colon surgery. *Surgery*. 2014;156(5):1253–1260.
9. Ho VP, Stein SL, Trencheva K, et al. Differing risk factors for incisional and organ/space surgical site infections following abdominal colorectal surgery. *Dis Colon Rectum*. 2011;54(7):818–825.
10. Stevens DL, Bisno AL, Chambers HF, et al. Practice guidelines for the diagnosis and management of skin and soft tissue infections: 2014 update by the Infectious Diseases Society of America. *Clin Infect Dis*. 2014;59(2):e10–e52.
11. Khuri SF, Henderson WG, DePalma RG, et al. Determinants of long-term survival after major surgery and the adverse effect of postoperative complications. *Ann Surg*. 2005;242(3):326–341; discussion 341–343.
12. Drapeau CM, Pan A, Bellacosa C, et al. Surgical site infections in HIV-infected patients: results from an Italian prospective multicenter

observational study. *Infection*. 2009;37(5):455–460.

13. Neumayer L, Hosokawa P, Itani K, El-Tamer M, Henderson WG, Khuri SF. Multivariable predictors of postoperative surgical site infection after general and vascular surgery: results from the patient safety in surgery study. *J Am Coll Surg*. 2007;204(6):1178–1187.
14. Hechenbleikner EM, Hobson DB, Bennett JL, Wick EC. Implementation of surgical quality improvement: auditing tool for surgical site infection prevention practices. *Dis Colon Rectum*. 2015;58(1):83–90.
15. Young PY, Khadaroo RG. Surgical site infections. *Surg Clin North Am*. 2014;94(6):1245–1264.
16. Alexander JW, Solomkin JS, Edwards MJ. Updated recommendations for control of surgical site infections. *Ann Surg*. 2011;253(6):1082–1093.
17. Sharma A.C., Singla Mamta, Shuaib Mohammad, Kumar Spandan. Evaluation of wound complications in elective abdominal surgery. International Journal of Contemporary Medical Research 2016;3(10):3095-3098.
18. Murtaza B, Saeed S, Shareef MA. Post-operative Complications In Emergency vs Elective Laparotomies At A Peripheral Hospital. J. Ayub Medical College, Abbottabad 2010;22:42-47.
19. Krukowski ZH, Stewart MPM, Alsayer HM, Matheson NA. Infection After

Abdominal Surgery: 5 Year Prospective Study. BMJ. 1984;288:278-280.

20. Sorensen LT, Hemmingsen U, Kallehave U, Wille- Jorgenson P, Kjaergaard J, Moller LN, Jorgenson T. Risk Factors For Tissue And Wound Complications In Gastrointestinal Surgery. Ann. Surg. 2005;241:654-658.
21. Col C, Soran A, Col M. Can Post-Operative Abdominal Wound Dehiscence Be Predicted? Tokai J. Exp. Clin. Med. 1998;23;123-27.
22. Lilani SP, Jangale N, Chowdhary A,Daver GB.Surgical infection in clean and clean contaminated cases. Indian J Med Microbiol 2005; 23(4): 249-52.
23. Makela JT, Kiviniemi H. Factors Influencing Wound Dehiscence After Midline Laparotomy. Am. J. Surg. 1995; 170:387-90.
24. C.D. Owens, K. Stoessel. Surgical site infection: Epidemiology, microbiology and prevention. Journal of Hospital Infection (2008) 70(S2) 3–10.

25. CDC guidelines for SSI. Jan 2019 9-34.
26. Azoury SC, Farrow NE, Hu QL, Soares KC et al. Postoperative Wound Infections – Epidemiology, Risk Factors, Identification And Management. Chronic Wound Care Management and Research. 2015;2:137-148.
27. Ahmed M, Alam SA, Khan O, Manzar S. Postoperative Wound Infection: A Surgeon’s Dilemma. Pak. J. Surg. 2007;23:41-47.

28. Sahu SK, Shergill JS, Sachan PK, Gupta P. Superficial Incisional Surgical Site Infection In Elective Abdominal Surgery. The Internet Journal Of Surgery. 2011;26: No.1.

29. Gosain A, DiPietro LA (2004). Aging and wound healing. *World J Surg* 28:321-326.

30. Meszaros AJ, Reichner JS, Albina JE (2000), Macrophage-induced neutrophil apoptosis. *J Immunol* 165:435-441.

31. Swift ME, Burns AL, Gray KL, DiPietro LA (2001). Age-related alterations in the inflammatory response to dermal injury. *J Invest Dermatol* 117:1027-1035.

32. Gawronska-Kozak B, Bogacki M, Rim JS, Monroe WT, Manuel JA (2006) Scarless skin repair in immunodeficient mice. *Wound Repair Regen* 14:265-276.

33. Jameson J, Havran WL (2007). Skin gammadelta T-cell functions in homeostasis and wound healing. *Immunol Rev* 215:114-122.

34. Cha J, Falanga V (2007). Stem cells in cutaneous wound healing. *Clin Dermatol* 25:73-78.

35. Wu Y, Wang J, Scott PG, Tredget EE (2007). Bone marrow-derived stem cells in wound healing: a review. *Wound Repair Regen* 15(Suppl 1):S18-S26.

36. Bishop A (2008). Role of oxygen in wound healing. *J Wound Care* 17:399-402.

37. Tandara AA, Mustoe TA (2004). Oxygen in wound healing—more than a nutrient. *World J Surg* 28:294-300.

38. Rodriguez PG, Felix FN, Woodley DT, Shim EK (2008). The role of oxygen in wound healing: a review of the literature. *Dermatol Surg* 34:1159-1169.

39. Edwards R, Harding KG (2004). Bacteria and wound healing. *Curr Opin Infect Dis* 17:91-96.

40. Broughton G 2nd, Janis JE, Attinger CE (2006). The basic science of wound healing (retraction of Witte M., Barbul A. In: *Surg Clin North Am* 1997; 77:509-528). *Plast Reconstr Surg* 117(7 Suppl):12S-34S.

41. Emery CF, Kiecolt-Glaser JK, Glaser R, Malarkey WB, Frid DJ (2005). Exercise accelerates wound healing among healthy older adults: a preliminary investigation. *J Gerontol Med Sci* 60(A):1432-1436.

42. Shepherd AA (2003). Nutrition for optimum wound healing. *Nurs Stand* 18:55-58.

43. Arnold M, Barbul A (2006). Nutrition and wound healing. *Plast Reconstr Surg* 117(7 Suppl):42S-58S.

44. Gogia PP (1995). Physiology of wound healing. In: *Clinical wound management*. Gogia PP, editor. Thorofare, NJ: Slack Incorporated, pp 8-12.

45. Campos AC, Groth AK, Branco AB (2008). Assessment and nutritional aspects of wound healing. *Curr Opin Clin Nutr Metab Care* 11:281-288.

46. Tong BC, Barbul A (2004). Cellular and physiological effects of arginine. *Mini Rev Med Chem* 4:823-832.

47. da Costa MA, Campos AC, Coelho JC, de Barros AM, Matsumoto HM (2003). Oral glutamine and the healing of colonic anastomoses in rats. *JPEN J Parenter Enteral Nutr* 27:182-185.

48. McDaniel JC, Belury M, Ahijevych K, Blakely W (2008). Omega-3 fatty acids effect on wound healing. *Wound Repair Regen* 16:337-345.

49. Burgess C (2008). Topical vitamins. *J Drugs Dermatol* 7(7 Suppl):s2-s6.

50. Khoosal D, Goldman RD (2006). Vitamin E for treating children's scars. Does it help reduce scarring? *Can Fam Physician* 52:855-856.

51. Heyman H, Van De Looverbosch DE, Meijer EP, Schols JM (2008). Benefits of an oral nutritional supplement on pressure ulcer healing in long-term care residents. *J Wound Care* 17:476-478, 480.

52. Heys SD, Simpson WG, Eremin O. Surgical nutrition. In: Paterson-Brown S, editor. Emergency surgery and critical care. 7th ed. London: *WB Saunders,* 1997; 55.

53. Gibbs J, Cull W, Henderson W, Daley J, Hur K, Khuri SF. Preoperative Serum Albumin level as a predictor of operative mortality and morbidity. *Arch Surg* 1999; 134:36-42.

54. Williams JZ, Barbul A. Nutrition and wound healing. *Surg Clin North Am* 2003; 83:571.

55. trauma. IN Ivatury RR, Cayten CG (eds). Textbook of Penetrating Trauma. Philadelphia, PA, Williams & Wilkins, 1996, pp 142–150.

56. The Veteran Affairs Total Parenteral Nutrition Cooperative Study Group: Perioperative total parenteral nutrition in surgical patients. N Engl J Med 325:525–532, 1991.

57. Brennan MF, Pisters PWT, Posner M, et al: A prospective, randomizedtrial of total parenteral nutrition after major pancreatic resection for malignancy. Ann Surg 220:436–444, 1994.

58. Daly JM, Lieberman MD, Goldfine J, et al: Enteral nutrition with supplemental arginine, RNA, and omega-3 fatty acids in patients after operation: Immunologic, metabolic, and clinical outcome. Surgery 112:56–67, 1992.

59. Daly JM, Weintraub FN, Shou J, et al: Enteral nutrition during multimodality therapy in upper gastrointestinal cancer patients. Ann Surg 221:327–338, 1995.

60. Heslin MJ, Latkany L, Leung D, et al: A prospective, randomized trial of early enteral feeding after resection of upper gastrointestinal malignancy. Ann Surg 226:567–577, 1997.

61. Windsor JA, Hill GL: Protein depletion and surgical risk. Aust NZ J Surg 58:711–715, 1988.

62. Gibbs J, Cull W, Henderson W, et al: Preoperative serum albumin level as a predictor of operative mortality and morbidity: Results from the National Surgical Risk Study. Arch Surg 134: 36–42, 1999.

63. Hickman DM, Miller RA, Rombeau JL, et al: Serum albumin and body weight as predictors of postoperative course in colorectal cancer. JPEN 4:314–316, 1980.

64. Buzby GP, Mullen JL, Matthews DC, et al: Prognostic nutritional index in GI surgery. Am J Surg 139:160–167, 1980.

65. Reinhardt GF, Myscofski JW, Wilkens DB, et al: Incidence and mortality of hypoalbuminemic patients in hospitalized veterans. JPEN 4:357–359, 1980.

66. Mullen JL, Gertner MG, Buzby GP, et al: Implication of malnutrition in the surgical patient. Arch Surg 114:121–125, 1979.

67. Pikul J, Sharpe MD, Lowndes R, et al: Degree of preoperative malnutrition is predictive of postoperative morbidity and mortality in liver transplant recipients. Transplantation 57:469– 472, 1994.

68. Daley J, Khuri SF, Henderson W, et al: Risk adjustments of the postoperative morbidity rate for the comparative assessment of the quality of surgical care. Results of the National Veterans Affairs Surgical Risk Study. J Am Coll Surg 185:328–340, 1997.

69. Collins TC, Daley J, Henderson WH, et al: Risk factors for prolonged length of stay after major elective surgery. Ann Surg 230:251–259, 1999.

70. Khuri SF, Daley J, Henderson W, et al: The Department of Veterans Affairs' NSQIP. The first national, validated, outcomebased, risk-adjusted, and peer-controlled program for the measurement and enhancement of the quality of surgical care. Ann Surg 228:491–507, 1998.

71. Arora NS, Rochester DF: Respiratory muscle strength and maximal voluntary ventilation in undernourished patients. Am Rev Respir Dis 126:5–8, 1982.

72. Khuri SF, Daley J, Henderson W, et al: Risk adjustment of the postoperative mortality rate for the comparative assessment of the quality of surgical care: Results of the National Veterans Affairs Surgical Risk Group. J Am Coll Surg 185:315–327, 1997.

73. Studley HO: Percentage of weight loss: A basic indicator of surgical risk in patients with chronic peptic ulcer. JAMA 106: 458–460, 1936

74..Marsh, R. C., Coxe, J. W., Ross, W. L. and Stevens, G. A.: Factors Involved in Wound Dehiscence. Survey of 1000 Cases. J.A.M.A., 155:1197, 1954.

75.Mann, L. S., Sprimafola, A. J., Lindesmith, G. G., Levine, M. J. and Kvezerepa, W.: Disruption of Abdominal Wounds, J.A.M.A., 180: 1021, 1962.

76.Alexander, H. C. and Prudden, J. F.: The Causes of Abdominal Wound Disruption. Surg. Gynec. Obstet., 122:1223, 1966.

77.Guiney, E. J., Morris, P. J. and Donaldson, G. A.: Wound Dehiscence. Arch. Surg., 92:47, 1966.

78.Besser, E. L. and Ehrenhaft, J. L.: The Relationship of Acute Anemia to Wound Healing. Surgery, 14:239, 1943.

79.Trueblood, H. W., Nelsen, T. S. and Oberhelman, H. A.: The Effect of Acute Anemia and Non-deficiency Anemia on Wound Healing. Arch. Surg., 99:113, 1969.

80.Waterman, D. F., Birkhill, F. R., Pirani, C. L. and Levenson, S. M.: The Healing of Wounds in the Presence of Anemia. Surgery, 31:821, 1952.

81.Macon, W. L. and Pories, W. J.: The Effect of Iron Deficiency Anemia on Wound Healing. Surgery, 69:792, 1971.

82. Jacobson, M. J. and Van Prohaska, J.: The Healing of Wounds in Iron Deficiency. Surgery, 57:254, 1965.

83. Bains, J. W., Crawford, D. T. and Ketcham, A. S.: Effect of Chronic Anemia on Wound Tensile Strength. Correlation With Blood Volume, Total Proteins, Total Red Blood Cell Volumes. Ann. Surg., 164:243, 1966.

84. Hugo, N. E., Thompson, L. W., Zook, E. S. and Bennett, J. E.: The Effect of Chronic Anemia on the Tensile Strength of Healing Wounds. Surgery, 66:741, 1969.

85. Sandblom, P.: The Tensile Strength of Healing Wounds. Systemic Factors. Anemia and Dehydration. Acta Chir. Scand., 90 (Supp. 89): 71, 1944.

86. Sandberg, N. and Zederfeldt, B.: Influence of Acute Hemorrhage on Wound Healing in the Rabbit. Acta Chir. Scand., 118:367, 1960.

87. Hunt, T. K., Zederfeldt, B., Goldstick, T. K. and Conolly, W. B.: Tissue Oxygen Tensions During Controlled Hemorrhage. Surg. Forum, 18:3, 1967.

88. Hunt, T. K. and Pai, M. P.: The Effect of Varying Ambient Oxygen Tensions on Wound Metabolism and Collagen Synthesis. Surg. Gynec. Obstet., 135:561, 1972.

89. Ehrlich, H. P., Grislis, G. and Hunt, T. K.: Metabolic and Circulatory Contributions to Oxygen Gradients in Wounds. Surgery, 72:578, 1972.

90. Juva, K.: Hydroxylation of Proline in the Biosynthesis of Collagen. Acta Physiol. Scand., Supp. 308, 1968.
91. Niinikoski, J.: Effect of Oxygen Supply on Wound Healing and Formation of Experimental Granulation Tissue. Acta Physiol. Scand., Supp. 334, 1969.
92. Cruse PJ, Ford R. The Epidemiology Of Wound Infection: A Ten-Year Prospective Study Of 62,939 Wounds. Surg. Clin. North Am. 1980;60:27-40.
93. Satyanarayan V, Prashanth HV, Basavaraj B, Kavyashree AN. Study Of Surgical Site Infections In Abdominal Sugeries. Journal Of Clinical And Diagnostic Research. 2011;5:935-39.
94. Saxena A, Singh MP, Brahmachari S, Banerjee M. Surgical Site Infection Among Postoperative Patients Of Tertiary Care Centre In Central India – A Prospective Study. Asian J. Biomed. And Pharmaceutical Sciences. 2013;3:41-44.
95. Classen DC, Evans RS, Pestotnik SL, Horn HD, Menlore RL, Burke JP. The Timing Of Prophylactic Administration Of Antibiotic And Risk Of Surgical Wound Infection. N. Eng. J. Med. 1992;326:281-6.
96. Giacometti A, Cirioni O, Schimizzi AM, Del Prete MP, Barchiesi F, Derrico MM. Epidemiology And Microbiology Of Surgical Wound Infection. J. Clini. Microbiology. 2000: 38:918-22.
97. Wilson JA, Clark JJ. Obesity: Impediment To Wound Healing. Crit. Care. Nurs. Q 2003;26:119-32.

98. Ashraf SM, Mehdi SH, Umer MF, El-Muttaqi A. Comparative Study Of Wound Healing In Primary vs Delayed Primary Closure In Contaminated Abdominal Surgery. Pak. J. Surg. 2009;25:115-18.
99. Srivastava A, Roy S, Sahay KB, Seenu V, Kumar A, Chumber S, Bal S, Mehta S. A Randomized Trial Comparing Continuous vs Interrupted X-Sutures. Indian Journal Of Surgery. 2004;66:19-27.
100. Parmar G, Gohil A, Hathila V. Burst Abdomen – A Grave Postoperative Complication. Internet Journal Of Surgery. 2009;20: No. 1.
101. Rana KV, Singh G, Deshpande NA, Bharathan VK, Sridharan S. Postoperative Complications Of Mesh Hernioplasty For Incisional Hernia Repair And Factors Affecting Occurrence Of Complications. Med. J. DY Patil University. 2013;6:25-31.

1	LAKSHMI	60	F	134235	CHOLE	COLE	11	82	12000	3.6	40	1	SEROMA	3	ECOLI
2	HEMAMALINI	25	F	2423	APPEN	APPEN	11	88	6200	3.5	36	1			
3	VIVEK	47	M	138903	VENTRAL	MESH	10.7	88	7300	3.6	38	1			
4	ARUNACHALAM	59	M	4872	HERNIA	PLASTY	10.4	84	6400	3.2	38	1	SEROMA	3	MRSA
5	SHANMUGAM	57	M	136457	HERNIA	PLASTY	11.2	91	6600	3.8	30	1			
6	ALAMELU	46	F	6162	LAP	LAP	10.7	87	5400	3.8	53	1			
7	MOHANRAJ	52	M	5833	LAP	LAP	10.6	91	6500	3.9	26	1			
8	VASANTHA	38	F	10821	LAP	LAP	11.7	87	5475	4.1	18	1			
9	RAM	51	M	10688	VENTRAL	MESH	10.8	90	5400	3.4	54	1			
10	SELVI	40	F	11285	LAP	LAP	10.7	88	11500	3.7	34	1			
11	KUPPUSAMY	55	M	16237	HERNIA	PLASTY	11	87	7500	3.5	39	1			
12	EZHUMALAI	39	M	16409	HERNIA	PLASTY	11.4	90	5400	3.7	33	1			
13	LOGANATHAN	59	M	14312	LAP	LAP	12	86	8600	3.8	33	1			
14	GUNASEKAR	39	M	19406	CHOLE	CHOLE	11	87	5300	3.6	36	1			
15	GOVIND	52	F	17137	VENTRAL	MESH	10.5	86	7500	3.7	34	1			
16	BHAVANI	27	F	22516	APPEN	APPEN	11.7	91	5400	3.5	33	1			
17	MOORTHY	59	M	19432	VENTRAL	MESH	11	90	8900	3.8	34	1			
18	ANSARI	50	M	19417	HERNIA	PLASTY	11.4	82	5900	3.5	35	1	SEROMA	3	NO
19	JAGADEESA	57	M	10760	LAP	LAP	10.9	90	7500	3.7	31	1			
20	LAKSHA	40	M	25252	HERNIA	PLASTY	11	87	8200	3.8	29	1			
21	JAGADEESH	22	M	25666	HERNIA	PLASTY	10.3	94	5200	3.4	36	1			
22	LOGANATHAN	40	M	25156	APPEN	APPEN	11.5	86	6500	3.8	26	1			
23	MUNISAMY	56	M	16214	CHOLE	CHOLE	10.7	92	4400	3.6	36	1			
24	GANAPATHY	24	M	128371	HERNIA	PLASTY	11.6	89	7200	3.2	54	1			
25	SANDHYA	19	F	28205	APPNE	APPEN	10.8	92	5300	3.6	35	1			
26	RANI	35	F	22271	LAP	LAP	11.2	89	5400	3.8	23	1			
27	VASANTHA	40	F	31117	VENTRAL	MESH	11.2	88	4500	3.3	24	1			
28	MUNISAMY	53	M	28416	VENTRAL	MESH	11.4	95	5500	4	52	1			
29	RAMASAMY	58	M	29368	HERNIA	PLASTY	10.8	86	8600	4.2	32	1			
30	MOHAMMAD	35	M	35147	APPEN	APPEN	11	88	5400	3.9	40	1			
31	NEDU	46	M	34015	HERNIA	PLASTY	11.2	90	5700	3.9	54	1			
32	PRABAKAR	23	M	33777	VENTRAL	MESH	11	90	6300	3.3	33	1			
33	MANGAVAN	50	M	37177	HERNIA	PLASTY	10.6	88	10500	3.5	53	1			
34	REVATHY	50	F	136226	VENTRAL	MESH	10.9	91	7500	3.2	33	1			
35	VARADAN	49	M	34072	VENTRAL	MESH	10.9	88	6600	3.6	22	1			
36	SUBU	35	F	39135	APPEN	APPEN	11	87	8300	4	54	1			
37	SHYLAJA	34	F	4547	CHOLE	CHOLE	11	89	6300	4.3	39	1			
38	ARJUN	25	M	40714	HERNIA	PLASTY	10.2	89	6200	3.6	37	0			
39	DAS	55	M	42150	HERNIA	PLASTY	12	93	7800	3.9	35	1			
40	HARI	36	M	42190	HERNIA	PLASTY	10.7	89	7600	4	40	1			
41	NAGAMMAL	45	F	42414	VENTRAL	MESH	10.5	87	7500	3.4	28	1			
42	RAJA	35	M	44889	HERNIA	PLASTY	10.9	89	6300	4.3	32	1			
43	RANI	43	F	45062	CHOLE	CHOLE	11	88	6500	3.8	35	1			
44	PRIYANKA	24	F	50335	APPEN	APPEN	11	87	7200	3.8	38	1			
45	RAVI	52	M	47962	VENTRAL	MESH	10.1	81	8200	3.1	44	1	SEROMA	6	MRSA
46	SANJAYA	55	M	42351	VENTRAL	MESH	9.2	86	4800	2.9	40	1	SEROMA	3	NO
47	CHINNAPAYAN	55	M	42370	LAP	LAP	11.6	88	6500	3.7	32	1			
48	JOTHI	48	F	47896	VENTRAL	MESH	11.9	86	4700	3.2	35	1			
49	ANNADURAI	49	M	47866	VENTRAL	MESH	11	90	7600	3.7	32	1			
50	REVATHY	32	F	50300	CHOLE	CHOLE	10.4	87	8200	3.9	63	1			
51	BHAVANI	36	F	50542	VENTRAL	MESH	11.6	94	7200	3.8	36	1			
52	JALENDRAN	54	M	51049	HERNIA	PLASTY	11	86	6400	3.4	32	1			
53	SAROJA	45	F	53666	APPEN	APPEN	9.3	82	14000	3.1	38	1	SEROMA	2	ECOLI
54	ANAND	42	M	56228	HERNIA	PLASTY	10.7	91	6500	3.3	35	1			
55	RAMANIYA	59	M	50666	VENTRAL	MESH	11	86	5000	3.8	36	1	SEROMA	6	ECOLI
56	RUK	38	F	53428	APPEN	APPEN	10.8	88	7400	3.8	34	1			
57	SANKAR	45	M	62370	LAP	LAP	11	90	5700	3.5	23	1			

58	ABDUL	55	M	59526	VENTRAL	MESH	10.9	90	6500	4	33	1			
59	UMA	50	F	63419	VENTRAL	MESH	10.7	93	7500	3.5	23	1			
60	SANKAR	59	M	64954	HERNIA	PLASTY	10.6	87	4500	3.9	53	1			
61	RAJA	40	M	64971	HERNIA	PLASTY	11	90	7200	3.9	33	1			
62	GOVIND	58	M	70107	HERNIA	PLASTY	11	87	8500	4.2	33	1			
63	SENTHIL	30	M	72629	VENTRAL	MESH	11.2	90	7300	3.5	45	1			
64	INDIRA	53	F	72726	VENTRAL	MESH	10.9	91	7600	3.9	27	1			
65	MALAR	56	F	77134	LAP	LAP	10.4	86	9000	2.9	34	1	INCISION	30	ECOLI
66	PALANY	37	M	77861	HERNIA	PLASTY	10.9	90	7400	4.2	33	1			
67	GOVIND	55	F	78066	CHOLE	CHOLE	9.8	78	10000	3.7	30	1	SEROMA	3	ECOLI
68	GOWRI	36	F	79841	APPEN	APPEN	11	91	6300	3.7	34	1			
69	GNANA	50	M	80559	HERNIA	PLASTY	11	87	4400	3.6	29	1			
70	ELLAMAL	55	F	83714	HERNIA	PLASTY	11	88	5800	4.3	34	1			
71	JANANI	16	F	83591	APPEN	APPEN	11	90	8200	3.9	33	1			
72	DURAI	35	M	75264	HERNIA	PLASTY	10.8	90	4000	4.6	22	1			
73	GOWNDAR	59	M	78007	CHOLE	CHOLE	10.9	87	6600	3.4	33	1			
74	NEELA	38	F	85798	APPEN	APPEN	10.1	84	8000	3.6	35	1	SEROMA	3	ECOLI
75	KRISHANN	54	M	85661	VENTRAL	MESH	10.2	80	6800	2.7	28	1	SEROMA	6	PSEUDO
76	KANNIYAPPAN	40	M	88543	HERNIA	PLASTY	10.9	91	7400	3.4	34	1			
77	MANI	56	M	91246	HERNIA	PLASTY	12	90	4700	3.5	26	1	SEROMA	2	MRSA
78	SANJANA	60	F	91027	APPEN	APPEN	11	85	8800	3.8	32	1			
79	RAJA	48	M	88437	HERNIA	PLASTY	9.9	84	8000	3.2	28	1	SEROMA	3	MRSA
80	RAJAN	48	M	88431	HERNIA	PLASTY	11.3	90	7200	3.4	36	1			
81	MANONMANI	45	F	93026	CHOLE	CHOLE	11.4	88	7600	4.4	54	1			
82	NARASAMMA	55	F	93380	VENTRAL	MESH	10.9	87	7600	3.8	37	1			
83	SANTHOSH	20	M	9930	APPEN	APPEN	10.6	93	4200	3.5	33	1			
84	JENIFER	15	F	99120	LAP	LAP	10.8	91	6300	3.7	33	1			
85	SAMY	36	M	96569	VENTRAL	MESH	10.8	93	4500	3.9	37	1			
86	DANDAPANI	40	M	97943	HERNIA	PLASTY	10.8	88	7300	3.5	35	1			
87	RANGANATHAN	53	M	98044	HERNIA	PLASTY	11.2	88	4500	3.6	33	1			
88	SHANMUGAM	54	M	10162	HERNIA	PLASTY	13	90	6600	3.8	33	1			
89	DEVI	49	M	100675	VENTRAL	MESH	11.2	87	6500	3.9	30	1			
90	SIVAPALAN	31	M	100815	HERNIA	PLASTY	10.9	86	7200	3.8	25	2			
91	RANGANATHAN	39	M	100689	HERNIA	PLASTY	11.3	91	6200	3.8	32	1			
92	RAJ	35	M	103050	APPEN	APPEN	11	86	7500	4.2	32	0			
93	SYAMALA	24	F	106131	APPEN	APPEN	11.3	86	8200	3.9	37	1			
94	RAMADAS	46	M	103125	HERNIA	PLASTY	11.2	90	3700	2.6	38	1	SEROMA	4	MRSA
95	MANIKAM	59	M	106064	LAP	LAP	11	85	7700	4	36	1			
96	DANIEL	57	M	105814	HERNIA	PLASTY	11.3	91	6500	3.9	34	1			
97	KISORE	47	M	108958	CHOLE	CHOLE	10.5	85	6200	3.5	37	1			
98	KUMAR	44	M	108668	VENTRAL	MESH	11	91	5600	3.6	39	1			
99	SUBU	38	F	108992	VENTRAL	MESH	11.4	89	5400	3	54	1			
100	RANGANATHAN	58	M	109124	HERNIA	PLASTY	9.6	85	5600	3.7	32	1	SEROMA	3	MRSA
101	RAJESWAI	59	F	110254	HERNIA	PLASTY	10.8	86	5500	3.8	30	1			
102	VIJAYA	40	F	116654	APPEN	APPEN	10.8	92	5400	3.4	36	1			
103	YASODA	49	F	108949	LAP	LAP	11	86	6500	3.6	32	1			
104	SANKAR	35	M	103213	LAP	LAP	10.5	91	6000	4.7	37	1			
105	PALANIAMMA	55	F	11453	LAP	LAP	9.9	84	10000	3.9	54	1	SEROMA	4	NO
106	HARIHARAN	51	M	120066	HERNIA	PLASTY	12.4	86	4200	3.6	20	1	SEROMA	3	PSEUDO
107	JEEVA	27	M	114030	HERNIA	PLASTY	10.7	92	12000	3.4	35	1			
108	RAMU	52	M	114224	HERNIA	PLASTY	10.7	90	4500	3.8	55	1			
109	THANGAIYA	44	M	116621	HERNIA	PLASTY	10.6	92	5400	3.7	34	1			
110	VANAJA	43	F	108830	VENTRAL	MESH	10.8	87	6600	3.9	32	1			
111	GODANDAM	31	M	116681	HERNIA	PLASTY	11	85	5400	3.7	38	1			
112	THANGAVEL	60	M	120011	HERNIA	PLASTY	11	84	8000	3.1	32	1	SEROMA	3	PSEUDO
113	ETHIAN	60	M	125560	LAP	LAP	9	80	11000	2.4	60	1	BURST	10	ECOLI
114	MANNU	53	M	123703	HERNIA	PLASTY	11.5	87	8900	3.5	34	1			
115	AMREEN	34	F	123705	CHOLE	CHOLE	10.6	84	6200	4.2	26	1			

116	SULTHAN	43	M	125227	HERNIA	PLASTY	10.8	90	6700	3.2	33	2			
117	ARUNACHALAM	34	M	130970	CHOLE	CHOLE	10.7	88	7200	4.1	53	1			
118	ALI	56	M	130751	HERNIA	PLASTY	12	92	7500	3.2	35	1			
119	MANIKANDAN	31	M	133852	CHOLEDO	CBD	10.7	89	7200	3.6	36	1			
120	SIVARAMAN	55	M	128364	HERNIA	PLASTY	11.4	94	4500	3.8	33	2			