Dissertation

"LEUCOCYTOSIS AFTER POST TRAUMATIC SPLENECTOMY- A PHYSIOLOGICAL EVENT OR INDICATOR OF SEPSIS"

Dissertation submitted to

THE TAMIL NADU Dr. M.G.R. MEDICAL UNIVERSITY

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

M.S. (General Surgery)

Branch – I

THE TAMILNADU Dr. MGR MEDICAL UNIVERSITY

CHENNAI

May 2019

CERTIFICATE

This is to certify that, the dissertation entitled **"LEUCOCYTOSIS AFTER POST TRAUMATIC SPLENECTOMY- A PHYSIOLOGICAL EVENT OR INDICATOR OF SEPSIS"** is the bonafide work done by **DR. V. SWETHA** during her **M.S. (General Surgery)** course **2016-2019**, done under my supervision and is submitted in partial fulfillment of the requirement for the M.S.(BRANCH-I)- General Surgery of The Tamil nadu Dr.MGR Medical University, May 2019 examination.

Prof. M.ALLI M.S., DGO
Professor & Head of the Department
Institute of General Surgery
Madras Medical College
Chennai – 03.

Prof.P.THANGAMANI M.S.FMAS.,FAES.,
Professor
Institute of General Surgery
Madras Medical College
Chennai – 03.

DR. R. JAYANTHI M.D., FRCP,

THE DEAN

Madras Medical College & Rajiv Gandhi Government
General Hospital

Chennai-03

DECLARATION

I, certainly declare that this dissertation titled **"LEUCOCYTOSIS AFTER POST TRAUMATIC SPLENECTOMY- A PHYSIOLOGICAL EVENT OR INDICATOR OF SEPSIS"** represents a genuine work of mine. The contributions of any supervisors to the research are consistent with normal supervisory practice, and are acknowledged.

I also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other University board, either in India or abroad. This is submitted to The TamilNadu Dr. M.G.R Medical University, Chennai in partial fulfillment of the rules and regulations for the award of Master of Surgery Degree Branch I (General Surgery).

DATE: **DR. V. SWETHA**

PLACE: (POST GRADUATE)

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"To acknowledge is to know gratitude".

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CERTIFICATE OF APPROVAL

To

Dr.V.Swetha
I Year PG in MS General Surgery
Institute of General Surgery
Madras Medical College
Chennai 600 003

Dear Dr.V.Swetha,

The Institutional Ethics Committee has considered your request and approved your study titled **"LEUCOCYTOSIS AFTER POST TRAUMATIC SPLENECTOMY – A PHYSIOLOGICAL EVENT OR INDICATORS OF SEPSIS "** - **NO.01062017(A)**

The following members of Ethics Committee were present in the meeting hold on **20.06.2017** conducted at Madras Medical College, Chennai 3

1. Prof.Dr.C.Rajendran, MD.,	:Chairperson
2. Prof.R.Narayana Babu,MD.,DCH., MMC,Ch-3	: Deputy Chairperson
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6. Prof.Rema Chandramohan,Prof.of Paediatrics,ICH,Chennai	: Member
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10.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person

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

The Institutional Ethics 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.

24/8

Member Secretary – Ethics Committee

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CONTENTS

INTRODUCTION

INTRODUCTION

Blunt injury abdomen causes a variety of injuries, the commonest being solid organ injury. Among the solid organs, the spleen is most commonly injured.

Operative management plays a major role in treatment of blunt injury abdomen.

Various postoperative complications can occur following emergency laparotomy including surgical site infection, abdominal abscess, urinary tract infection and lower respiratory tract infection.

Diagnosing these infections becomes particularly challenging following splenectomy because of the unusual physiological response to leucocyte count and platelet count.

The aim of this study is to assess the three risk factors i.e Total Leucocyte Count, Platelet count/Total Leucocyte Count Ratio and Injury Severity Score in patients undergoing splenectomy and to compare them with other patients undergoing laparotomies other than splenectomy for blunt injury abdomen in order to achieve a cut off value beyond which persistence of leucocytosis may denote infection.

AIMS AND OBJECTIVES

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1. To study the WBC Count and Platelet Count(PC)/WBC Count ratio in infected and non infected individuals who have undergone post traumatic splenectomy compared to other blunt abdomen trauma patients who have undergone laparotomy.
2. To study the relationship of three prognostic factors : WBC count, PC/WBC count ratio and Injury Severity Score in individuals who have undergone emergency laparotomy after trauma and their role in post operative infection.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

BLUNT TRAUMA ABDOMEN

According to WHO by the year 2020, trauma will become the first or second leading cause of “loss of productive years of life” for both developed and developing countries.[1] Blunt abdominal trauma is the third most common form of injury in road traffic accidents and the victims mostly are young, productive adults and hence it has got enormous socioeconomic impact.[2] Mortality rates are higher in patients with blunt abdominal trauma than in those with penetrating wounds, because of the lack of early diagnostic facilities and optimal management[3] Blunt injuries are thought to result from a combination of crushing, deforming, stretching and shearing forces. The magnitude of these forces directly relates to the rate of their acceleration and deceleration as well as the relative direction of impact.[4] The spleen and liver are the most commonly injured solid organs. Injuries to pancreas, bowel and mesentery, bladder, and diaphragm, retroperitoneal structures like kidneys, abdominal aorta, are less common. Injuries to the kidney and urinary bladder may be associated with pelvic fractures and retroperitoneal haemorrhage.[5]

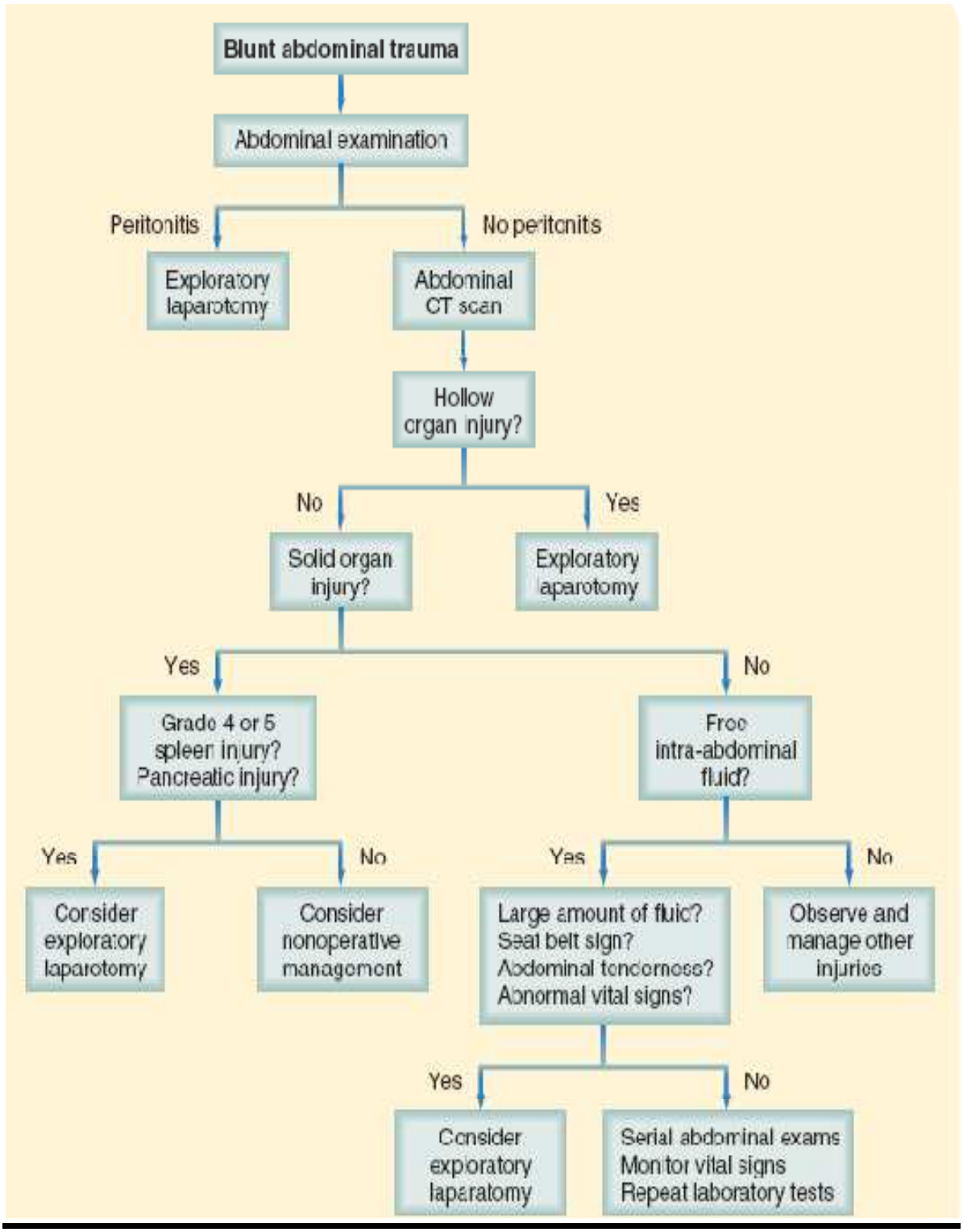
Blunt injury abdomen can be explained by 3 mechanisms:

1. DECELERATION: Rapid deceleration causes differential movement among adjacent structures leading to shear forces, causing hollow,solid, visceral organs and vascular pedicles to tear at fixed points of attachment.
2. CRUSHING: Intra abdominal contents are crushed between anterior abdominal wall and vertebral coloumn or posterior thoracic cage. Solid organs are more vulnerable.
3. EXTERNAL COMPRESSION: Direct blows or external compression against a fixed object. Causes sudden, dramatic rise in intraabdominal pressure and causes rupture of a hollow viscus (BOYLE'S LAW)

   FAST and CECT Abdomen are very useful in diagnosing extent and severity of abdominal injury.

Non operative management with careful monitoring may be considered when patient is haemodynamically stable, especially in liver injuries due to the firm architecture of liver[6].

ALGORITHM FOR EVALUATION AND MANAGEMENT OF BLUNT ABDOMINAL TRAUMA



SPLEEN

HISTORICAL BACKGROUND

Hippocrates in the fourth century bc was one of the first to write on the spleen. Hippocrates wrote of a direct connection between the brain and spleen and its particular association with the black bile.[7] Aristotle wrote about how the "hot nature" of spleen aided digestion.[8] in the early 17th century, Malphigi thought that spleen was associated with anger and was paradoxically also the "seat of laughter."[9]

The first known splenectomy was performed in 1549 on a 24 yrs old female by Adrian Zacarelli for splenomegaly. The first successful partial splenectomy was recorded in the year 1590 by Franciscus Rosetti for trauma.[10]

The total splenectomy for trauma was first done by Nicolaus Matthias in a patient whose spleen protruded through the flank wound. It was performed in Capetown, South Africa in 1678 and partial splenectomy was replaced by total splenectomy in trauma cases.[11] Judicious tamponade of the organ and its first successful suture repair was reported by Ziskoff in Russia in the year 1895 for a case of lacerated spleen.

Role of spleen in immunity has been vastly studied. In 1965

Shumacker and Kling elicited that immunity is compromised in children whose spleen has been removed in cases of hematological disorders.

EMBRYOLOGY

Development begins through the formation of the splanchnic mesodermal plate, derived from the mesoderm, at embryonic day 12. The embryonic spleen is first colonized by erythroid and myeloid progenitor cells at 2 weeks of gestation.

The spleen assumes an important hematopoietic role until the fifth month of gestation. The organ continues its differentiation and migration to the left upper quadrant, where it comes to rest with its smooth, diaphragmatic surface facing posterosuperiorly.[12]

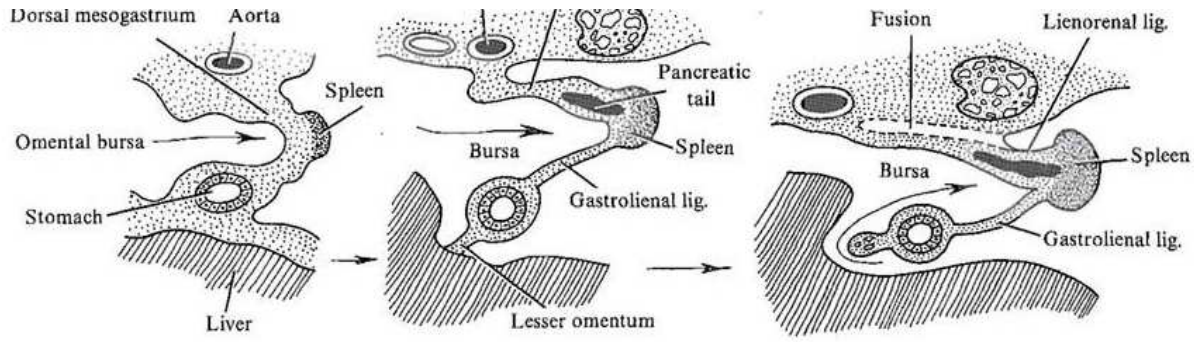


Fig 1- Development and position of spleen in dorsal mesogastrium

ANATOMY

STRUCTURE AND POSITION

In a healthy adult, spleen is approximately 12cm long and 7cm wide and weighs 75 to 100gms. It is placed deep to 9^{th}, 10^{th} and 11^{th} ribs in the posterior aspect of left upper quadrant. Its long axis is aligned along the 10^{th} rib. It has 2 surfaces: diaphragmatic and visceral. The visceral surface faces the abdominal cavity

and contains gastric, colic, renal, and pancreatic impressions. Ligaments of spleen are splenocolic ligament, gastrosplenic ligament, phrenosplenic ligament and splenorenal ligament. The gastrosplenic ligament contains the short gastric vessels; the remaining ligaments are avascular. [13]

Blood supply:

The splenic artery which is a branch of the coeliac trunk provides the main blood supply. The spleen also receives some of its blood supply from the short gastric vessels that branch from the left gastroepiploic artery running within the gastrosplenic ligament.

Venous drainage:

Major venous drainage is through splenic vein which joins superior mesenteric vein to form portal vein.

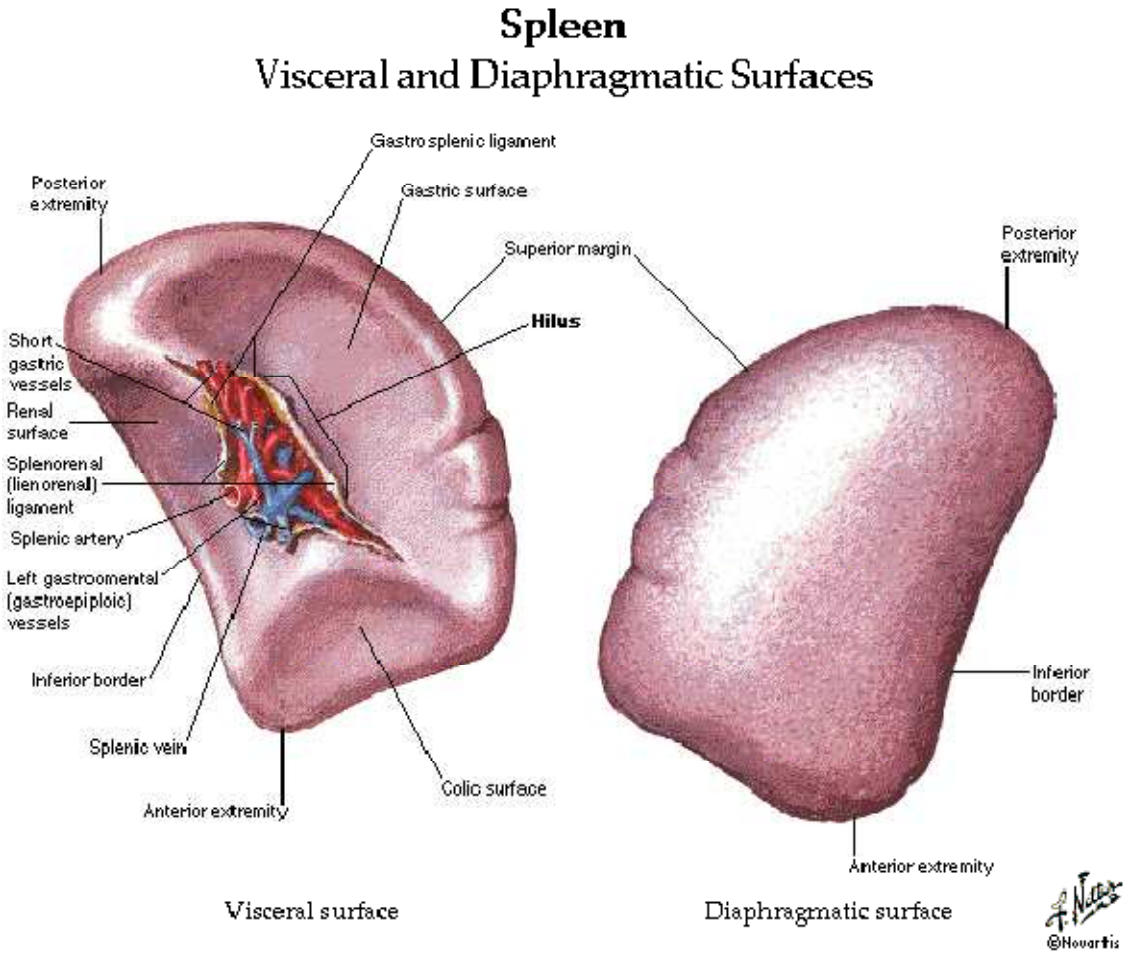


Fig 2 – Gross anatomy of spleen

The splenic parenchyma is composed of two main elements: the red pulp and the white pulp . At the interface between the red and white pulp is the narrow marginal zone. Blood enters the red pulp through cords comprised of fibroblasts and reticular fibers, which contain many macrophages and lack an endothelial lining. The blood then passes from these “open” cords to venous sinuses, which are surrounded and separated by the same reticulum, and ultimately

drains into tributaries of the splenic vein. Sinuses of the red pulp are lined

by endothelial cells. These cells contain unique stress fibers that connect the endothelial cells and that contain actin and myosin–like filaments capable of producing a sliding action. When activated, these filaments can create slits or gaps between the endothelial cells through which blood can then pass from the cords.[14] Aging erythrocytes with stiffer membranes get stuck trying to pass into the sinus and are phagocytized by macrophages within the red pulp.[15]

Around the terminal part of splenic arterioles, a periarticular lymphatic sheath is present comprised of T lymphocytes and intermittent aggregations of B lymphocytes or lymphoid follicles. When antigenically stimulated, the follicles, serving as centers of lymphocyte proliferation, develop germinal centers, which regress as the stimulus or infection subsides.

PHYSIOLOGY AND PATHOPHYSIOLOGY

Spleen has 2 major functions:

- Cellular
- Immunological

CELLULAR: Major site of extra medullary Haematopoiesis, storage, removal of Heinz bodies, Howell-Jolly bodies, and hemosiderin granules, removal of aged or abnormal red cells.

Spleen is the most important site of selective erythrocyte sequestration.

There is an accountable relation between the platelets and splenic cells. Normally, about one third of the platelet mass is pooled in the spleen, and this pool exchanges freely with the circulating platelets that have a life span of about 10 days. This is the main reason for thrombocytosis in a post splenectomy patient.

IMMUNOLOGICAL: Both innate and adaptive immune responses occur within the spleen. It plays a major role in Generation of lymphocytes, production of propredin, opsonin, tuftsin, interferon and antibody synthesis (IgM).

This is the reason for the risk posed by pneumococcus and *Haemophilus influenzae* to an asplenic patient.[16-18]

Indications for splenectomy

Trauma

- Accidental
- Operative

Oncological

- Part of *en bloc* resection
- Diagnostic
- Therapeutic

Haematological

- Spherocytosis
- Purpura (ITP)
- Hypersplenism
- Portal hypertension
- Variceal surgery

SPLENIC INJURY

The spleen is the most commonly injured abdominal organ in trauma with 23.8% of abdominal trauma patients demonstrating splenic injuries. Many splenic

injuries are self-limited, demonstrating no evidence of ongoing bleeding; others require splenectomy, which in most cases is straightforward. Despite this, the mortality after blunt splenic injury is 9.3%. Direct compression of

the spleen with parenchymal fracture is the most common pathophysiologic

mechanism followed by rapid deceleration.Spleen is the most commonly bleeding intra-abdominal organ, as noted in unstable patients with intra-abdominal fluid on focused assessment with sonography in trauma (FAST). Splenic injuries are identified during laparotomy in unstable patients taken to operating room emergently. In stable patients, the mainstay of diagnosing splenic injuries is by abdominal CT with IV contrast.

Splenic injuries may appear as disruptions in the normal splenic parenchyma, with surrounding hematoma and free intra-abdominal blood. On occasion, active bleeding can be identified by visualizing extravasation of contrast material that appears as a highdensity blush or accumulation of contrast-laden blood.

Splenic injuries may also present as subcapsular haematomas, pseudoaneurysm or complete devascularisation at the hilum.

Penetrating splenic trauma is less common but is still present in 8.5% of all penetrating abdominal injuries in the National Trauma Data Bank.

Splenic injuries are graded by the American Association for the Surgery of Trauma organ injury scaling system, which relies on the parenchymal or subcapsular characteristics and the vascular involvement.

Splenic injury patients who are haemodynamically stable may be managed by conservative management or by angiography and selective embolisation.

AAST SPLEEN INJURY SCALE

INJURY GRADE	INJURY TYPE	DESCRIPTION OF INJURY
I	Hematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1 cm parenchymal depth
II	Hematoma	Subcapsular, 10% to 50% surface area; intraparenchymal, <5 cm in diameter
	Laceration	Capsular tear, 1 to 3 cm parenchymal depth that does not involve a trabecular vessel
III	Hematoma	Subcapsular, >50% surface area or expanding; ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma ≥5 cm or expanding
	Laceration	>3 cm parenchymal depth or involving trabecular vessels
IV	Laceration	Laceration involving segmental or hilar vessels producing major devascularization (>25% of spleen
V	Hematoma	Completely shattered spleen
	Laceration	Hilar vascular injury devascularizes spleen

Table 1 : AAST grading of splenic injuries.

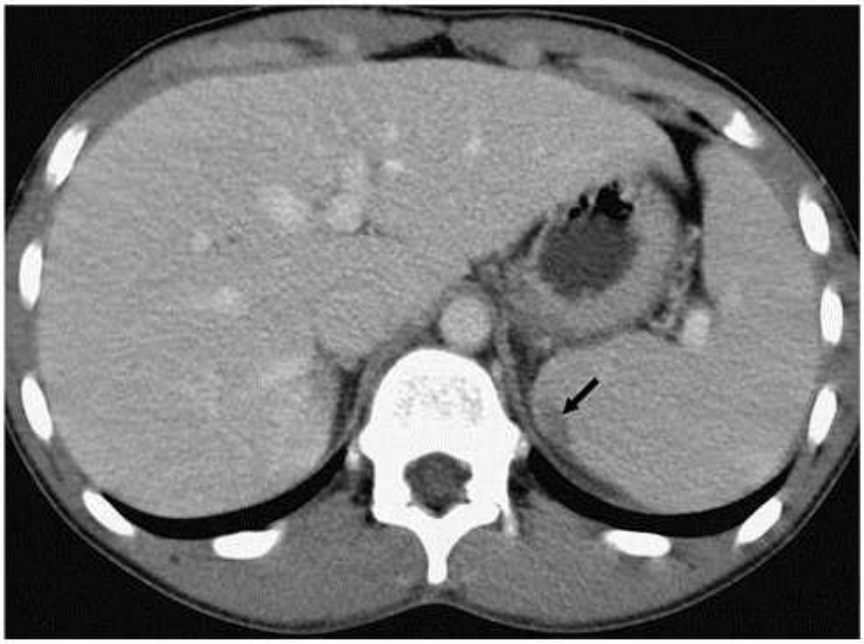


Fig 3 : Grade I: sub-capsular fluid involving < 10% of the splenic surface.

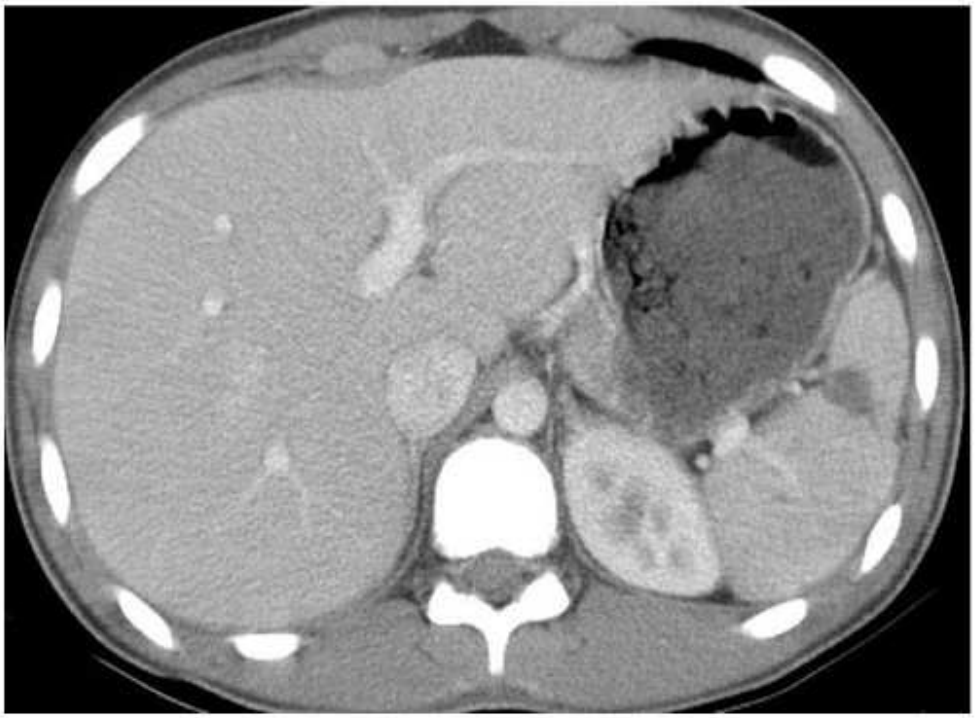


Fig 4- Grade I: sub-capsular fluid involving < 10% of the splenicsurface. Capsular tear < 1cm depth.

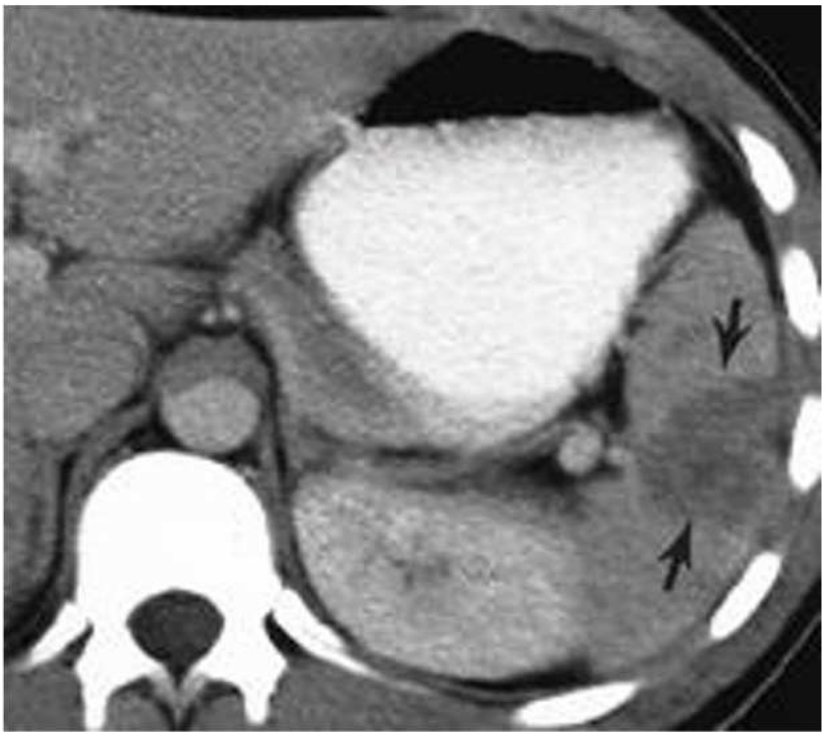


Fig 5- Grade II: sub-capsular hematoma, 10% to 50% surface area; intra-parenchymal hematoma, < 5 cm in diameter.

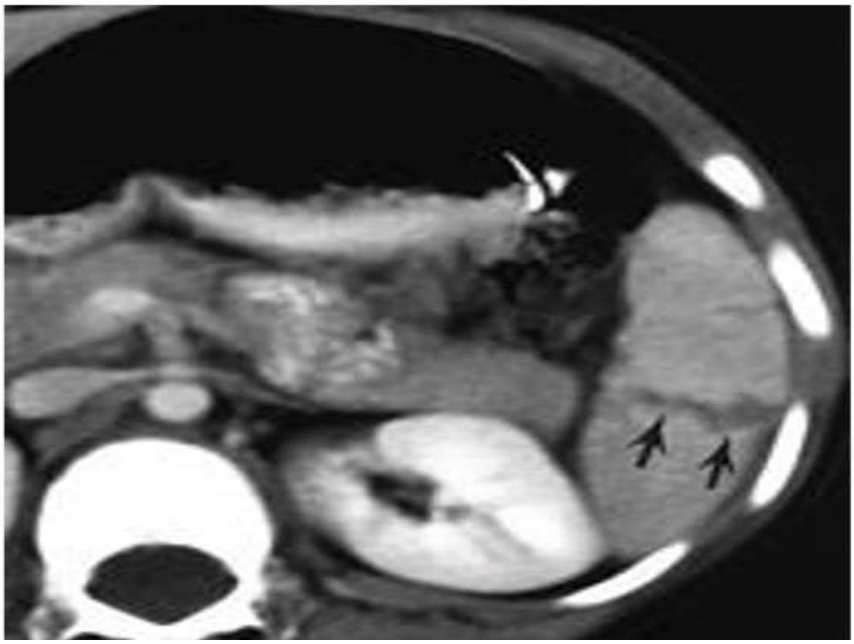


Fig 6- Grade II: capsular tear, 1 to 3 cm parenchymal depth that does not involve a trabecular vessel.

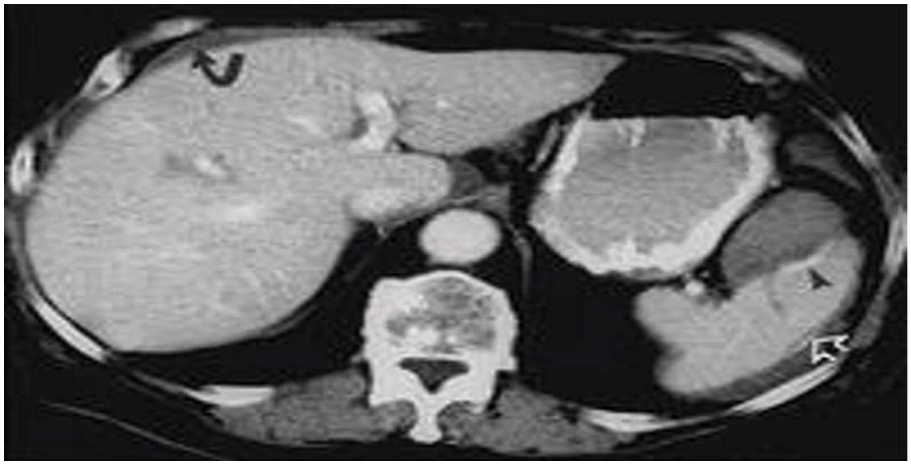


Fig 7- Grade III: sub-capsular hematoma, laceration and subcapsular contrast extravasation.

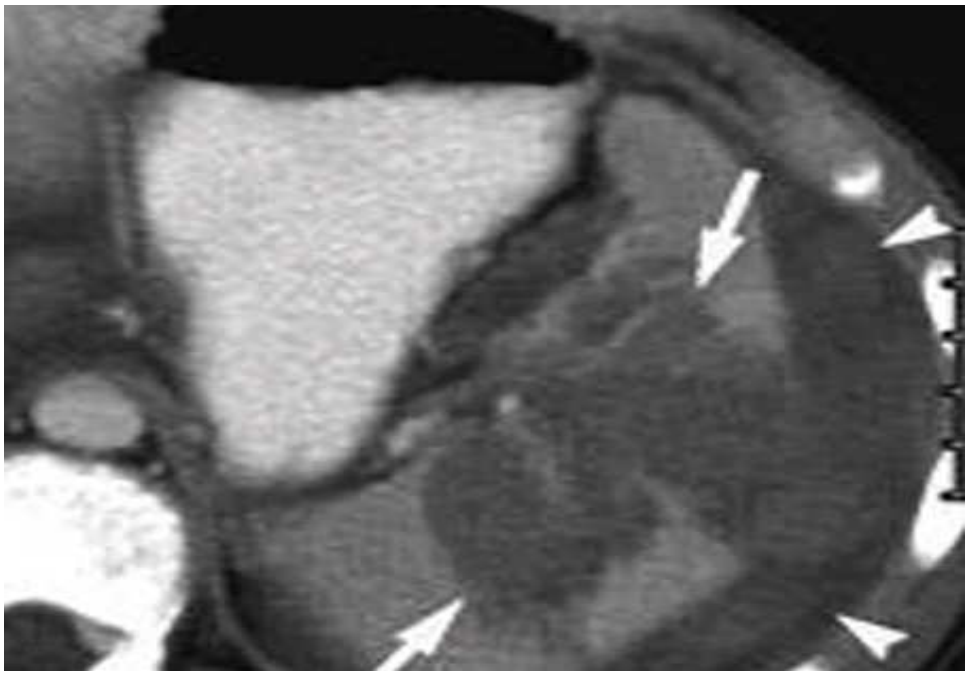


Fig 8- Grade III: laceration of more than 3 cm in depth radiating from the splenic hilum.

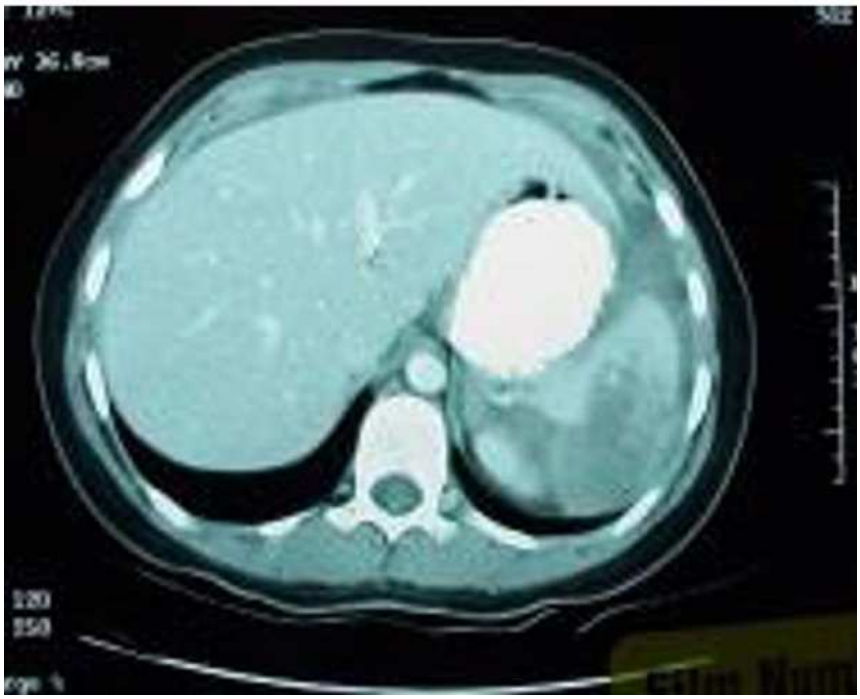


Fig 9- Grade IV: laceration involving segmental or hilar vessels producing major devascularization (>25% of spleen).

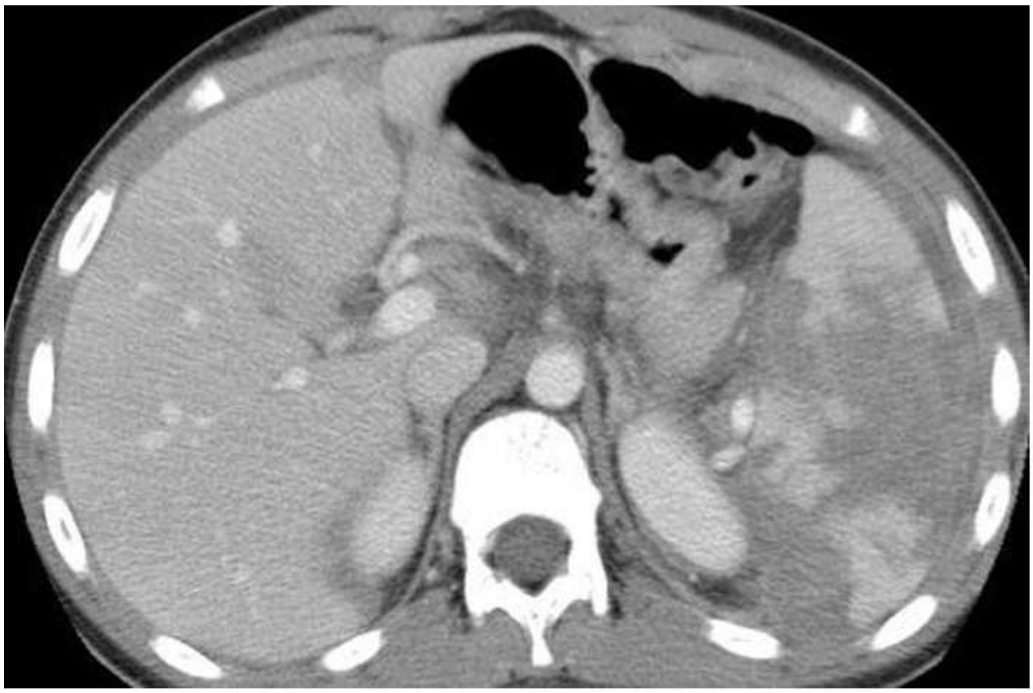


Fig 10- Grade V: shattered spleen and hilar vascular injury.

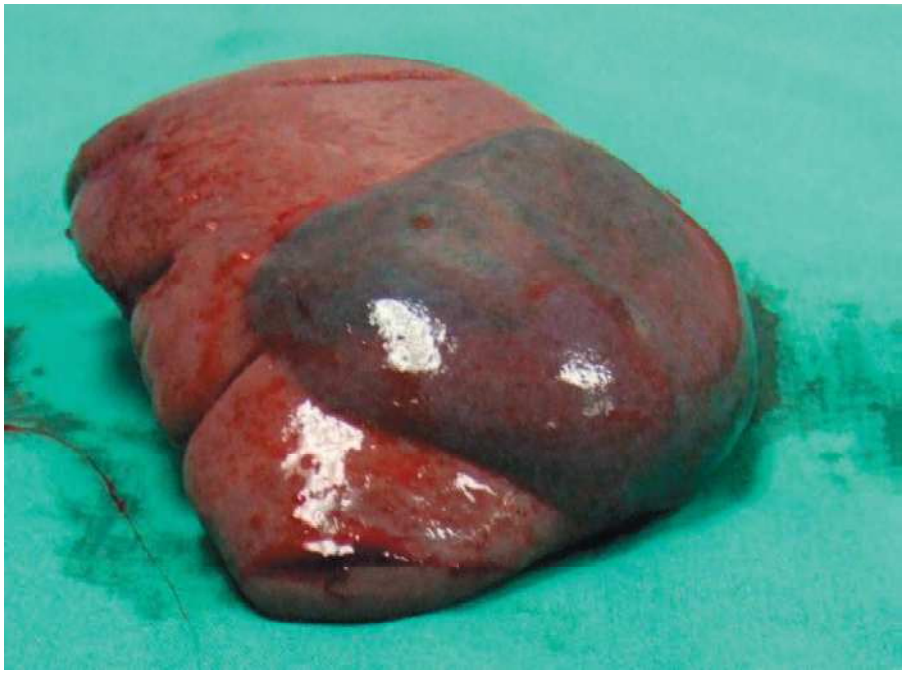


Fig 11- Splenic injury with sub-capsular hematoma. Despite only a 1-cm capsular tear, this injury demonstrated ongoing hemorrhage.

Non operative management of splenic injuries is strictly restricted to haemodynamically stable patients with no physiologic indication of ongoing blood loss. Physiologic stability includes , lack of tachycardia, a normal blood pressure, no physical examination findings indicating shock, and absence of metabolic acidosis. It is more labour intensive than operative management due to the need for intensive monitoring of the patient. Until intravascular equilibrium occurs, the haemoglobin levels cannot reflect the blood loss. Candidates with mild hemodynamic instability, but responding to crystalloid infusion can be considered for non-operative management. Non-operative management is reserved for grade I, II injuries and isolated grade III injuries.

Angiography and selective embolization represent the recent advance in management of splenic injury. One major benefit of angiography is the potential to obstruct sites of bleeding endovascularly by angioembolization. Stable patients who are found to have a pseudoaneurysm on CT may benefit from angioembolization to eliminate blood flow through the injured segment of spleen.

Haemodynamic instability at presentation or failure of conservative management is the indication for operative management.

OPERATIVE PROCEDURE : OPEN SPLENECTOMY

A Midline laparotomy incision is made with packing of all four quadrants in an unstable patient. A retractor is used to expose left upper quadrant. The beginning of splenectomy is marked by retracting the spleen posteromedially to visualize retroperitoneal attachments and dividing the peritoneum laterally. Division begins at the white line of Toldt and continued superiorly till short gastric vessels are exposed. After dividing the peritoneum, a blunt plane is created posterior to the spleen in a medial direction, extending behind the tail of the pancreas, mobilizing the entire spleen and distal pancreas, allowing the spleen to be delivered up into the wound. Short gatric vessels are ligated and divided taking great care to avoid greater curvature of stomach. Hilar vessels are then clamped and ligated and splenectomy done taking great care to avoid

injuring tail of pancreas. Drain is kept only when injury to tail of pancreas is suspected. Postsplenectomy

vaccines must be provided to ensure protection from encapsulated

bacteria, including Streptococcus pneumoniae, Neisseria meningitidis,

and Haemophilus influenzae.

Since patients who usually benefit from splenic salvage techniques are managed nonoperatively, these techniques are less commonly used now.

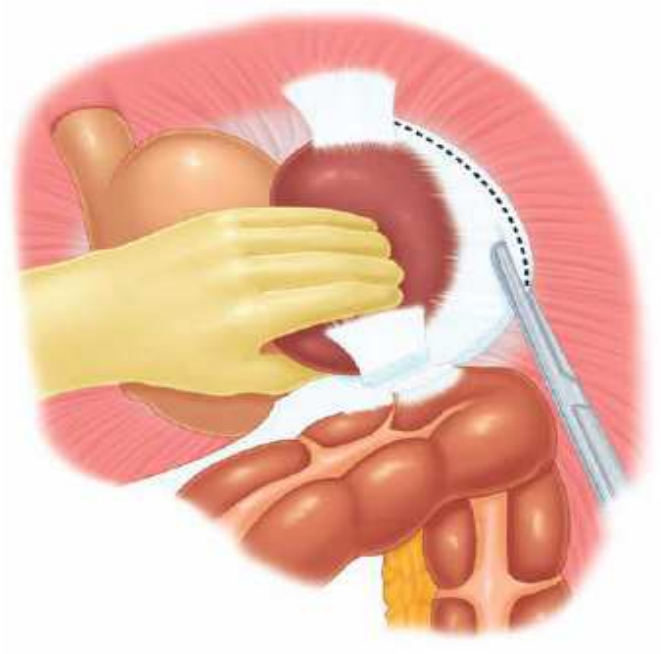


FIG 12: Splenocolic ligament divided at the beginning of splenectomy

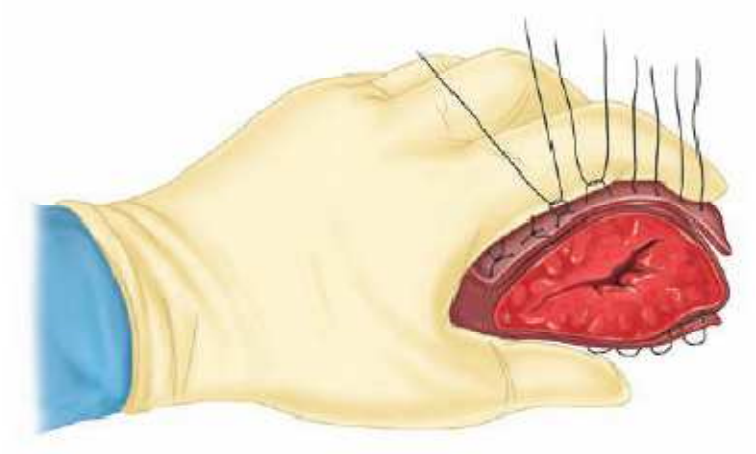


Fig 13: Interrupted pledgeted sutures may effectively control hemorrhage from the cut edge of the spleen.

POST SPLENECTOMY COMPLICATIONS

- Haemorrhage: Slippage of ligature
- Left basal atelectasis and pleural effusion.
- Injury to stomach: 1. Gastric mucosal damage causing haematemesis
  2.Injury to greater curvature causing fistula
- Injury to tail of pancreas causing pancreatitis, abscess,fistula
- Thrombocytosis leading to Deep vein thrombosis and pulmonary embolism.
- Overwhelming Post Splenectomy Infection : Capsulated organisms like Streptococcus pneumoniae, Neisseria meningitides, Haemophilus influenzae and Escherichia coli.

POST SPLENECTOMY LEUCOCYTOSIS

Mounting of an efficient immune response requires fast mobilization and distribution of lymphocytes. The kinetic aspects of lymphocyte trafficking is responsible for postsplenectomy leukocytosis. Lymphocytes cross from blood across a specialized endothelium of post capillary venule into lymph node and exit via efferent lymphatica and thoracic duct.[19,20] Most lymphocytes circulate through spleen because direct access to marginal zone bypasses the specialized endothelium.

The total lymphocyte pool in is estimated to be approximately $50*10^{10}$ cells of which $15*10^{10}$ circulate through lymphoid tissues[21]. At any given time only $1*10^{10}$ lymphocytes circulate in the blood with a short transit time of 20-36minutes for exchangeable cells resulting in high turnover of migratory lymphocytes about 50 times per day[22,23] and total daily exchange of $50* 10^{10}$ between blood and tissues. The spleen has highest lymphocyte uptake of about 40% during the early stages of recirculation. [24] During intermediate and late stages of recirculation, lymphocytes are predominantly found in lymph nodes. The transit time of migrating lymphocytes through the spleen is significantly shorter than through lymph nodes and a higher number of lymphocytes pass through the spleen than through the thoracic duct.

Hence splenectomy results in a slower overall clearance of lymphocytes from peripheral blood. Moreover neutrophils are destroyed in spleen. Hence, splenectomy may result in neutrophilia. As a result of slower clearance of lymphocytes and reduced destruction of neutrophils, there is physiological leukocytosis post-splenectomy .

POST SPLENECTOMY THROMBOCYTOSIS

Spleen is the major site where destruction of platelets takes place. Hence, there is physiological thrombocytosis following splenectomy.

LIVER INJURY

Within the National Trauma Data Bank, liver injuries occurred in 3.0% of all patients, whereas 22.2% of patients with blunt mechanisms sustained

hepatic trauma, making it the second most common organ to be injured in trauma.

Mechanisms of blunt hepatic trauma include compression with direct parenchymal damage and shearing forces, which tear hepatic tissue and disrupt vascular and ligamentous attachments.

Liver injuries are mostly first diagnosed on entering the abdomen in the unstable patient explored for free fluid on FAST examination.

Those who do not require immediate operation should be imaged with abdominal

Contrast enhanced CT, which is capable of providing excellent anatomic detail that allows highly accurate characterization of injuries. Common findings on CT indicative of liver injury include disruption of the hepatic parenchyma with perihepatic blood or hematoma and hemoperitoneum.

Bleeding from the liver can be seen on CT as extravasation of contrast material either within the liver parenchyma or into the peritoneal space.

Depending on CT findings, liver injuries are classified according to AAST classification.

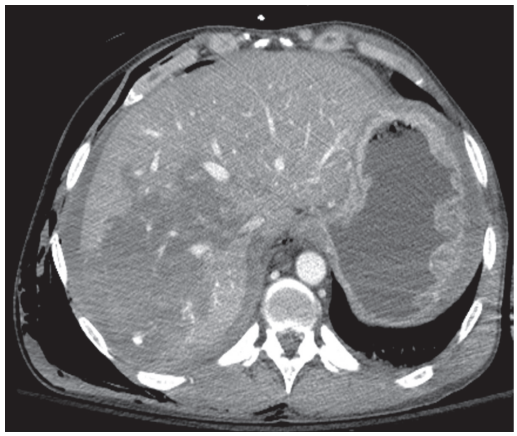
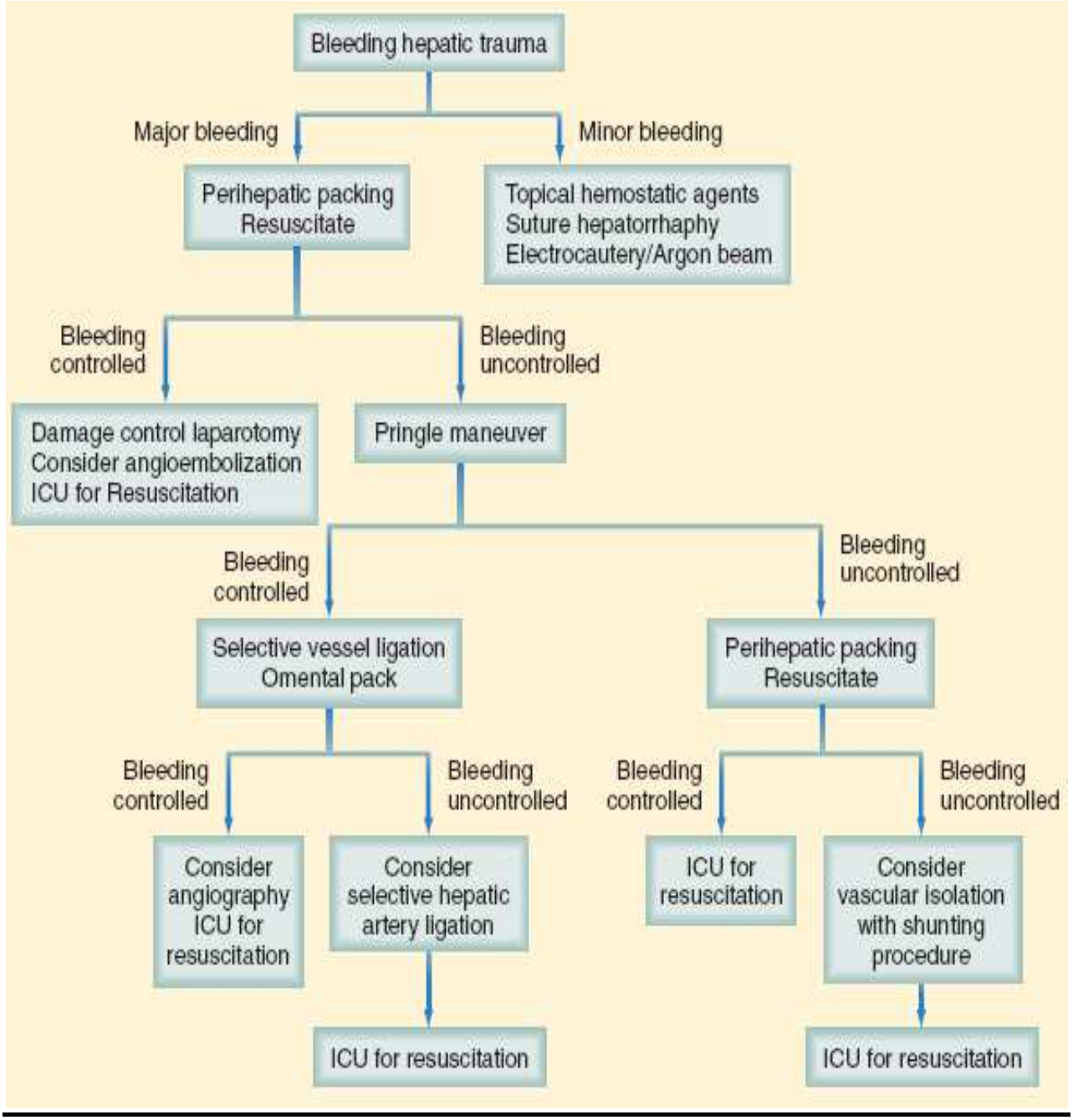


Fig 14: Grade IV liver laceration involving the right hepatic lobe on abdominal CT. Note the focus of active extravasation of contrast material within the injured liver parenchyma at the periphery of the injury as identified by the *arrow*.

AAST LIVER INJURY SCALE

INJURY GRADE	INJURY TYPE	DESCRIPTION OF INJURY
I	Hematoma	Subcapsular, <10% surface area
	Laceration	Capsular tear, <1 cm parenchymal depth
II	Hematoma	Subcapsular, 10% to 50% surface area; intraparenchymal, <10 cm in diameter
	Laceration	Capsular tear, 1 to 3 cm parenchymal depth, <10 cm in length
III	Hematoma	Subcapsular, >50% surface area of ruptured subcapsular or parenchymal hematoma; intraparenchymal hematoma >10 cm or expanding
	Laceration	>3 cm parenchymal depth
IV	Laceration	Parenchymal disruption involving 25% to 75% hepatic lobe or 1 to 3 Couinaud segments
V	Laceration	Parenchymal disruption involving >75% of hepatic lobe or >3 Couinaud segments within a single lobe
	Vascular	Juxtahepatic venous injuries (i.e., retrohepatic vena cava/central major hepatic veins)
VI	Vascular	Hepatic avulsion

OPERATIVE MANAGEMENT OF LIVER TRAUMA



ABBREVIATED INJURY SCALE[24]

Created by the Association for Advancement of Automotive Medicine.

The score describes three aspects of injury

- Type
- Location
- Severity

  It is denoted by seven numbers ranging from 1-7.

  1. Body region
  2. Type of anatomical structure

     3,4. Specific anatomical structure

     5,6. Level

     7. Severity of score

Severity of score is classified as:

AIS 1 : Minor

AIS 2 : Moderate

AIS 3 : Serious

AIS 4 : Severe

AIS 5 : Critical

AIS 6 : Maximal(Currently untreatable)

INJURY SEVERITY SCORE (ISS) & NEW INJURY SEVERITY SCORE (NISS)

It is based on AIS and correlates with mortality, morbidity and other measures of severity.

It is calculated as the sum of the squares of the highest AIS scores in each of the three most injured body regions;

- Head or neck
- Face
- Chest
- Abdominal or pelvic contents.
- Extremities or pelvic girdle.
- External

  Score ranges from 1-75. If an injury is assigned an AIS score of 6, the ISS score is automatically assigned as 75.

ISS

SCORE	SEVERITY
1-8	MINOR
9-15	MODERATE
16-24	SERIOUS
25-49	SEVERE
50-74	CRITICAL
75	MAXIMUM

Since multiple injuries within the same body region are given a single score, a modification of the ISS, the "New Injury Severity Score" (NISS), has been given shape.

Three most severely injures organs are assigned a score and their sum of squares will yield the New Injury Severity Score.

POST OPERATIVE INFECTIONS

Postoperative infection includes any infection that affects a post operative patient and not just those that require an surgical intervention as thought earlier.

The most common infections are:

- Surgical site infections
- Hospital or ventilator acquired pneumonia

- Aspiration pneumonitis post endotracheal intubation
- Urinary tract infection
- Central line associated blood stream infections

Surgery's inherent invasiveness creates portals of entry for pathogens to invade the host through natural epithelial barriers.

Surgical illness is immunosuppressive (e.g., trauma, burns, malignant tumors), similar to therapeutic immunosuppression after solid organ transplantation.

Postoperative infections are easier to prevent than to treat and it is prudent for every physician who is in contact with his patient, to take strict aseptic precautions.

Universal hand washing techniques should be practiced.

Wound dressing must be done under strict aseptic precautions. Drains and catheters should be avoided as much as possible. If unavoidable, must be removed as early as possible. Judicious use of prophylactic and therapeutic antibiotics is necessary to maintain the balance between preventing infection and multidrug resistance.

RISK FACTORS :

HOST FACTORS:

The host is defined by genotype, expressed phenotypically as characteristic traits. Innate immunity provides continuous surveillance against tissue invasion by foreign antigens in the interstitial spaces just beneath epithelial barriers.

Innate immunity is responsible for providing epithelial barrier

preventing the invasion of foreign antigens. Even though commensals are present throughout the body, infection occurs only when there is a portal of entry through a breech in epithelial barrier like surgery or intravenous catheter insertion. Surgical stress produces cortisol which further reduces immunity. Injury also stimulates a repair response (inflammation), which may cause a wide-ranging autodestructive augmentation of the inflammatory response.

Older age (age ≥65 years) is a definite risk factor for adverse outcomes from infection, related to immune senescence and an increased incidence of nosocomial infection. Even transient Hyperglycemia induces immune cell dysfunction and is a major risk factor for infection.

STRESS RESPONSE TO INJURY
Activation of the autonomic nervous system
Peripheral insulin resistance
Activation of hypophyseal-pituitary-adrenal axis
Production of reactive oxygen and nitrogen intermediates

Production of proinflammatory and anti-inflammatory cytokines and lipid mediators
Acute-phase changes of hepatic protein synthesis
Recruitment and activation of neutrophils, monocytes-macrophages, and lymphocytes
Upregulation of procoagulant activity

Factors causing increased risk of post operative infections

• Extremes of age (neonates, very old adults)

• Malnutrition

• Obesity

• Diabetes mellitus

• Prior site irradiation

• Hypothermia

• Hypoxemia

• Coexisting infection remote to surgical site

• Corticosteroid therapy

• Recent operation, especially of chest or abdomen

• Chronic inflammation

• Hypocholesterolemia

Genetics and Genomics of Trauma and Sepsis

No studies have so far shown a sex predeliction for infection and sepsis.[25,26]

Genomic variability may correlate with disease susceptibility in infections. Nucleotide structures of genes containing single nucleotide polymorphisms (SNPs) and single point mutations related to inflammation like tumor necrosis factor-α [TNF-α], interleukin [IL]-1, IL-6, and IL-8), the anti-inflammatory factors (e.g., IL-10, IL-1 receptor antagonist), the innate immune receptor (e.g., Toll-like receptor 4), and the coagulation system (e.g., factor V, plasminogen activator inhibitor-1) have been associated with a predisposition to sepsis.[27]

Due to heterogenicity of infection and the response mounted against it, it is difficult to pinpoint a single nucleotide pleomorphism to characterize increased risk of infection in an individual.

INTERACTION BETWEEN HOST AND THERAPY

Risk of infection is increased by the following factors:

- Injury itself.
- Impairment of host defences
- Resuscitation
- Definitive care

Hypothermia

Hypothermia may occur due to evaporative loss in a large exposed wound or intracavitary surgery, resuscitation with unwarmed iv fluids and blood products and exposure. Hypothermia leads to vasoconstriction and decreased microcirculation, which is aggravated by hypovolemia, inflammatory response, coagulation pathway decreased transfused red cell deformability[28,29]

This affects cardiovascular performance and decreases immunity leading to post operative morbidity and mortality.

Tissue hypoxia

Tissue hypoxia predisposes to Surgical site infection.

It occurs due to massive trauma to face, chest,airway or lung, massive blood loss, Acute Respiratory Distress Syndrome and caediovascular instability.[30] Supplemental oxygen decreases risk of surgical site infection.[31]

RESUSCITATION

Injudicious resuscitation can have as many adverse outcomes than insufficient resuscitation. The amount and nature of fluid to be administered has to be calculated meticulously. Earlier crystalloids were preferred over colloids due to cost effectiveness and almost equal effectiveness[32]. Now resuscitation with colloids show less mortality.[33] Resuscitation of immune system is the most important factor, failure of which can lead to increased mortality.[34]

BLOOD TRANSFUSION

Though blood transfusion is life saving in trauma, it is associated with increased exponential risk of infection even after a single transfusion and becoming near certainity after 15 units of transfused blood products.[35,36] Altered leukocyte antigen presentation and a shift to the T helper 2 cell phenotype causes immuno-suppression following blood transfusion.

Risk of infection following blood transfusion is increased 3 times in surgical patients and 5 times in trauma patients. Critically ill and ventilator dependant patients are also at a higher risk of infection following blood transfusion.[37]

Prolonged storage of banked blood causes loss of high energy membrane phosphates which leads to impaired red cell deformability, disruption of microcirculation, and impaired oxygen delivery[38].

As a result blood transfusion does not increase oxygen consumption [39] but instead increase organ dysfunction [40]. So it is better to be conservative while deciding on blood transfusion to stable patients in intensive care unit[41]

BLOOD SUGAR CONTROL

Hyperglycemia reflects catabolism and insulin resistance assosciated with surgical stress and also impairs host immune defence. Inadequate glycemic control during the peri operative period increases the risk of infection and worsens the outcome from sepsis in both diabetic and non diabetics. Blood glucose more than 200 mg/dl is associated with four times increased risk of surgical site infection.

Blood glucose level below 110 mg/dL is associated with a 40% decrease in mortality among critically ill postoperative patients and also fewer nosocomial infections and less organ dysfunction. [42]

Effects of Hyperglycemia on Immune Cell Function

- Decreased respiratory burst of alveolar macrophages
- Decreased insulin-stimulated chemokinesis
- Glucose-induced protein kinase C activation
- Increased adherence
- Increased adhesion molecule generation
- Spontaneous activation of neutrophils

Effects of Stress Response on Carbohydrate Metabolism

- Enhanced peripheral glucose uptake
- Hyperlactatemia
- Increased gluconeogenesis
- Depressed glycogenolysis
- Peripheral insulin resistance

In order to reduce risk of hypoglycemia following insulin therapy, the maintenance blood glucose level has been increased from 110 mg/dl to 140-180 mg/dl[43].

NUTRITION

Nutrition plays a vital role in preventing post operative morbidity and mortality. In order to convert the catabolic state of surgical stress back to anabolism, excess calories of 25-30kcal/day and 1g nitrogen/kg/day is required in excess of basal requirements. It is challenging to provide adequate calories and protein while simultaneously avoiding hyperglycemia. Parenteral nutrition may offer no advantage over not feeding the patient at all due to catheter related infection and hyperglycemia. Early enteral nutrition within 48 hrs is preffered and id found to reduce infections.[44,45].

CONTROL OF INFECTION

General principles of surgical care, critical care, and infection

control must be adhered to at all times.

Resuscitation must be precise and rapid; Both overresuscitation and underresuscitation increase the risk of infection. It is also necessary to immediately identify the underlying pathology and take measures to treat it. Central venous catheters placed under sub optimal conditions, urinary catheters and drains are all sources of infection and must be identified and rectified immediately. Infection control is an individual and collective responsibility.

Hand hygiene is the most effective means to reduce the spread of infection, but compliance is a continual challenge.[46] Alcohol gel

hand cleansers are effective,[47] except against the spores of *Clostridium*

difficile, which requires cleansing with soap and water.[48]

Universal precautions—cap, mask, gown, gloves, and protective eyewear—must be observed whenever there is a risk of splashing of body fluids.

Endogenous flora are the most common source of infection. Skin surfaces, artificial airways, gut lumen, wounds, catheters, and inanimate surfaces (e.g., bed rails, computer terminals)[49] may become colonized. Any break in natural epithelial barriers (e.g., incisions, percutaneous catheters, airway or urinary catheters) creates a portal of entry for invasion of pathogens. The fecal-oral route is the most common manner whereby pathogens reach the portal, but health care workers facilitate the transmission of pathogens on their hands. Contact isolation is an important part of infection control and should be used selectively to prevent the spread of pathogens such as methicillin-resistant *Staphylococcus aureus* (MRSA), vancomycinresistant enterococci (VRE), or MDR gram-negative bacilli.

However, contact isolation may decrease the amount of direct patient contact.[50] An appropriate balance must be struck because reduced nurse staffing of ICUs has been independently associated with an increased risk of a number of nosocomial infections.[51]

CATHETER CARE:

Optimal catheter care includes:

- Insertion only when necessary.

- Appropriate skin preparation and barrier protection
- Appropriate catheter selection (antiseptic or antimicrobial coated)
- Proper dressing of indwelling catheters
- Removal as soon as no longer needed, or as is practicable, but no longer than 24 hours after insertion under less than ideal circumstances (e.g.,trauma bay, cardiac resuscitation).

High risk catheters are nontunneled central venous catheters and pulmonary artery catheters pose the highest risk for infection. Other catheters associated with increased risk of infection includes endotracheal tubes, intercostal thoracostomy

catheters, ventriculostomy catheters for intracranial pressure monitoring and urinary bladder catheters.

Risk of pneumonia increases by 1% to 3% for every day of mechanical ventilation and endotracheal intubation [51] .

Most common skin antiseptic that is used is chlorhexidine gluconate,

a phenolic biguanide derivative, in concentrations of 0.5% to 4% alone or in lower concentrations in combination with an alcohol. This antiseptic has cidal activity i.e., bactericidal, viricidal and fungicidal which is slow but persistent. Chlorhexidine has been most commonly used for vascular catheter insertion and it has been found to be superior to povidone iodine solution. It is also being recommended for surgical site preparation, topical bathing of critically ill patients and as an antiseptic coating for indwelling

catheters. For microbicidal effect of povidone iodine solution, one must apply the solution and allow it to dry. Unless a mucous membrane has to be prepared its use has been discouraged [52, 53, 54, 55, 56]

It is mandatory to have full barrier precautions during bedside catheterization procedures except for arterial and urinary bladder catheterization for which sterile gloves and field is more than enough if maintained meticulously.

If a central venous catheter is inserted under suboptimal conditions, then ensure that the catheter is changed to a different site as soon as patients' hemodynamic condition improves, but not more than 24 hours of insertion.

A stat dose of first generation cephalosporin does prevent infections following tube thoracostomy or ventriculostomy, but is not indicated for vascular or bladder catheterization.

It is necessary to maintain dressings carefully which becomes challenging in cases of agitated patients and irregular body surface.

Mentioning the date and time of dressing change over the dressing itself is simple and effective.

One should not shift dressing cart from patient to patient, instead sufficient dressing materials should be kept in the patient's room. Inanimate fomites such as scissors can transmit pathogens from one patient to another.

Hence it is prudent to implement care bundles and catheter care teams to reduce the risk of catheter line associated bloodstream infections and urinary

tract infections [57, 58].

Catheter choice also plays an important role in reducing the risk of infection related to endotracheal tubes, central venous catheters and urinary catheters. Those areas that cannot be reached by routine endotracheal suctioning such as the sub-glottic region may be cleared by continuous aspiration of sub-glottic secretions through an endotracheal tube provided with an extra lumen which opens to the airway just above the balloon.

Continuos aspiration of subglottic secretions decrease the incidence of ventilator associated pneumonia by 50%. Endotracheal tubes impregnated with silver are highly effective in reducing the risk of ventilator associated pneumonia and mortality. Catheter related infections in high prevalence units can be reduced by antibiotic coated or antiseptic coated tubes. Silver coated urinary catheters are associated with decreased incidence of catheter related bacterial cystitis [59, 60].

SPECIFIC INFECTIONS

SURGICAL SITE INFECTIONS:

Surgical procedures are classified into

Clean procedures - affect only skin structures and other soft tissues.

Clean contaminated procedures- open a hollow viscus under controlled circumstances (e.g., elective aerodigestive or genitourinary tract surgery)

Contaminated procedures- introduce a large inoculum of bacteria into a normally

sterile body cavity, but too briefly for infection to become established

during surgery (e.g., penetrating abdominal trauma, enterotomy

during adhesiolysis for mechanical bowel obstruction).

Dirty procedures - performed to control established infection

(e.g., colon resection for perforated diverticulitis)

RISK FACTORS FOR DEVELOPING SURGICAL SITE INFECTIONS

Patient Factors

Ascites (for abdominal surgery)

Chronic inflammation

Corticosteroid therapy

Obesity

Diabetes

Extremes of age

Hypocholesterolemia

Hypoxemia

Peripheral vascular disease (for lower extremity surgery)

Postoperative anemia

Prior site irradiation

Recent operation

Remote infection

Skin or nasal carriage of staphylococci

Skin disease in the area of infection (e.g., psoriasis)

Undernutrition

Environmental Factors

Contaminated medications

Inadequate disinfection or sterilization

Inadequate skin antisepsis

Inadequate ventilation

Treatment Factors

Drains

Emergency procedure

Hypothermia

Inadequate antibiotic prophylaxis

Oxygenation (controversial)

Prolonged preoperative hospitalization

Prolonged operative time

Factors determining microbiology of surgical site infections include the nature of the procedure, whether a body cavity or a hollow viscus is entered during surgery and location of the incision. Most surgical site infections are the result of microorganisms that enter through the surgical incision wound. Hence the most common organism responsible for surgical site infection includes all the gram positive organisms - Staphylococcus

epidermidis, Staphylococcus aureus, and Enterococcus species. For those surgeries that are done through infrainguinal incision and intracavitatory surgery, gram negative organisms such as Escherichia coli and Klebsiella spp are the most common pathogens. Anaerobic organisms are the potential pathogens in pharynx, female genitourinary and lower gastrointestinal surgeries. Hence antibiotic prophylaxis must be directed appropriately against these antigens. Statistics indicate that the incidence of surgical site infections vary from less than 5% for clean surgeries to about 20% for dirty procedures.

The factors included under National Nosocomial Infections Surveillance System (NNIS) and its successor program, the National Healthcare Safety Network (NHSN) is

1. Wound classification
2. ASA class 3 or higher
3. Prolonged operative time, where time is longer than the 75^{th} percentile for the given procedure [61, 62, 63].

According to NNIS-NHSN risk of surgical site infection increases with increase in the number of risk factors irrespective of the type of surgery performed. Laparoscopic surgeries are associated with decreased incidence of surgical site infection. Factors responsible for decreased incidence of surgical site infection includes decreased wound size, limited use of cautery in the abdominal wall and a diminished stress response to tissue injury.

Hypothermia is another important risk factor for surgical site infection which occurs because of water loss due to evaporation, administration of normothermic fluids and other factors [64].

Controversies exist whether peri-operative oxygen administration is a boon for infection prevention [65]. The ischemic milieu of fresh surgical incision is vulnerable to bacterial invasion. Moreover administration of oxygen is found to have a beneficial antibacterial effect. Though there are no convincing studies to suggest the usefulness of oxygen in preventing surgical site infections, but there exists one meta-analysis suggesting the advantage of oxygen in reducing the risk of infection.

It has been found that drains instead of preventing infections, is seen to increase the risk of infection. Drains prevent wound epithelialisation and become a conduit, creating a portal of entry for the pathogens that has been colonising the skin. Several studies conducted on placing the drains in clean or clean contaminated procedures has shown that they increase the chances of infection rather than decreasing the risk [66, 67].

Surgical Infection Prevention Project was incorporated into Surgical Care Improvement Project with additional recommendations which includes the following

- Antibiotic Prophylaxis
- Glucose Control
- Hair Removal

- Hypothermia

POST OPERATIVE PNEUMONIA

Post-operative patients especially patients requiring ventilators are susceptible to pneumonia. Ventilator associated pneumonia (VAP) is defined As pneumonia presenting 48-72 hours after intubation.

Early onset.

Occurring within 5 days of intubation.

Commonly seen in trauma patients mainly due to aspiration of gastric contents. Causative organisms include MRSA, Streptococcus pneumoniae and Haemophilus influenzae.

Late onset ventilator associated pneumonia

Defined as that occurring on or after 5 days after intubation. Most common organisms involved in causing late onset pneumonia are the multidrug resistant pathogens. For e.g. Acinetobacter, Pseudomonas aeruginosa, MRSA.

Risk factors associated with ventilator associated pneumonia :

- Age ≥60 yr
- Acute respiratory distress syndrome
- Chronic obstructive pulmonary disease or other underlying pulmonary
- disease

- Coma or impaired consciousness
- Serum albumin level <2.2 g/dL
- Burns, trauma
- Blood transfusion
- Organ failure
- Supine position
- Large-volume gastric aspiration

- Sinusitis
- Immunosuppression
- Prolonged mechanical ventilation\

Non-invasive intermittent positive pressure ventilation should be used whenever possible in place of mechanical ventilation. Orotracheal intubation is preferred over nasotracheal intubation because of increased risk of sinusitis in the latter.

Attempts must be made to assess daily the readiness to extubate the patient, to adopt standard weaning protocols and increase ICU manpower.

Various methods to reduce the risk of aspiration pneumonitis includes:[68, 69, 70]

- Maintenance of cuff pressure around 20 cm H_2O
- Using newer cuff materials which helps to establish tight seal

- Continuous aspiration of subglottic secretions.
- Semirecumbent position
- Post pyloric feeding
- Promotility agents such as erythromycin

Shorr AF, Duh MS, Kelly KM, et al found that enteral nutrition started within 48 hours of intubation is associated with increased chances of aspiration pneumonitis.

Shorr AF, Duh MS, Kelly KM, et al also found that blood transfusion is also associated with increased risk of pneumonia.

Clinical Pulmonary Infection Score (CPIS) [71] incorporates the Following:

- Temperature
- Leukocyte count
- Chest x-ray infiltrates
- Appearance and volume of tracheal secretions
- PaO2, FIO2
- Culture and Gram stain of tracheal aspirate

Each factor is awarded 0-2 points each to yield a maximum of 12 points. A score >6 is associated with increased chances of developing pneumonia. However the specificity of this score is increased when cultures are taken into account.

Organisms responsible for causing ventilator associated pneumonia

include Pseudomonas aeruginosa, Enterobacteriaceae, Streptococcus pneumoniae, Staphylococcus aureus, and Haemophilus influenzae. Streptococci viridans, Enterococci species, Candida species and Coagulasenegative

staphylococci can also cause respiratory dysfunction

Urinary Tract Infection

Catheter-associated bacteriuria or candiduria typically presents as a colonization picture. It is mostly asymptomatic, and is not a likely cause of fever or secondary bloodstream infection [72, 73], even in immunocompromised patients [74]*83*, unless there is urinary tract obstruction, history of recent urologic manipulation, injury, or surgery, or neutropenia.

As effective prevention tactics, emphasis is now being placed on avoidance or brief duration of catheterization (e.g., <48 hours for elective surgery patients) [75'76] and on the use of silver alloy–coated catheters[77,78] when and where instrumentation is deemed appropriate. The typical signs and symptoms (e.g., dysuria, urgency, pelvic or flank pain, fever or chills) that correlate with bacteriuria in noncatheterized patients are rarely reported in ICU patients with documented catheterassociated bacteriuria or candiduria (>105 CFU/mL)[79,80] .

In the intensive care unit, most urinary tract infections are related to urinary catheters and are caused by multiresistant, nosocomial, gram-negative bacilli other than E. coli, Enterococcus species and yeasts [81].

Confirmation is by collecting a urine specimen which should be examined by direct microscopy, Gram stain and quantitative culture [82]

The specimen should be aspirated from the catheter sampling port after disinfecting the port with 70% to 90% alcohol, and should not be collected from the drainage bag.

Contrary to community-acquired urinary tract infections, pyuria may be absent with catheter-associated urinary tract infection. Even if present, pyuria is not a reliable predictor of UTI in the presence of a catheter [83]. The concentration of urinary bacteria or yeast required to cause any symptomatic urinary tract infection or fever is unclear, though it is clearly predictive that counts higher than 10_3 CFU/mL represent true bacteriuria or candiduria in catheterized patients [84]

Intra-Abdominal Infection

Intra abdominal infections are dichotomized into uncomplicated (uIAI) and complicated (cIAI) [85] and, more recently, as to whether they arose in the community associated (CAIAI) or hospital-associated (HA-IAI) setting (e.g., associated with a colon anastamotic dehiscence), and whether they are low, moderate, or high risk for clinical failure, morbidity, or death.

In uIAIs, the infection is restricted to a single organ and there may be no perforation of the gastrointestinal (GI) tract. Uncomplicated IAIs is never

associated with serious illness but a complicating hospital acquired infection may worsen the matter [86].

cIAIs will extend beyond the involved organ and further into the peritoneal cavity through the perforated viscus, thereby resulting in a greater SIRS response. The severity of infection depends on the extent to which it is contained by local intra-peritoneal defenses. In cases of high-risk or hospital acquired cIAI, broad-spectrum empirical antimicrobial therapy is indicated because of an increased risk of causative MDR pathogens [87,88]

There is a mortality rate of 25-35% in patients with abdominal sepsis [89, 90], but may reach upto 70% [91, 92]. Abdominal sepsis can be managed by drainage of the collection or the focus, resecting the infected foci segment (ranges from percutaneous drainage to serial laparotomies and open abdominal wound management in severe cases) [93].

Health care–associated non-postoperative IAIs, are those arising in patients hospitalized for reasons other than abdominal pathology, present with a poor prognosis [94]. There is a delay in diagnosis because of low suspicion, poor general condition, and altered mental status. Healthcare–associated IAIs are associated with pathogens that are multidrug resistant [95] and as a result they are treated inadequately as compared to patients with CA-IAIs, resulting in failure of treatment and a higher incidence of morbidity and mortality [96].

METHODOLOGY: (MATERIALS AND METHODS)

STUDY CENTRE : INSTITUTE OF GENERAL SURGERY, MMC AND RGGGH

SAMPLE SIZE: 30cases of splenectomy/ 30 cases of blunt injury abdomen who underwent other laparotomies

DURATION OF STUDY: June 2017 to October 2018

STUDY DESIGN : Prospective Observational Comparative Study

INCLUSION CRITERIA:

1. All patients undergoing splenectomy after trauma.
2. Other blunt trauma patients who underwent laparotomy

Exclusion criteria: Patients undergoing splenectomy for reasons other than trauma.

Assessment of parameters:

- WBC count
- Platelet count
- Injury Severity Score

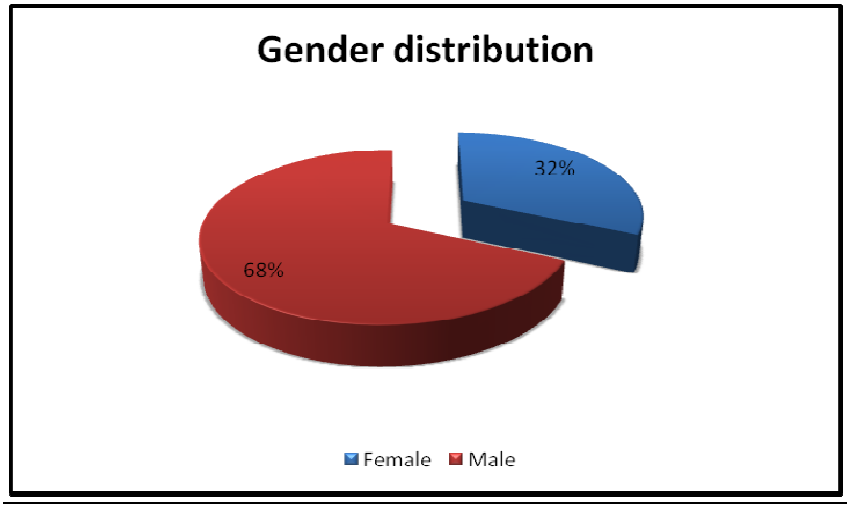
- Presence of postoperative infections such as pneumonia, abdominal abscess septicaemia, urinary tract and wound infections.

DATA ANALYSIS AND RESULTS

STATISTICAL ANALYSIS

GENDER DISTRIBUTION

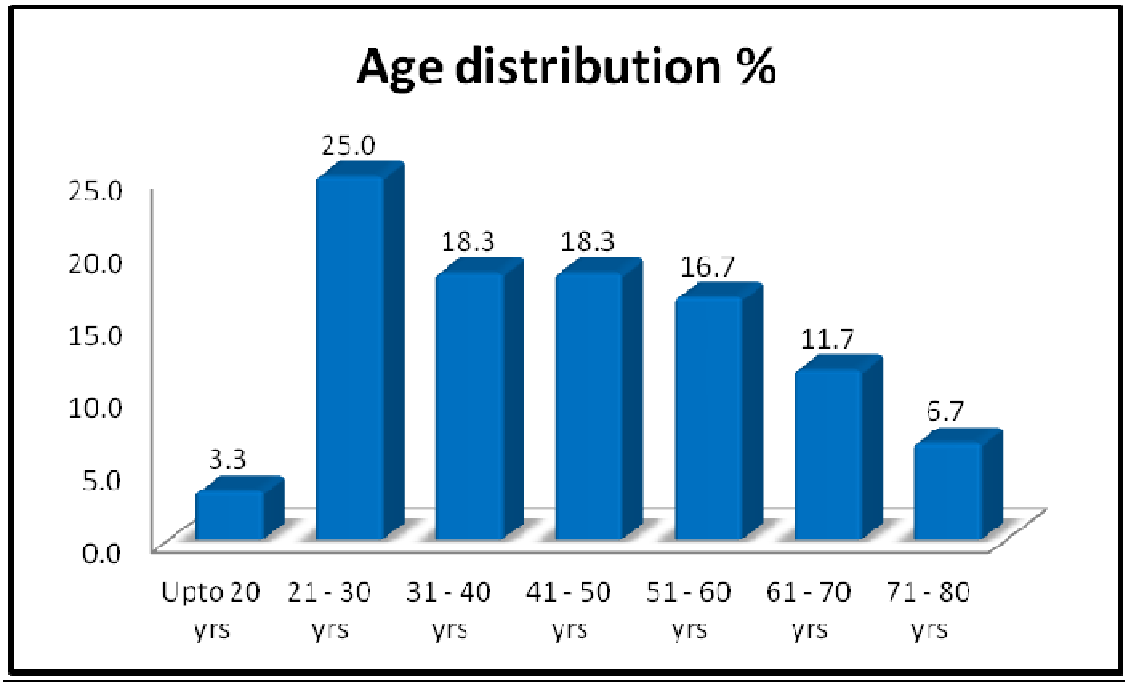
		Frequency	Percent
Valid	Female	19	31.7
	Male	41	68.3
	Total	60	100.0



In our study, there were a total of 41 males(68.3%) and 19 females (31.7%)

AGE DISTRIBUTION

		Frequency	Percent
Valid	Upto 20 yrs	2	3.3
	21 - 30 yrs	15	25.0
	31 - 40 yrs	11	18.3
	41 - 50 yrs	11	18.3
	51 - 60 yrs	10	16.7
	61 - 70 yrs	7	11.7
	71 - 80 yrs	4	6.7
	Total	60	100.0

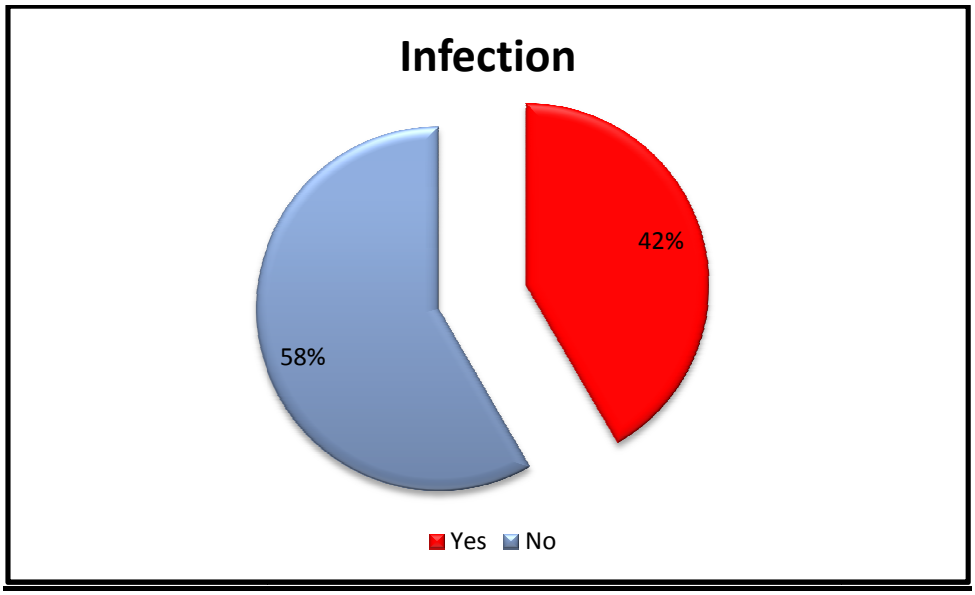


In our study, the majority study population was between 21-30 yrs.

PRESENCE OF INFECTION

INFECTION

		Frequency	Percent
Valid	Yes	25	41.7
	No	35	58.3
	Total	60	100.0

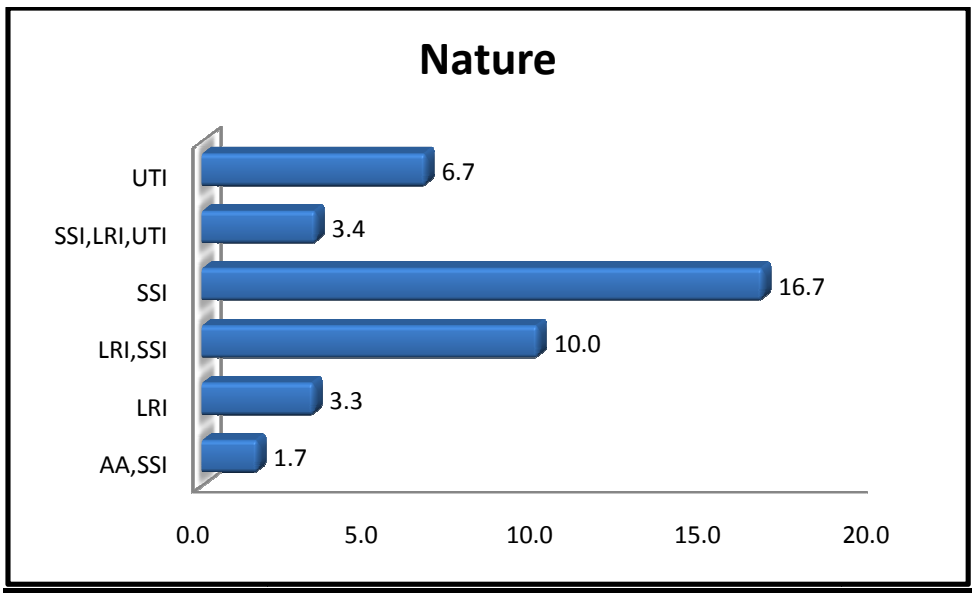


Out of 60 patients, 35 were found to be non infected and 25 were diagnosed with postoperative infection.

NATURE OF INFECTION

NATURE

		Frequency	Percent
Valid		35	58.3
	AA,SSI	1	1.7
	LRI	2	3.3
	LRI,SSI	6	10.0
	SSI	10	16.7
	SSI,LRI,UTI	2	3.4
	UTI	4	6.7
	Total	60	100.0



Surgical site infection was the most common infection and 15.1% infected individuals had more than one infection.

MORTALITY

EXPIRED

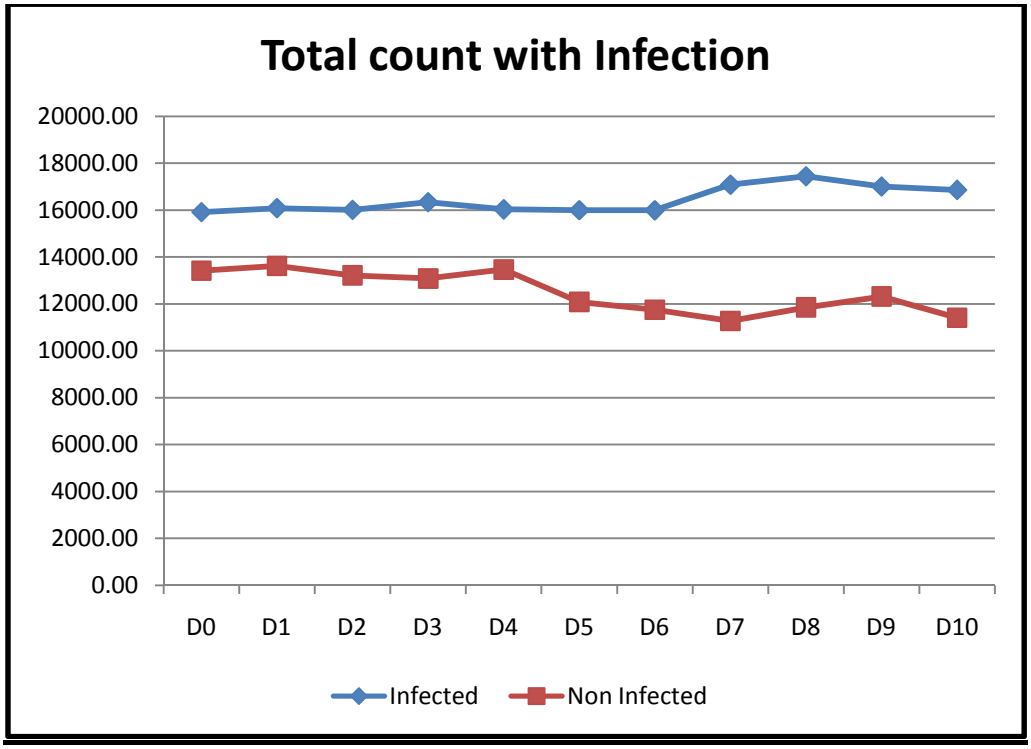
		Frequency	Percent
Valid	Alive	57	95.0
	Dead	3	5.0
	Total	60	100.0

In our study, we had a mortality of 5%.

COMPARISON OF TOTAL COUNT OF INFECTED VS NON INFECTED PERSONS

Group Statistics

INFECTION		N	Mean	Std. Deviation	Std. Error Mean
TC0	Yes	25	15920.00	3449.034	689.807
	No	35	13425.71	4291.122	725.332
TC1	Yes	25	16088.00	4751.782	950.356
	No	35	13625.71	3960.269	669.408
TC2	Yes	25	16016.00	3772.895	754.579
	No	35	13222.86	3845.860	650.069
TC3	Yes	25	16340.00	2889.060	577.812
	No	35	13088.57	3551.369	600.291
TC4	Yes	25	16036.00	2875.164	575.033
	No	35	13471.43	4714.220	796.849
TC5	Yes	25	16000.00	2592.457	518.491
	No	35	12088.57	2874.203	485.829
TC6	Yes	25	15996.00	2899.351	579.870
	No	35	11754.29	3067.989	518.585
TC7	Yes	25	17084.00	4703.162	940.632
	No	35	11277.14	3596.083	607.849
TC8	Yes	25	17444.00	4290.695	858.139
	No	35	11857.14	3590.124	606.842
TC9	Yes	25	17012.00	3434.788	686.958
	No	35	12320.00	3856.454	651.860
TC10	Yes	25	16864.00	3200.766	640.153
	No	35	11411.43	3565.832	602.736

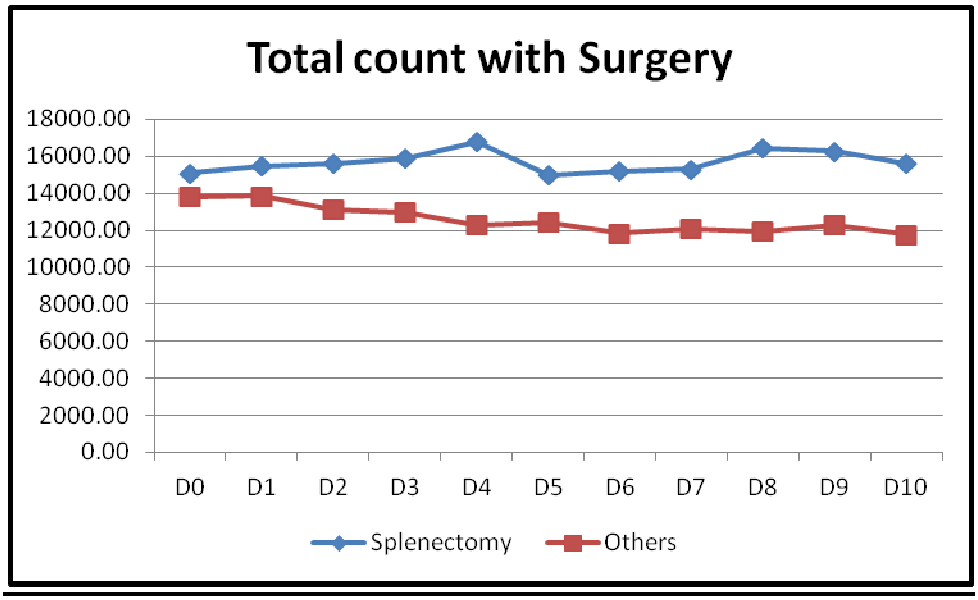


The infected and non-infected patients had similar counts till postoperative day 5. After post-operative day 5 the infected group had a total count > 15 x 10^3 / µL and a persistently higher total count than the non infected patients.

COMPARISON OF MEAN TOTAL COUNT OF SPLENECTOMY VS OTHER LAPAROTOMIES

Group Statistics

SURGERY		N	Mean	Std. Deviation	Std. Error Mean
TC0	S	30	15096.67	3319.169	605.995
	O	30	13833.33	4766.502	870.240
TC1	S	30	15463.33	4044.876	738.490
	O	30	13840.00	4732.762	864.080
TC2	S	30	15633.33	3382.749	617.603
	O	30	13140.00	4287.882	782.857
TC3	S	30	15893.33	2240.987	409.146
	O	30	12993.33	4199.830	766.781
TC4	S	30	16770.00	3029.755	553.155
	O	30	12310.00	4090.978	746.907
TC5	S	30	14996.67	1375.771	251.180
	O	30	12440.00	4203.250	767.405
TC6	S	30	15206.67	2282.155	416.663
	O	30	11836.67	3994.693	729.328
TC7	S	30	15310.00	4922.738	898.765
	O	30	12083.33	4561.313	832.778
TC8	S	30	16433.33	3995.543	729.483
	O	30	11936.67	4429.485	808.710
TC9	S	30	16263.33	2864.826	523.043
	O	30	12286.67	4678.029	854.087
TC10	S	30	15593.33	3454.825	630.762
	O	30	11773.33	4342.011	792.739

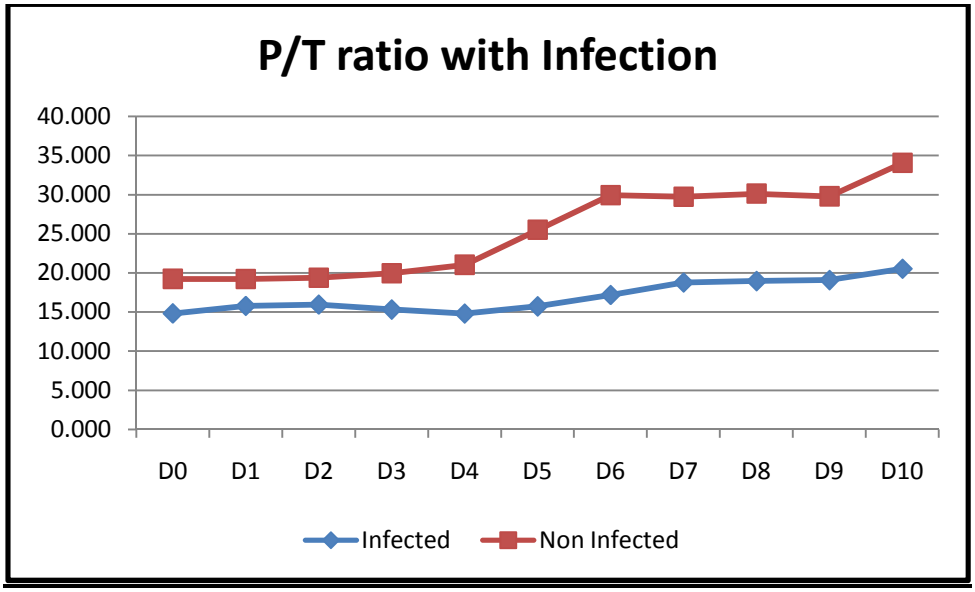


Patients who underwent splenectomy had a consistently higher total count compared to those who underwent other laparotomies

COMPARISON OF PLATELET/TOTAL COUNT RATIO IN INFECTED VS NON INFECTED PATIENTS

Group Statistics

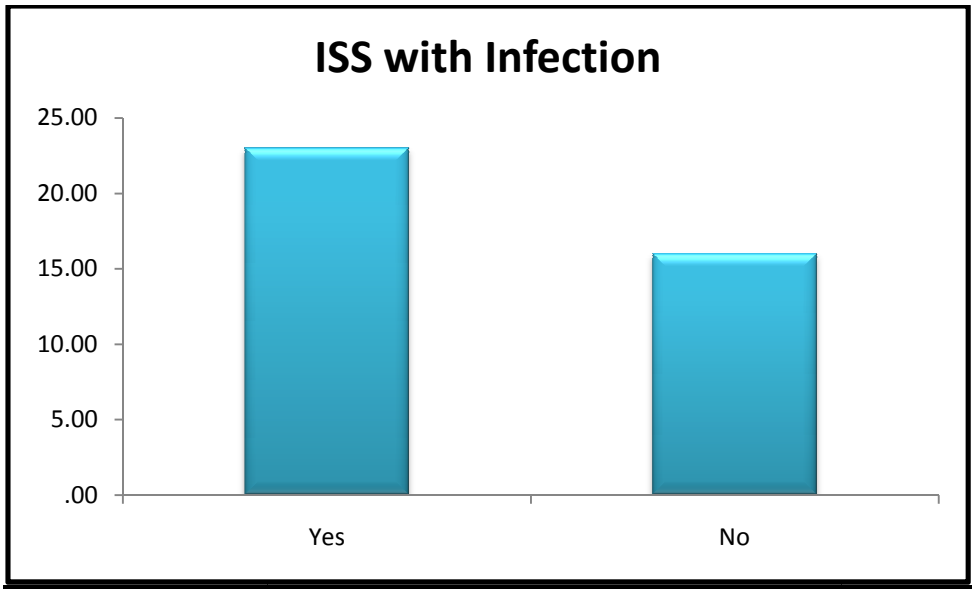
INFECTION		N	Mean	Std. Deviation	Std. Error Mean
PT0	Yes	25	14.800	7.0648	1.4130
	No	35	19.214	7.7456	1.3092
PT1	Yes	25	15.756	7.3683	1.4737
	No	35	19.206	7.9405	1.3422
PT2	Yes	25	15.928	6.5432	1.3086
	No	35	19.360	6.4717	1.0939
PT3	Yes	25	15.30	6.309	1.262
	No	35	19.93	6.187	1.046
PT4	Yes	25	14.788	5.6132	1.1226
	No	35	21.017	7.3913	1.2494
PT5	Yes	25	15.708	5.3141	1.0628
	No	35	25.486	6.0370	1.0204
PT6	Yes	25	17.156	5.0249	1.0050
	No	35	29.920	6.5257	1.1031
PT7	Yes	25	18.756	7.9006	1.5801
	No	35	29.717	6.7627	1.1431
PT8	Yes	25	18.944	8.7158	1.7432
	No	35	30.120	6.0815	1.0280
PT9	Yes	25	19.068	7.7676	1.5535
	No	35	29.769	6.4454	1.0895
PT10	Yes	25	20.512	8.6773	1.7355
	No	35	34.034	8.6751	1.4664



Patients who were infected were found to have a lower platelet count/Total count ratio than non infected patients: the difference became statistically significant from the 5th post operative day. (P/T Ratio <20)

SIGNIFICANCE OF ISS IN INFECTED INDIVIDUALS

INFECTION		N	Mean	Std. Deviation	Std. Error Mean
ISS	Yes	25	23.00	.426	.720
	No	35	16.00	.374	.750

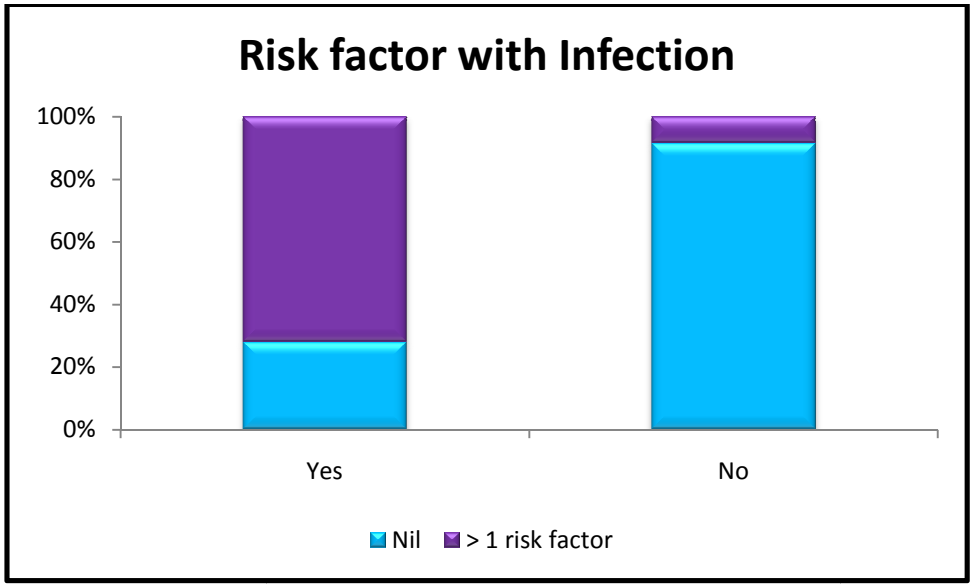


Individuals with higher ISS were found to have a significantly higher risk of infection (ISS>21)

SIGNIFICANCE OF PRESENCE OF MORE THAN 1 RISK FACTOR

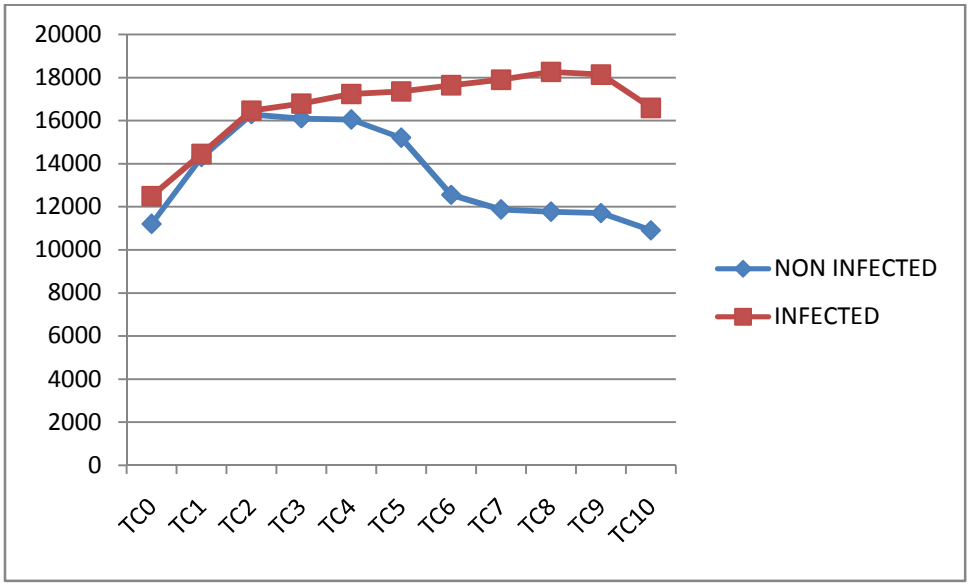
FOR INFECTION

			INFECTION		
			Yes	No	Total
IN	Nil	Count	7	32	39
		%	28.0%	91.4%	65.0%
	> 1 RF	Count	18	3	21
		%	72.0%	8.6%	35.0%
Total		Count	25	35	60
		% within INFECTION	100.0%	100.0%	100.0%



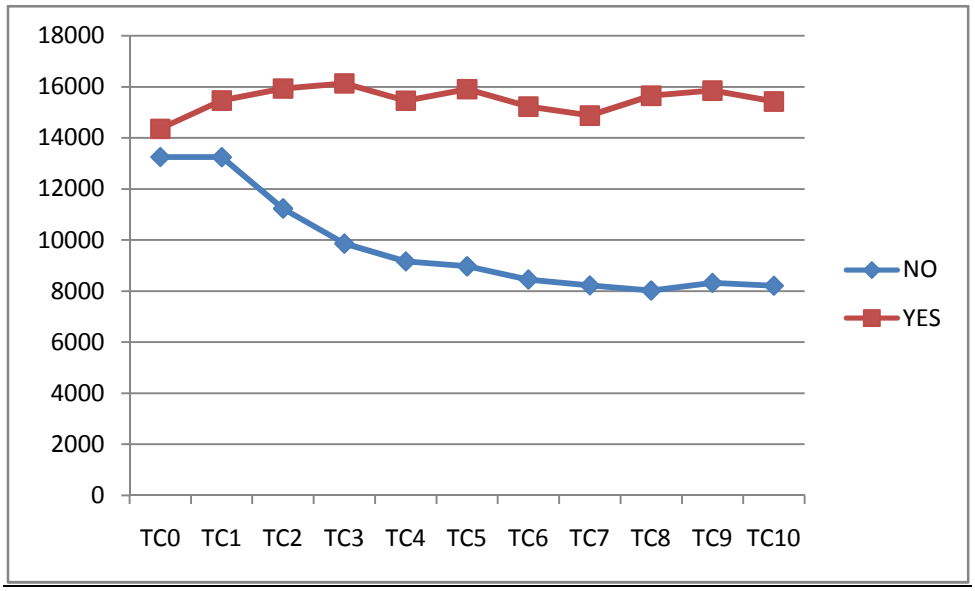
Presence of more than 1 risk factor increased risk of infection by 72%

COMPARISON OF TOTAL COUNTS OF INFECTED VS NON INFECTED PATIENTS WHO UNDERWENT SPLENECTOMY



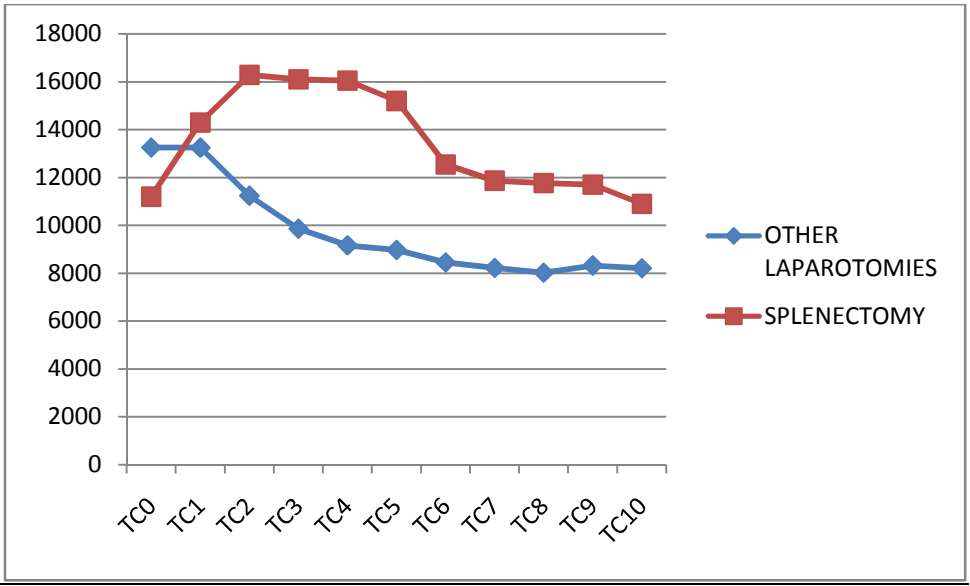
Even though both infected and non infected post splenectomy patients had higher than normal total counts, from the 5th post op day, the non infected patients showed a steady decline in the value reaching high normal values on the 10th post operative day, whereas infected patient's total counts continued to rise until treated. (Total count >15x 10^3 on POD 5 is significant)

COMPARISON OF TOTAL COUNTS INFECTED VS NON INFECTED PATIENTS WHO UNDERWENT LAPAROTOMIES OTHER THAN SPLENECTOMY



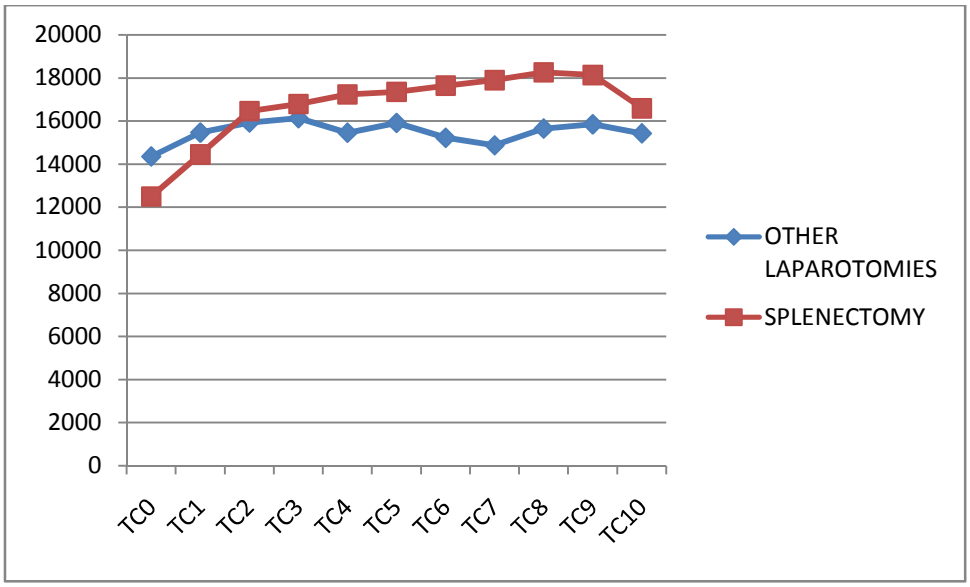
Though both groups had similar Total counts initially, non infected individuals had a steady fall of total count from post operative day 2.

COMPARISON OF TOTAL COUNTS OF NON INFECTED PATIENTS WHO UNDERWENT SPLENECTOMY VS OTHER LAPAROTOMIES



The total counts of non infected patients who underwent splenectomy was persistently higher than those who underwent laparotomies.

COMPARISON OF TOTAL COUNTS OF INFECTED PATIENTS WHO UNDERWENT SPLENECTOMY VS OTHER LAPAROTOMIES



Significant difference was not found in total counts of infected individuals who underwent splenectomy vs other laparotomies.

DISCUSSION

DISCUSSION

The current study validates the three risk factors:

- Total count
- Platelet/ total count ratio
- Injury severity score

for post-splenectomy infections in trauma patients compared to blunt abdomen injury patients who underwent laparotomies other than splenectomy.

A similar prospective study was published by Weng J, Brown CV, Rhee P, Salim A, Chan L, Demetriades D, Velmahos GC in the journal of trauma in May, 2005 but it did not compare splenectomy with other laparotomies.

Injury severity score was significant in the current study whereas it was not found to be significant in the previous study.

Another similar study published by Toutouzas KG, Velhamos GC, Kaminski A, Chan L, Demetriades D in 2002 in Jama surgery journal in August 2002, which took into consideration all three factors and found Injury Severity Score >16 to be an independent risk factor and presence of more than 1 risk factor predicted a 50% increased risk of infection . However in the current study, Injury Severity Score > 21 was found to be a risk factor and presence of more than 1 risk factor predicts a 72% increased risk of infection.

The following are the results of the study

- Injury severity score >21 is a significant risk factor.
- Post operative day 5 TC more than 15000 indicates infection.
- PC/TC ratio < 20 on the 5th post operative day indicates infection.
- Patients who underwent laparotomies other than splenectomy showed elevated Total count and Decreased platelet/total count ratio only if infected.
- Presence of more than 1 risk factor is associated with 72% chance of infection.

CONCLUSION

CONCLUSION

- Post operative day 5 is the earliest time that infected and non infected patients can be distinguished on the basis of total count and PC/TC ratio.

Risk factors for infection

□□Total count >15,000 on 5th post operative day

□□PC/TC ratio< 20 on 5th post operative day

□□ISS > 21

- Presence of more than one risk factor carries 72% increased chance of infection and these patients should be monitored with high degree of suspicion.

- Presence of increased total count and decreased platelet/total count on any post operative day in a patient who has undergone laparotomy other than splenectomy should be considered an indicator of infection and should be treated promptly.

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ANNEXURES

PATIENT CONSENT FORM

STUDY TITLE: **"LEUCOCYTOSIS AFTER POST TRAUMATIC SPLENECTOMY- A PHYSIOLOGICAL EVENT OR INDICATOR OF SEPSIS "–**

Name: Date:

Age: IP no :

Sex:

The details of the above study have been provided to me in writing and explained to me in my own language.

I confirm that I have understood the above study and had the opportunity to ask questions.

I understand that my participation in the study is voluntary and I am free to withdraw at any time without giving any reason, without the medical care that will normally be provided by the hospital being affected.

I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purposes.

I have been given an information sheet giving details of the study.

I hereby consent to participate in this study of "**LEUCOCYTOSIS AFTER POST TRAUMATIC SPLENECTOMY- A PHYSIOLOGICAL EVENT OR INDICATOR OF SEPSIS "–**

Date:

Place: Signature/ thumb impression of the patient

Patient's name:

Signature of the Investigator:

Name of the investigator:

INFORMATION SHEET

- Your blood sample from the first ten post operative days will be accepted.

- We are conducting a study on patients undergoing splenectomy after trauma in Rajiv Gandhi Government General Hospital and for that your blood sample will be valuable to us.
- Patients will be divided into two groups:

1.Patients undergoing post traumatic splenectomy.

2. Blunt trauma patients undergoing laparotomy for causes other than splenectomy.

- The purpose of this study is to identify the infection and physiologic response of splenectomy after trauma with the help of WBC count, Platelet count / WBC count ratio and Injury Severity Score.

- We are selecting certain cases and if your blood sample is found eligible, we may be using your blood sample to perform certain tests and special studies which in any way do not affect your final report or management.

- 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.

- The results of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the investigator Signature of the participant

Date:

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு
விபத்து காயங்களுக்காக மண்ணீரல் அகற்றப்பட்ட நோயாளிகளில் வழக்கமான மற்றும் கிருமி தாக்கத்தினால் இரத்த அணுக்களில் ஏற்படும் மாற்றங்களைக் கண்டறியும் ஆய்வு

பெயர்	:	தேதி	:
வயது	:	உள் நோயாளி எண்	:
பால்	:	ஆராய்ச்சி சேர்க்கை எண்	:

இந்த ஆராய்ச்சின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்துகொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

எனக்கு இந்த ஆராய்ச்சிக்காக இரத்தப் பரிசோதனை செய்துகொள்ள சம்மதம்.

இந்த ஆராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பத்தின்பேரில் பங்கு பெறுகின்றேன். இந்த ஆராய்ச்சியில் இருந்து நான் எந்நேரமும் பின்வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் ஏற்படாது என்பதையும் நான் புரிந்துகொண்டேன்.

நான் இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட ஆராய்ச்சித் தகவல் தாளைப் பெற்றுக் கொண்டேன்.

இதன் மூலம் எந்த பின்விளைவும் ஏற்படாது என்று மருத்துவர் மூலம் தெரிந்து கொண்டு, நான் என்னுடைய சுய நினைவுடனும் மற்றும் முழு சுதந்திரத்துடனும் இந்த மருத்துவ ஆராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதம் தெரிவிக்கிறேன்.

கையொப்பம்

ஆராய்ச்சி தகவல் தாள்

தங்களது இரத்தம் இங்கு பெற்றுக்கொள்ளப்பட்டது.

சென்னை இராஜீவ்காந்தி அரசு பொது மருத்துவனையில் விபத்து காயங்களுக்காக மண்ணீரல் அகற்றப்பட்ட நோயாளிகளின் வழக்கமான மற்றும் கிருமி தாக்கத்தினால் இரத்த அணுக்களில் ஏற்படும் மாற்றங்களைபற்றி அறிய மேற்கொள்ளப்படும் ஆராய்ச்சி இங்கு நடைபெற்று வருகின்றது.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். இந்த ஆராய்ச்சியில் உங்களுடைய இரத்தம் எடுத்து சில சிறப்புப் பரிசோதனைக்கு உட்படுத்தி அதன் தகவல்களை ஆராய்வோம். அதனால் தங்களது நோயின் ஆய்வறிக்கையோ அல்லது சிகிச்சையோ பாதிப்பு ஏற்படாது என்பதையும் தெரிவித்துக்கொள்கிறோம்.

முடிவுகளை அல்லது கருத்துக்களை வெளியிடும்போதோ அல்லது ஆராய்ச்சியின்போதோ தங்களது பெயரையோ அல்லது அடையாளங்களையோ வெளியிட மாட்டோம் என்பதை தெரிவித்துக்கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின்பேரில்தான் இருக்கிறது. மேலும் நீங்கள் எந்த நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

இந்த சிறப்பு பரிசோதனைகளின் முடிவுகளையும் நோயின் தன்மை பற்றியும் ஆராய்ச்சியின்போது அல்லது ஆராய்ச்சியின் முடிவின்போது தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்துக்கொள்கிறோம்.

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ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

PATIENT PROFORMA

Name : Age : Sex :

IP No. :

DOA: DOP: DOD:

Diagnosis :

Procedure Done:

Mode of injury:

List of injuries:

Presenting complaints:

Co-morbid illness:

Past surgical /Medical history:

On examination:

General condition:

VITALS:

PR: BP: RR:

CVS:

RS:

P/A:

PR:

Investigation chart:

Post operative day	WBC count	Platelet count (PC)	PC / WBC ratio
1			
2			
3			
4			
5			
6			
7			
8			
9			
10			

Injury Severity Score:

Post Operative Complications:

Wound infection:

Respiratory Infection:

Urinary tract infection:

Abdominal abscess:

Septicaemia:

Secondary outcomes:

Length of ICU stay:

Length of hospital stay:

Mortality:

Condition on discharge:

S.NO	NAME	AGE	SEX	IP NO	ISS	TC0	P0	PT0	TC1	P1	PT1	TC2	P2	PT2	TC3	P3	PT3	TC4	P4	PT4	TC5	P5	PT5	TC6	P6	PT6	TC7	P7	PT7	TC8	P8	PT8	TC9	P9	PT9	TC10	P10	PT10	SURGERY	INFECTION	NATURE	EXPIRED	DURATION OF STAY
1	Thirupathy	45	M	328456	9	10800	196000	18.1	9800	186000	19	8800	171000	19.4	10700	196000	18.3	14200	165000	11.6	14400	351000	24.4	10400	562000	54	16500	600000	36.4	15400	450000	29.2	14600	648000	44.4	10900	650000	59.6	SPLENECTOMY	NO		NO	12
2	Raman	52	M	412356	25	13000	195000	15	18000	265000	14.7	16800	165000	9.8	17100	196000	11.5	16800	183000	10.9	16800	146000	8.7	20600	406000	19.7	19000	475000	25	16000	452000	28.3	19300	432000	22.4	20400	425000	20.8	SPLENECTOMY	YES	SSI	NO	16
3	Kumar	44	M	213457	22	15400	198000	12.9	14000	120000	8.6	14200	195000	13.7	18000	190000	10.6	16500	198000	12	15600	193000	12.4	15800	213000	13.5	35200	235000	6.7	28800	260000	9	25300	291000	11.5	24100	358000	14.9	SPLENECTOMY	YES	LRI	NO	19
4	Paneerselvam	65	M	984356	24	18000	70000	3.9	6100	60000	9.8	13200	140000	10.6	15200	275000	18.1	16300	27000	16.6	16900	309000	18.3	15400	244000	15.8	15400	635000	41.2	28400	632000	22.3	16500	540000	32.7	19800	525000	26.5	SPLENECTOMY	YES	SSI	NO	15
5	Shanthi	26	F	213478	16	17800	174000	9.8	19400	104000	5.4	17800	346000	19.4	17500	354000	20.2	18500	321000	17.4	15100	456000	30.2	14900	398000	26.7	14800	364000	24.7	14200	485000	34.2	15200	645000	42.4	13200	520000	39.4	SPLENECTOMY	NO		NO	11
6	Priyan	30	M	334578	21	17000	215000	12.6	24000	224000	9.3	16900	230000	13.6	17000	265000	15.6	16300	198000	12.1	16500	257000	15.6	15800	298000	18.9	19700	356000	18.1	22500	456000	20.3	15200	415000	27.3	15800	546000	34.6	SPLENECTOMY	YES	LRI,SSI	NO	23
7	Shanmugam	46	M	221435	14	7600	144000	18.9	11000	193000	17.5	11600	275000	23.7	11800	129000	10.9	14300	150000	10.5	14700	275000	18.7	14500	324000	22.3	14000	256000	18.3	13200	456000	34.5	14200	465000	32.7	13200	545000	41.3	SPLENECTOMY	NO		NO	12
8	Punitha	50	F	65432	21	17500	241000	13.8	16500	166000	10.1	17400	195000	11.2	16400	265000	16.2	15000	398000	26.5	14800	412000	27.8	14200	321000	22.6	13600	432000	31.8	12600	654000	51.9	13500	425000	31.5	15400	326000	21.2	SPLENECTOMY	NO		NO	13
9	Fernandez	25	M	217857	22	13200	168000	12.7	9300	268000	28.8	11200	238000	21.3	17400	355000	20.4	16600	240000	14.5	16900	272000	16.1	20600	365000	17.7	17200	452000	26.3	18900	398000	21.1	20900	412000	19.7	17900	395000	22.1	SPLENECTOMY	YES	LRI, SSI	NO	18
10	Ashok	16	M	214567	22	13000	200000	15.4	19000	265000	13.9	15800	265000	16.8	16000	198000	12.4	17000	176000	10.4	16500	198000	12	16800	265000	15.8	17000	495000	29.1	15400	540000	35.1	19900	451000	22.7	22900	513000	22.4	SPLENECTOMY	YES	SSI	NO	14
11	Santhosh	44	M	224567	27	14100	210000	14.9	16600	122000	7.3	17800	218000	12.2	17000	260000	15.3	16200	220000	13.6	14500	420000	29	13600	422000	31	14000	590000	42.1	14700	425000	28.9	12500	569000	45.5	13000	540000	41.5	SPLENECTOMY	NO		YES	15
12	Pavithra	28	F	314567	9	19000	187000	9.8	13600	199000	14.6	17500	225000	12.9	16400	245000	14.9	18800	200000	10.6	16000	212000	13.3	16100	320000	19.9	15800	394000	24.9	17000	265000	15.6	15400	360000	23.4	12000	574000	47.8	SPLENECTOMY	NO		NO	11
13	Madhan	30	M	564789	29	10000	251000	25.1	7400	200000	27	8100	156000	19.3	10800	220000	20.4	11500	165000	14.3	14000	431000	30.8	14300	432000	39.3	15700	600000	38.2	16200	645000	39.8	18500	365000	19.7	19400	465000	24	SPLENECTOMY	NO		NO	13
14	Shankar	53	M	223145	35	12500	345000	27.6	12600	356000	28.3	14500	325000	22.4	13600	256000	18.8	16100	387000	24	14600	365000	25	13500	456000	33.8	12600	387000	30.7	13000	364000	28	13100	432000	33	13100	582000	44.4	SPLENECTOMY	NO		NO	11
15	Nandini	45	F	345987	24	15400	248000	16.1	16500	113000	6.8	14500	221000	15.2	16500	298000	18.1	16000	375000	23.4	14100	290000	20.6	13500	421000	31.2	13400	356000	26.6	16400	426000	26	14300	410000	28.7	19700	625000	31.7	SPLENECTOMY	NO		NO	12
16	Puniyakodi	76	M	908678	25	15900	254000	16	20300	310000	15.3	17000	180000	10.6	16900	198000	11.7	15800	148000	9.4	14400	210000	14.6	14000	613000	43.8	1200	610000	50	14400	510000	35.4	15900	545000	34.3	12300	560000	45.5	SPLENECTOMY	NO		NO	11
17	Murugan	30	M	45632	21	14600	326000	22.3	20000	320000	16	23500	373000	15.9	18000	265000	14.7	20400	365000	17.9	15400	412000	26.8	21100	500000	23.7	17400	465000	26.7	14900	684000	45.9	18500	640000	34.6	14300	650000	45.5	SPLENECTOMY	YES	SSI	NO	17
18	Madhavan	48	M	2134	24	16400	321000	19.6	17200	236000	13.7	17800	365000	20.5	18900	190000	10.1	17500	165000	9.4	18600	300000	16.1	16000	324000	20.3	16600	394000	23.7	19800	395000	19.9	15600	374000	24	15300	458000	29.9	SPLENECTOMY	YES	UTI	YES	20
19	Ethiraj	51	M	56746	14	19000	110000	5.8	16500	298000	18.1	17400	246000	14.1	16500	297000	18	18400	198000	10.8	14900	378000	25.4	14700	478000	32.5	13500	398000	29.5	14900	487000	32.7	13700	521000	38	17500	465000	26.6	SPLENECTOMY	NO		NO	12
20	Ram	37	M	234432	11	17400	320000	18.4	17000	364000	21.4	16500	212000	12.8	17400	224000	12.9	28500	398000	14	12800	385000	30.1	14000	421000	30.1	14700	384000	26.1	11000	410000	37.3	17400	698000	40.1	12000	540000	45	SPLENECTOMY	NO		NO	11
21	Janaki	36	F	56234	11	18600	142000	7.6	18000	116000	6.4	17500	222000	12.7	16300	223000	13.7	15600	198000	12.7	14600	212000	14.5	14200	463000	32.6	12100	469000	38.8	16000	420000	26.3	13900	398000	28.6	14900	390000	26.2	SPLENECTOMY	NO		NO	11
22	Sengottayan	80	M	23245	17	14900	267000	17.9	15900	345000	21.7	11300	225000	19.9	13600	248000	18.2	16600	300000	18.1	13700	298000	21.8	14300	337000	23.6	15900	265000	16.7	16500	356000	21.6	14200	420000	29.6	12800	567000	44.3	SPLENECTOMY	NO		NO	15
23	Mohan	39	M	12367	27	24000	162000	6.8	16000	321000	20.1	15600	254000	16.3	16000	300000	18.8	18500	321000	17.4	15300	465000	30.4	14600	356000	24.4	13500	345000	25.6	14000	452000	32.3	19800	354000	17.9	12500	542000	43.4	SPLENECTOMY	NO		NO	11
24	Annapoorni	64	F	34523	9	14600	210000	14.4.	14300	387000	27.1	15200	175000	11.5	15400	215000	14	14900	320000	21.5	14900	354000	23.8	13600	352000	27.9	13500	328000	24.3	16800	554000	33	19400	374000	19.3	12800	526000	41.1	SPLENECTOMY	NO		NO	13
25	Thulasi	43	F	87456	25	12500	221000	17.7	17000	132000	7.8	18000	271000	15.1	18600	298000	16	17900	268000	15	14900	365000	24.5	14200	421000	29.6	13300	565000	42.5	13600	453000	33.3	16900	421000	24.9	13100	452000	34.5	SPLENECTOMY	NO		NO	12
26	Rangaraj	38	M	183758	21	15000	241000	16.1	19300	465000	24.1	20100	245000	12.2	16800	275000	16.4	17200	210000	12.2	15700	219000	13.9	16500	265000	16.1	17400	398000	22.9	16300	420000	25.8	16500	425000	25.8	19300	523000	27.1	SPLENECTOMY	YES	LRI, SSI	NO	27
27	Sujatha	52	F	34565	14	17500	198000	11.2	16500	389000	23.6	16900	385000	22.8	18500	359000	19.4	20900	398000	19	15000	398000	26.5	16900	387000	22.9	16500	488000	29.6	14600	384000	26.3	14800	383000	25.9	14200	412000	29	SPLENECTOMY	NO		NO	13
28	Chakravarthy	32	M	72234	22	14000	113000	8.1	10200	225000	22.1	12000	210000	17.5	12200	225000	18.4	14500	222000	15.3	12500	300000	24	13000	265000	20.4	13100	332000	25.3	17400	468000	26.9	13900	484000	34.8	17400	450000	25.9	SPLENECTOMY	YES	UTI	NO	19
29	Vadivel	62	M	98345	21	14500	365000	25.2	14900	345000	23.2	13600	254000	18.7	13800	172000	12.5	12500	398000	31.8	12900	201000	15.6	14200	421000	29.6	13200	374000	28.3	13600	341000	25.1	15400	425000	27.6	14000	452000	32.3	SPLENECTOMY	NO		NO	11
30	Vinod	47	M	34623	16	9700	165000	17	17000	132000	7.8	20500	165000	8	16500	210000	12.7	13800	210000	15.2	12900	574000	44.5	14900	554000	37.2	13500	400000	29.6	16500	398000	24.1	13600	430000	31.6	14600	465000	31.8	SPLENECTOMY	NO		NO	13
31	Shahida	59	F	12675	11	21000	172000	8.19	22000	174000	7.9	16000	189000	11.8	14000	180000	12.8	12800	180000	14	11700	250000	21.3	9800	235000	23.9	8700	210000	24.1	8100	226000	27.9	7600	232000	30.5	6500	246000	37.8	PRIMARY CLOSURE JEJUNAL PERFORATION	NO		NO	11
32	Michael	34	M	23876	13	13600	256000	18.8	14300	238000	16.6	11900	221000	18.5	10600	223000	21	9500	243000	25.5	8800	245000	27.8	7600	243000	31.9	7700	245000	31.8	7600	243000	31.9	8000	226000	28.2	7600	234000	30.7	PRIMARY SUTURING LIVER LACERATION	NO		NO	12
33	Kanthamani	55	F	87456	17	12200	276000	22.6	13400	254000	18.9	12800	236000	18.4	10300	245000	23.7	9800	235000	23.9	9600	234000	24.3	8500	234000	27.5	8700	210000	24.1	7400	228000	30.8	7600	226000	29.7	7200	257000	35.6	NEPHRECTOMY	NO		NO	16
34	Malarvizhi	65	F	65896	21	22800	194000	8.5	23800	200000	8.4	21900	186000	8.4	18700	195000	10.4	15400	210000	13.6	16500	220000	13.3	18400	212000	11.5	19200	187000	9.7	20000	186000	9.3	22100	216000	9.7	21000	226000	10.7	RESECTION ANASTAMOSISOF JEJUNUM	YES	SSI	NO	22
35	Duraisamy	72	M	43456	11	8900	234000	26.2	8800	245000	27.8	8700	210000	24.1	9500	243000	25.5	9600	234000	24.3	8500	234000	26.2	8100	226000	27.9	7600	232000	30.5	7600	243000	31.9	8700	210000	24.1	7600	232000	30.5	PRIMARY CLOSURE OF BLADDER RENT	NO		NO	12
36	Vignesh	24	M	34786	19	14000	180000	12.8	12800	236000	18.4	15400	210000	13.6	16500	210000	12.7	12800	180000	14	12800	236000	18.4	15400	210000	13.6	14600	223000	15.2	16200	233000	14.3	16500	398000	24.1	14600	465000	31.8	PRIMARY CLOSURE JEJUNAL PERFORATION	YES	SSI,LRI	NO	22
37	Varadharaj	46	M	98567	16	9800	235000	23.9	9600	234000	24.3	8800	245000	27.8	9500	243000	25.5	8800	245000	27.8	8700	210000	24.1	8800	245000	27.8	7600	235000	30.9	7700	245000	31.8	8700	226000	25.9	8100	226000	27.9	NEPHRECTOMY	NO		NO	13
38	Elumalai	58	M	108090	16	8700	210000	24.1	9500	243000	25.5	8800	245000	27.8	8100	226000	27.9	7800	236000	30.2	8100	226000	27.9	8500	234000	26.2	8700	210000	24.1	8100	226000	27.9	8400	253000	30.1	8100	213000	26.2	PRIMARY CLOSURE OF DIAPHRAGMATIC RENT	NO		NO	12
39	Imchan	19	M	11878	21	18700	218000	11.6	17800	224000	12.5	18100	247000	13.6	16700	245000	14.6	16500	210000	12.7	16500	220000	13.3	15400	223000	14.4	16500	220000	13.3	16200	233000	14.3	12800	180000	14	12800	236000	18.4	PRIMARY CLOSURE ILEAL PERFORATION	YES	SSI	NO	18
40	Paulraj	22	M	11675	22	8900	320000	35.9	8700	312000	35.8	9200	305000	33.1	8900	290000	32.5	9800	300000	30.6	10200	280000	27.4	10700	290000	27.1	11000	221000	20	9900	223000	22.5	10100	224000	22.1	10200	280000	27.4	PRIMARY SUTURING LIVER LACERATION	YES	LRI	NO	17
41	Farooq	26	M	23145	19	15400	210000	13.6	16500	220000	13.3	13400	254000	18.9	15400	210000	13.6	12800	236000	18.4	16500	220000	13.3	16200	233000	14.3	18400	212000	11.5	16500	210000	12.7	15400	210000	13.6	16000	232000	14.5	RESECTION ANASTAMOSISOF JEJUNUM	YES	AA,SSI	NO	32
42	Hari	30	M	31465	17	22300	213000	9.5	22100	212000	9.5	21800	223000	10.2	18100	247000	13.6	17800	224000	12.5	16500	210000	12.7	12800	236000	18.4	18100	247000	13.6	16700	245000	14.6	15400	210000	13.6	14600	223000	15.2	RESECTION ANASTAMOSISOF ILEUM	YES	SSI,LRI	NO	36
43	Sowmya	26	F	679009	16	21900	186000	8.4	18900	196000	10.3	17800	221000	12.4	18200	214000	11.7	17600	218000	12.3	14500	222000	15.3	12500	300000	24	14600	223000	15.2	16200	233000	14.3	22100	216000	9.7	16500	220000	13.3	RIGHT HEMICOLECTOMY	YES	SSI,LRI,UTI	YES	35
44	Sridevi	28	F	878908	18	9200	305000	33.1	8900	290000	32.5	8900	320000	35.9	8700	312000	35.8	8900	290000	32.5	10700	290000	27.1	9800	300000	30.6	8500	234000	27.5	9500	243000	25.5	10900	243000	22.2	12300	212000	17.2	PRIMARY CLOSURE OF BLADDER RENT	YES	UTI	NO	22
45	Bujiammal	72	F	568743	36	17800	224000	12.5	18100	247000	13.6	18700	218000	11.6	15400	210000	13.6	14600	223000	15.2	16200	233000	14.3	16500	210000	12.7	18100	247000	13.6	17800	224000	12.5	16500	210000	12.7	16500	220000	13.3	PRIMARY CLOSURE ILEAL PERFORATION	YES	SSI	NO	28
46	Krishnammal	63	F	237986	22	8500	234000	26.2	8700	210000	24.1	8800	245000	27.8	8500	234000	26.2	8100	226000	27.9	7600	232000	30.5	7600	243000	31.9	8500	234000	27.5	8700	210000	24.1	7400	228000	30.8	7600	243000	31.9	NEPHRECTOMY	NO		NO	12
47	Parthiban	36	M	346727	24	9800	300000	30.6	10200	280000	27.4	9500	243000	25.5	8800	245000	27.8	8100	226000	27.9	9600	234000	24.3	8800	245000	27.8	8100	226000	27.9	7400	228000	30.8	8500	234000	26.2	8700	210000	24.1	PRIMARY SUTURING LIVER LACERATION	NO		NO	11
48	Kalaivani	36	F	236787	11	8800	245000	27.8	8500	234000	26.2	8100	226000	27.9	9200	305000	33.1	8900	290000	32.5	8100	226000	27.9	8500	234000	26.2	8700	210000	24.1	8500	234000	27.5	8900	290000	32.5	7900	243000	30.7	PRIMARY CLOSURE OF BLADDER RENT	NO		NO	14
49	Appukutti	69	M	78690	14	14500	232000	16	13800	213000	15.4	13200	223000	16.8	12900	224000	17.3	10900	218000	20	9800	221000	22.5	8800	245000	27.8	8100	226000	27.9	7600	232000	30.5	8500	234000	27.5	8400	253000	30.1	PRIMARY CLOSURE ILEAL PERFORATION	NO		NO	11
50	Sridhar	23	M	56478	22	17800	221000	12.4	18200	214000	11.7	17600	218000	12.3	14500	222000	15.3	21900	186000	8.4	18900	196000	10.3	17800	224000	12.5	18100	247000	13.6	16500	210000	12.7	18100	247000	13.6	17800	224000	12.5	RESECTION ANASTAMOSISOF ILEUM	YES	SSI,LRI	NO	25
51	Senthil	47	M	349876	18	16500	210000	12.7	18700	218000	11.6	17800	224000	12.5	18100	247000	13.6	16700	245000	14.6	17800	221000	12.4	18200	214000	11.7	17600	218000	12.3	16500	220000	13.3	15400	223000	14.4	16500	220000	13.3	PRIMARY CLOSURE JEJUNAL PERFORATION	YES	SSI	NO	17
52	Raghuvaran	57	F	231456	16	8500	234000	27.5	8700	210000	24.1	7400	228000	30.8	7600	243000	31.9	8700	312000	35.8	9200	305000	33.1	8900	290000	32.5	8800	245000	27.8	8100	226000	27.9	9600	234000	24.3	8400	253000	30.1	PRIMARY CLOSURE OF BLADDER RENT	NO		NO	11
53	Rahul	23	M	98976	19	13400	254000	18.9	15400	210000	13.6	12800	236000	18.4	16500	220000	13.3	16200	233000	14.3	21800	223000	10.2	18100	247000	13.6	17800	224000	12.5	16500	220000	13.3	15400	223000	14.4	16500	220000	13.3	RESECTION ANASTAMOSISOF ILEUM	YES	SSI	NO	14
54	Vimala	56	F	223654	11	8900	290000	32.5	9800	300000	30.6	10200	280000	27.4	10700	290000	27.1	8500	234000	27.5	8700	210000	24.1	7400	228000	30.8	7600	243000	31.9	8700	312000	35.8	8500	234000	26.2	8700	210000	24.1	PRIMARY SUTURING LIVER LACERATION	NO		NO	13
55	Prabhakar	66	M	400987	9	8700	312000	35.8	8900	290000	32.5	10700	290000	27.1	8700	210000	24.1	8800	245000	27.8	8500	234000	26.2	8100	226000	27.9	8500	234000	26.2	8700	210000	24.1	7600	232000	30.5	7200	221000	30.6	PRIMARY CLOSURE OF BLADDER RENT	NO		NO	14
56	Eswaran	58	M	300954	22	16200	228000	14	15800	230000	14.5	12800	221000	17.2	11900	246000	20.6	8700	228000	26.2	8800	245000	27.8	8500	234000	26.2	7400	228000	30.8	8100	226000	27.9	8400	253000	30.1	8100	213000	26.2	PRIMARY CLOSURE ILEAL PERFORATION	NO		NO	11
57	Nagaraj	37	M	78690	22	18100	247000	13.6	17800	224000	12.5	16500	210000	12.7	17800	224000	12.5	18100	247000	13.6	18700	218000	11.6	15400	210000	13.6	17600	218000	12.3	14500	222000	15.3	17800	224000	12.5	18100	247000	13.6	RESECTION AND ILEOSTOMY	YES	SSI,UTI,LRI	NO	37
58	Riyaz	29	M	321098	17	15400	210000	13.6	14600	223000	15.2	16200	233000	14.3	16500	210000	12.7	13800	213000	15.4	13200	223000	16.8	12900	224000	17.3	10900	218000	20	18200	214000	11.7	17600	218000	12.3	14500	222000	15.3	RESECTION ANASTAMOSISOF ILEUM	YES	UTI	NO	26
59	Sathish	31	M	312876	16	16200	233000	14.3	12800	180000	14	12800	236000	18.4	21900	186000	8.4	18900	196000	10.3	17800	221000	12.4	18200	214000	11.7	18100	247000	13.6	16500	210000	12.7	17600	218000	12.3	16500	220000	13.3	PRIMARY CLOSURE ILEAL PERFORATION	YES	SSI	NO	19
60	Maheshwari	39	F	324679	14	8500	234000	26.2	8100	226000	27.9	7600	232000	30.5	7600	243000	31.9	8500	234000	27.5	8900	290000	32.5	8900	320000	35.9	8700	312000	35.8	8100	226000	27.9	8500	234000	26.2	8700	210000	24.1	PRIMARY SUTURING LIVER LACERATION	NO		NO	12