Dissertation

"STUDY ON NORMAL AND INFECTION RESPONSE AFTER SPLENECTOMY FOR TRAUMA"

M.S. BRANCH - I

GENERAL SURGERY

MADRAS MEDICAL COLLEGE
THE TAMILNADU
Dr. MGR MEDICAL UNIVERSITY
CHENNAI – TAMILNADU

APRIL 2015

CERTIFICATE

This is to certify that, the dissertation entitled **"STUDY ON NORMAL AND INFECTION RESPONSE AFTER SPLENECTOMY FOR TRAUMA"** is the bonafide work done by **Dr.KANNAH.E** during his MS (General Surgery) course 2012-2015, done under my supervision and is submitted in partial fulfillment for the requirement of the M.S.(BRANCH-I)- General Surgery, April 2015 examination of The Tamilnadu Dr.MGR Medical University.

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DECLARATION

I, declare that this dissertation titled **"STUDY ON NORMAL AND INFECTION RESPONSE AFTER SPLENECTOMY FOR TRAUMA"** 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).

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INSTITUTIONAL ETHICS COMMITTEE
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CERTIFICATE OF APPROVAL

To

Dr.Kannah. E,
Postgraduate,
Institute of General Surgery,
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Dear **Dr.Kannah. E,**

The Institutional Ethics Committee of Madras Medical College, reviewed and discussed your application for approval of the proposal entitled **"Study on infection and normal response after Splenectomy for Trauma"** No.34042014.

The following members of Ethics Committee were present in the meeting held on 11.03.2014 conducted at Madras Medical College, Chennai-3.

1. Dr. C.Rajendran, M.D, -- Chairperson
2. Prof. Kalaiselvi, M.D, -- Member Secretary
   Vice Principal, MMC, Ch-3
3. Prof. Nandhini, M.D, -- Member
   Inst. of Pharmacology, MMC, Ch-3
4. Prof.Bhavani Sankar, M.S, -- Member
   Prof & HOD General Surgery, MMC, Ch-3
5. Prof.V.Padmavathi, M.D, -- Member
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6. Thiru. S. Govindasamy, BA., BL -- Lawyer
7. Tmt.Arnold Saulina, MA MSW -- Social Scientist
8. Thiru.S.Ramesh Kumar, -- Lay Person
   Administrative Officer, MMC, Ch-3.

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

Sd/Chairman & Other Members

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.

Member Secretary, Ethics Committee

MEMBER SECRETARY
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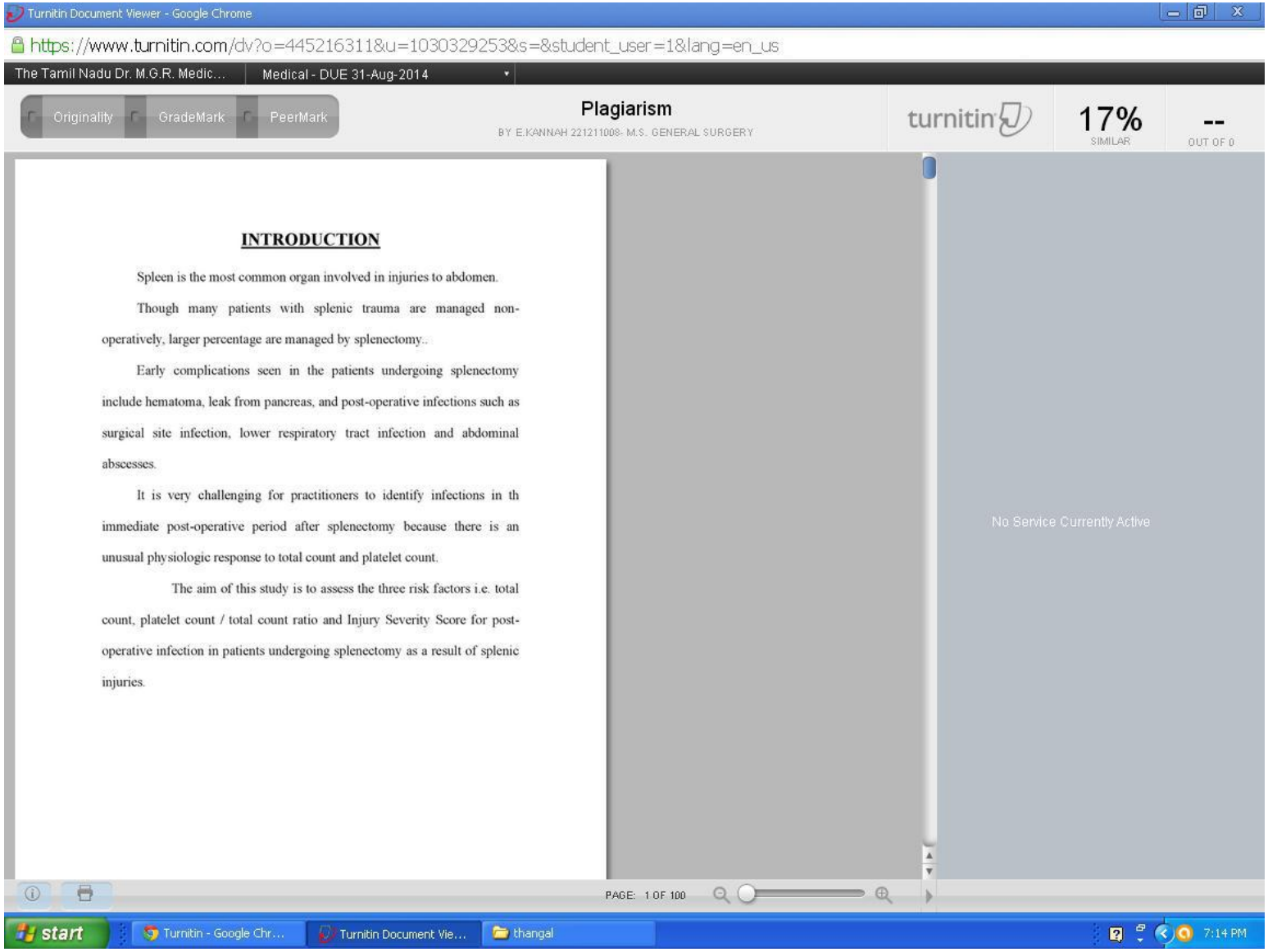
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INTRODUCTION
Spleen is the most common organ involved in injuries to abdomen.
Though many patients with splenic trauma are managed non-operatively, larger percentage are managed by splenectomy..
Early complications seen in the patients undergoing splenectomy include hematoma, leak from pancreas, and post-operative infections such as surgical site infection, lower respiratory tract infection and abdominal abscesses.
It is very challenging for practitioners to identify infections in th immediate post-operative period after splenectomy because there is an unusual physiologic response to total count and platelet count.
The aim of this study is to assess the three risk factors i.e. total count, platelet count / total count ratio and Injury Severity Score for post-operative infection in patients undergoing splenectomy as a result of splenic injuries.
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ACKNOWLEDGEMENT

As I walk down the memory lane, I realize with a deep sense of humility that what I have done now would not have been possible, but for certain luminaries, who have enlightened my path to wisdom.

"Surgery is learnt by apprenticeship and not from textbooks, not even from one profusely illustrated" – Ian Aird.

While I put these words together it is my special privilege and great pleasure to record my deep sense of gratitude to my revered Professor and Guide **Prof. T. Bavanisankar, M.S.,** and **Prof. A. Affee Asma. M.S.,** but for whose constant guidance, help and encouragement this research work would not have been made possible. The unflinching academic, moral and psychological support will remain ever fresh in my memory for years to come. Words cannot simply express my gratitude to them for imparting to me the surgical skills that I have acquired.

It is my pleasure to record my profound gratitude and indebtness to **Prof.S.Deivanayagam M.S.,** and **Prof. P. Ragumani M.S.,** for their support, keen interest and the constant encouragement they have given during the course of this thesis work.

I would like to express my sincere thanks to **Dr. A. Anandi, M.S., Dr. S. Nedunchezhian, M.S., Dr. S, Selvakumar, M.S.,** Assistant

Professors of Surgery, for all of them have given me invaluable advice, guided me and have been most kind and patient to me.

My heartfelt thanks to the entire Institute of Biochemistry for granting me permission and helping me to conduct this study.

All along the way, I have been supported and encouraged by all my associate professors and assistant professors who helped me to reach where I am.

I also thank my fellow postgraduates, friends and colleagues who have extended their co-operation in my work.

I thank the Dean, MMC & RGGGH for permitting me to conduct this study.

I would be failing in my duty if I do not show my deep sense of gratitude to all the patients who had helped me to become a surgeon and especially those who consented to be part of this study.

With deep reverence, I salute my parents and I thank the Almighty for blessing me with a wonderful family to whom I have dedicated this thesis and leave unsaid what they mean to me.

CONTENTS

INTRODUCTION

Spleen is the most common organ involved in injuries to abdomen.

Though many patients with splenic trauma are managed non-operatively, larger percentage are managed by splenectomy..

Early complications seen in the patients undergoing splenectomy include hematoma, leak from pancreas, and post-operative infections such as surgical site infection, lower respiratory tract infection and abdominal abscesses.

It is very challenging for practitioners to identify infections in the immediate post-operative period after splenectomy because there is an unusual physiologic response to total count and platelet count.

The aim of this study is to assess the three risk factors i.e. total count, platelet count / total count ratio and Injury Severity Score for post-operative infection in patients undergoing splenectomy as a result of splenic injuries.

AIMS & OBJECTIVES

1. To study the total count and platelet count (PC) / total count ratio in patients undergoing splenectomy for trauma and their response in the infected individuals.
2. To study the association of the three risk factors i.e. total count, PC/ total count ratio and Injury Severity Score in those individuals who have underwent splenectomy for trauma and their role in post operative infection.

REVIEW OF LITERATURE

HISTORICAL BACKGROUND:

The spleen in the ancient times was famously known to have had different purposes by different people, for Gable it was an “organ of mystery”; it was considered as an unwanted organ by Aristotle; Pliny regarded it as an organ that produced laughter and mirth, which was again reinforced by the Babylonian Talmud. Pliny also regarded it as an organ that hindered with the speed of professional athletes.

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.

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.

The first splenectomy in the United States was done in the year 1816, by a Royal Navy Surgeon, O’Brien. In England, Sir Thomas Spencer Wells recorded the first successful splenectomy in 1866.

In an autopsy done in 1881, Billroth stated that a splenic injury might heal spontaneously, but in 1927, Hamilton Bailey asserted the need for surgical intervention.

During the first two decades of the 20th century, many theories began to appear regarding the purpose and use of 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.

Campos Christo in 1962 reported the first successful partial splenectomy for the modern times.

The role of spleen in fighting infections has always been under consideration.

Experiments by Bullock and Morris with rat plague bacillus in the year 1919 proved that by removing the spleen, we are robbing the body of its resistance.

Most physicians and surgeons have considered that splenetomy doesn't hamper the host defense system, until recently when in 1965 Shumacker and Kling elicited that immunity is compromised in children whose spleen has been removed in cases of hematological disorders.

Singer's review of literature in 1973 further elicited the incidence of post splenectomy sepsis in infants and children.

ANATOMY:

EMBRYOLOGY:

The spleen is formed by the mesenchymal differentiation along the left side of the dorsal mesogastrium in juxtaposition to the anlage of the left gonad in the 8-mm embryo as shown in Fig 1.

STRUCTURE AND POSITION:

In a healthy adult the spleen weighs from approximately 75 to 100 gms and is placed deep to the ninth, tenth and eleventh ribs in the posterior portion of the left upper quadrant with its long axis corresponding to the tenth rib.

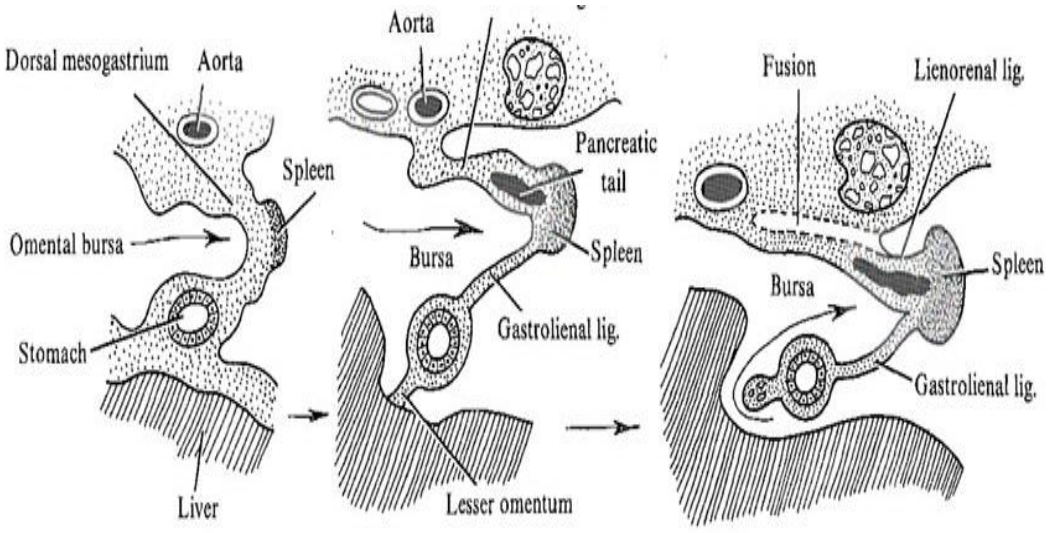


Fig 1- Development and position of spleen in dorsal mesogastrium

Its superior, convex and the lateral surface lies adjacent to the underside of the left leaf of the diaphragm. Its medial surface is concave due to the impressions made on it by the stomach, pancreas, splenic flexure and the kidneys as shown in Fig 2.

Several suspensory ligaments help in keeping the spleen in position. These include gastrosplenic, splenophrenic, splenocolic, and splenorenal ligaments (Fig 3). Usually these ligaments are avascular except the gastrosplenic ligament which contains the short gastric vessels that course to the splenic hilum from the greater curvature. But these ligaments may become vascular in patients with portal hypertension and myeloproliferative disorders.

BLOOD SUPPLY:

The splenic artery provides the main blood supply to the spleen. It is a branch of the coeliac trunk. Further branching of the arteries occur proximal to the hilus which includes a frequent branch to the inferior pole ehich originates centrally.

VENOUS DRAINAGE:

The major venous drainage occurs through the splenic vein, which usually receives the inferior mesenteric vein centrally, and then joins the superior mesenteric vein to form the portal vein.

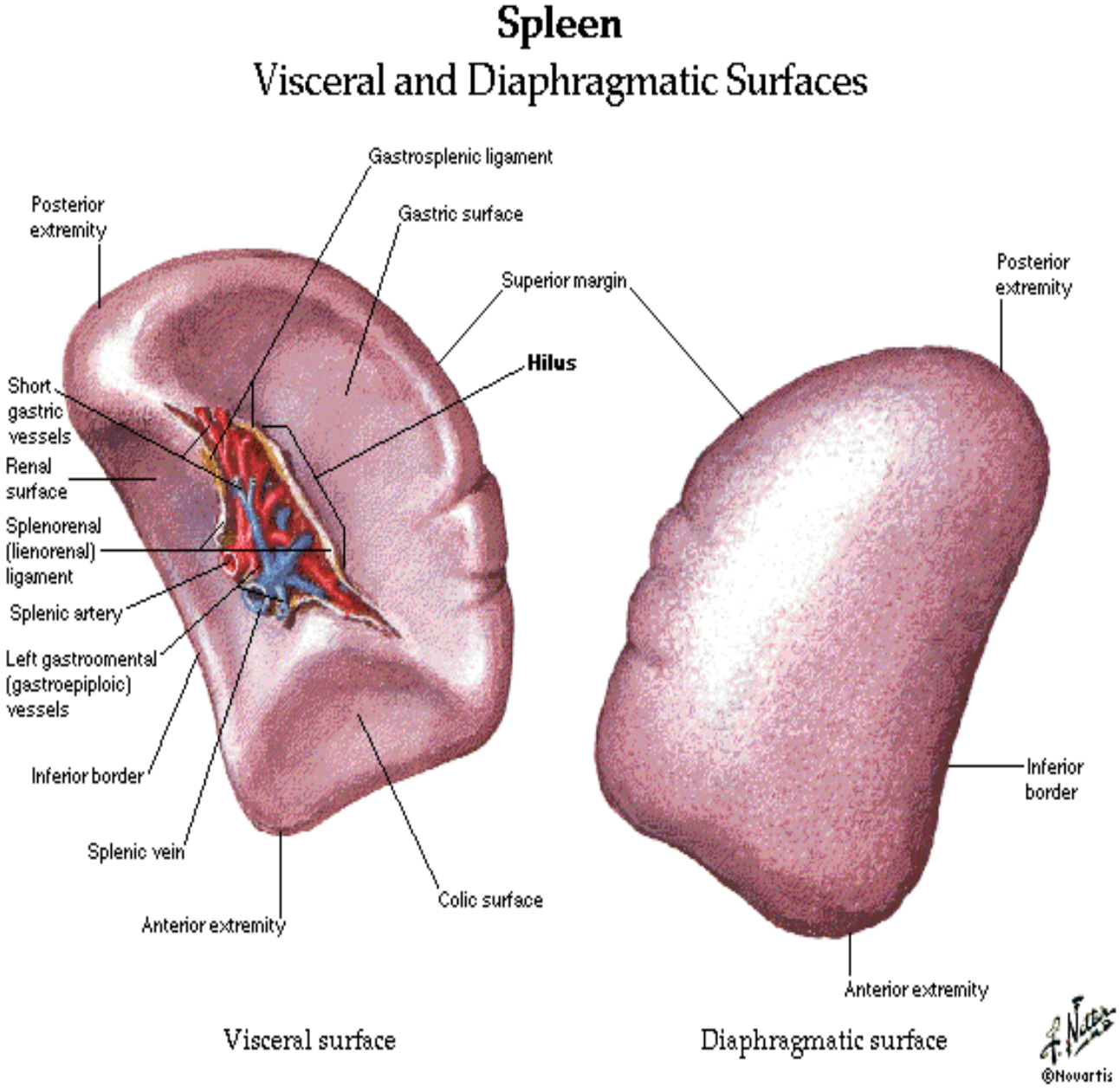


Fig 2 – Gross anatomy of spleen

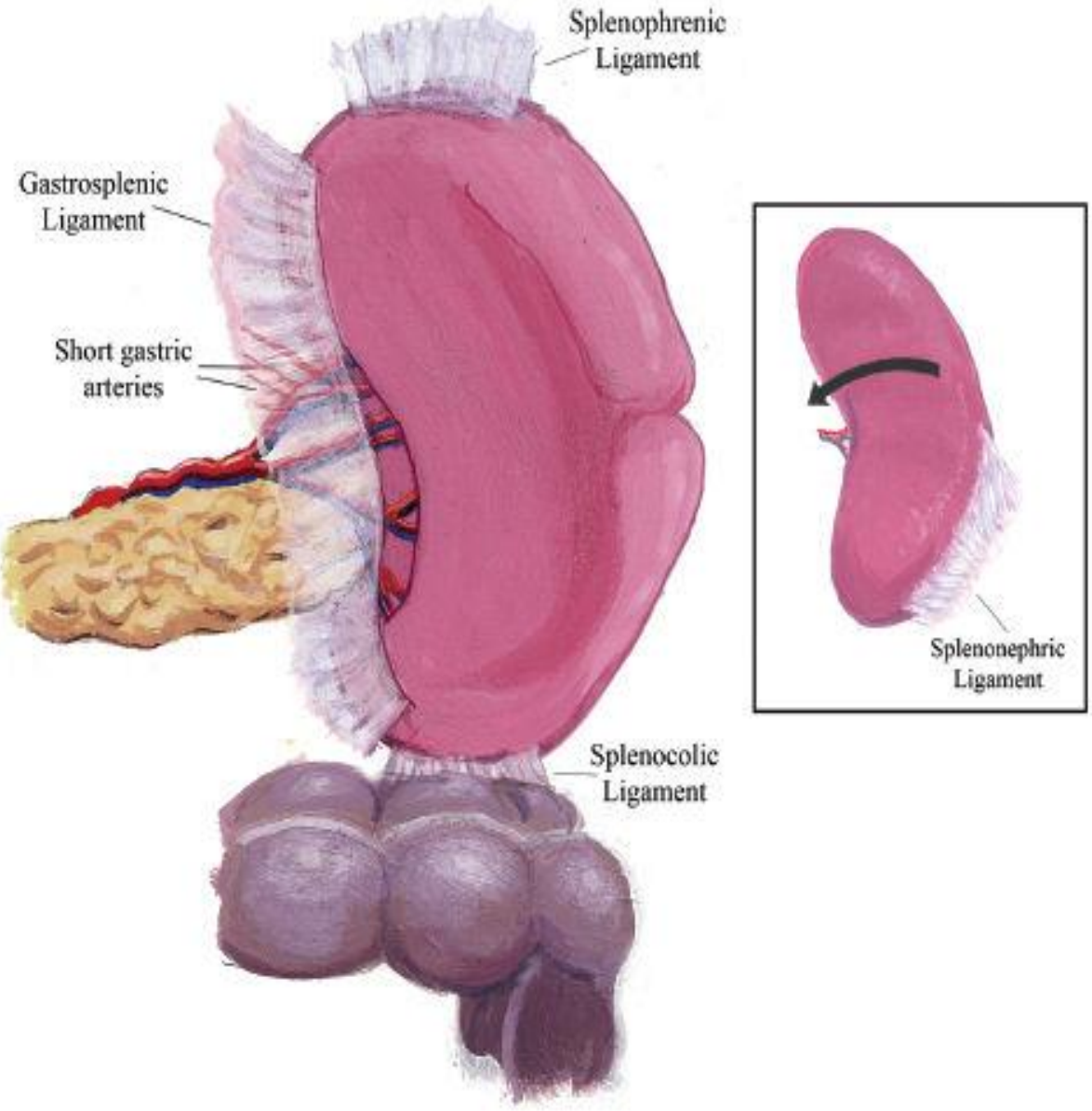


Fig 3- Ligaments of spleen

ACCESSORY SPLEEN (Fig 4)**:**

The classification includes two types of accessory spleens made of blood, sinuses and Malphigian bodies:

(1) The most common is a separate distinct mass usually found in patients with hematological disorders. It has been reported in about 14-30% of the patients.

(2) The uncommon is the constricted part of the main organ, to which it is attached by a fibrous tissue

The splenic artery also supplies the accessory spleen. They occur in decreasing order of frequency in the hilus of the spleen, the gastrosplenic ligament, splenorenal ligament, and the great omentum.

In females, accessory spleen may be seen in the pelvis, either in the presacral region or adjacent to the left ovary, and in males, in the scrotum in juxtaposition to the left testicle.

The spleen is surrounded by a capsule that is 1-2 mm thick and made up of trabeculae that invaginate the pulp. The parenchyma of the spleen acts as the immunological organ and is made of white pulp. The red pulp phagocytizes the particulate matter from the marginal zone and the blood. The white pulp is made of lymphatic nodules with germinal centers and periarterial lymphatic sheaths that constitute a reticular network filled with

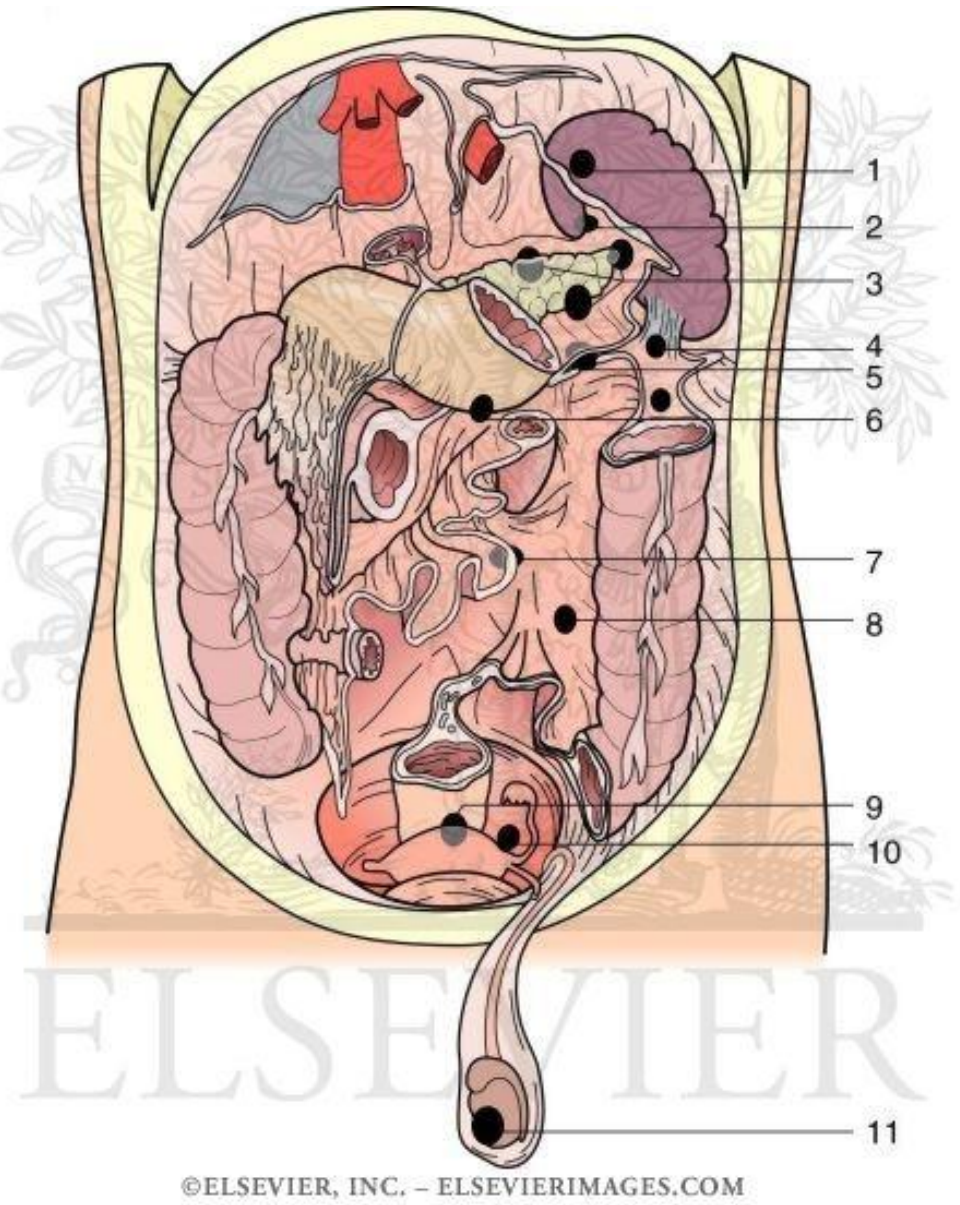


Fig 4- Usual location of accessory spleens: (1) gastrosplenic ligament, (2) splenic hilum, (3) tail of the pancreas, (4) splenocolic ligament, (5) left transverse mesocolon, (6) greater omentum along the greater curvature of the stomach, (7) mesentery, (8) left mesocolon, (9) left ovary, (10) Douglas pouch, (11) left testis.

lymphocytes and macrophages. It lies centrally and surrounds a central artery (Fig 5).

The marginal zone contains lymphocytes and macrophages and red blood cells (RBCs) that have exited from terminal arteries. The marginal zone also contains the marginal sinus that filters material from the centrally located white pulp.

It lies peripheral to the white pulp and contains end arteries arising from the central artery and from peripheral penicilliary arteries. Locally produced immunoglobulins enter the marginal zone, eventually coursing to the blood stream. Peripheral to the marginal zone lies the red pulp. This pulp consists of cords and sinuses that contain cellular elements of blood in transit. Most of the blood flow passing through the spleen courses through an "open" circulation in which the blood passes from arterioles to reticular cell-lined networks of the splenic cords and to the sinuses.

PHYSIOLOGY:

The spleen though is not a life saving organ, it performs some major functions that can be grouped into two categories:

(1) Those that are immunological in nature

(2) Those that are related to the circulating blood and blood products.

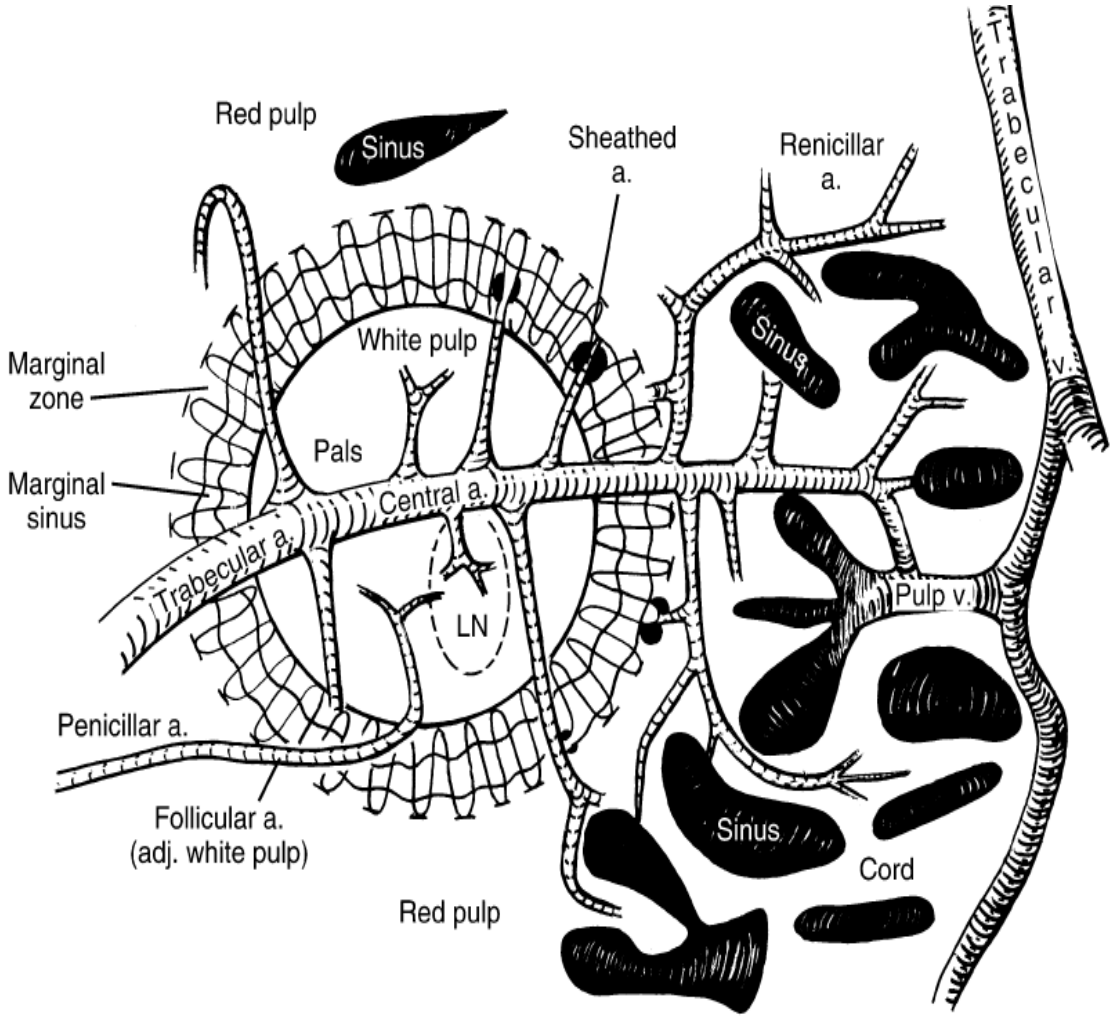


Fig 5 - Splenic compartments and potential vascular supply routes.

The cellular functions mainly concern hematopoiesis, storage, "pitting" and "culling". Hematopoiesis, essentially ceases by the seventh month in intrauterine life. It supplies erythroid, myeloid, lymphoid cells, and platelets in fetal life,. In human beings, the spleen does not serve as an

important reservoir for blood cells, except platelets. At any given time, about one third of the total platelet mass is in the spleen.

“Pitting” involves the removal of rigid structures from red cells such as Heinz bodies, Howell-Jolly bodies, and hemosiderin granules. This process concerns the elimination of nondeformable intracellular substances from deformable cells. The rigid body is phagocytized while the deformable cytoplasmic mass passes into the sinus and returns to the general circulation. The postsplenectomy blood smear is usually characterized by circulating erythrocytes with Howell-Jolly and Pappenheimer bodies (siderotic granules). Nucleated cells also have their nuclei removed in the same manner.

“Culling” refers to the spleen's ability to remove red cells that are aged or abnormal. Normally, as the red cell ages after a life span of approximately 120 days, it loses osmotic balance and membrane integrity, and therefore deformability. When these cells lose their deformability they are phagocytized by native macrophages. The spleen does not represent the only site for red cell destruction, and there is no difference in red cell survival following splenectomy. Naturally deformed cells and red cells that are affected by disease states are also removed by phagocytosis. In those instances in which there is a superabundance of reticulocyte formation, these

cells are being remodelled in the spleen and exit as mature cells. In the normal adult, the spleen is the most important site of selective erythrocyte sequestration. During its 120-day life cycle, the red cell spends an estimated minimum of 2 days within the spleen which, when normal, contains about 25 mL RBCs.

The neutrophil has a half-life of about 6 hours; thus 85% of neutrophils either emigrate at random into tissues or are destroyed within 24 hours. The role of the spleen is amplified in some hypersplenic states, with resulting neutropenia though the role of the spleen in the destruction of neutrophils under normal conditions is not well quantified. This removal can occur because of splenic enlargement and accelerated sequestration of granulocytes or because of enhanced splenic removal of altered granulocytes, as witnessed in immune neutropenias.

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. With splenomegaly, a large proportion of platelets is sequestered in the spleen (up to 80%) and this, coupled with accelerated platelet destruction in the spleen, accounts for thrombocytopenia. Splenic

phagocytosis of platelets occurs in normal states, but in pathologic states, such as immune thrombocytopenia, it is greatly increased.

Apart from the phagocytosis of antibody-coated cells, the immunologic functions of the spleen also include generation of lymphocytes, production of tuftsin, opsonins, properdin, and interferon and antibody synthesis (especially immunoglobulin M [IgM]).

The general indications for splenectomy are discussed in Table 1.

Hemolytic anemias	Purpuras	Secondary hypersplenism	Primary hematological disease	Other diseases
Hereditary spherocytosis	Idiopathic thrombocytopenic purpura (ITP)	Cirrhosis	Lymphoma	Pancytopenia in Felty's syndrome
Elliptocytosis		Cystic fibrosis	Chronic lymphocytic leukemia	Sarcoidosis
Enzyme defect hemolytic anemia	Thrombotic thrombocytopenic purpura (TTP)	Myelo- and lymphoproliferative disease	Myeloid metaplasia	Gaucher's disease
				Trauma
Thalassemia			Myelo- und lymphoproliferative disease	
Sickle-cell disease				Abscess
Idiopathic hemolytic autoimmune anemia				Splenic artery aneurysm
				Cyst
				Tumor

Table 1- Indications of splenectomy

ABBREVIATED INJURY SCALE [1]

In 1969 an anatomical scoring system Abbreviated Injury Scale (AIS) was introduced. The score has been revised and updated to provide an accurate ranking of the severity of injury. The latest revision of the AIS score was made in 1998. Association for the Advancement of Automotive Medicine monitors the AIS.

Injuries are given a score from 1 to 6, with 1 being minor, 2 moderate, 3 serious, 4 severe, 5 critical and 6 un-survivable. The score indicates the 'threat to life' related to the injury and does not mean to represent a comprehensive measure of severity. The AIS cannot be defined as an injury scale, in that the difference between AIS1 and AIS2 is not the same as that between AIS4 and AIS5. There are many identical features between the AIS scale and the Organ Injury Scales of the AAST.

INJURY SEVERITY SCORE (ISS) & NEW INJURY SEVERITY SCORE (NISS) [2]

The Injury Severity Score (ISS) is a versatile scoring system based on anatomic classification that provides an overall score for those patients who have sustained multiple injuries.

Each injury is allotted AIS and is assigned to any one of six body regions (Head, Face, Chest, Abdomen, Extremities (including Pelvis) and

External). Only those with the highest AIS score in each region of the body is taken into account. Sum of the squares of the three most severely injured organs will provide the injury severity score.

The ISS score ranges from 0 to 75. If an injury is associated with an AIS of 6 (un-survivable injury), the ISS score is by default assigned to 75. The ISS score is the only scoring system in use and correlates proportionally with hospital stay, mortality, morbidity and other measures of severity.

Its disadvantage is that any fault in the AIS scoring increases the error in ISS scores exponentially. Many different injury patterns can result in the same ISS score and injuries to different body regions are not taken into account. Since the full description of the patient injuries is incomplete prior to complete investigation & operation, the ISS (along with other anatomical scoring systems) is not very useful as a triage tool.

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.

SPLENIC INJURY

In the national trauma data bank, spleen is the most common organ involved in injuries to abdomen which accounts for 3.2% of all injured and

50.7% of blunt abdominal trauma patients. Similarly, 2.6% of injured persons sustained splenic injuries in a multicentre series with data from 1993 to 1997 [3].Anyone who treats blunt abdominal injury should have the ability to manage splenic injuries, since it has a mortality of 10.8%. This is mainly caused by associated injuries and pre-hospital delay.

A deceleration mechanism that tears the splenic capsule or parenchyma, at areas fixed to the retro-peritonium or direct compression of spleen in the left upper quadrant is the patho-physiology of splenic injuries.

A ruptured spleen, at the time of presentation can have ongoing bleed or the bleed would have stopped, which allows these injuries to be managed non-operatively. The re-initiation of bleeding can be delayed in splenic injuries, which is a concern among patients treated non-operatively. 10.6% have late bleeding in a large series, though it varies with the grade of injury [3].

Penetrating splenic trauma is less common. But still In national trauma data bank, it represents 14.5% of all penetrating abdominal injuries. Of the penetrating injuries involving spleen, this is higher compared to 9.2% and 7.6% reported in a large series from Grady memorial and Ben Taub hospitals during 1980s and 1990s, respectively [4].

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.

The splenic parenchyma is maximally enhanced with the contrast in portal venous phase, while still the vasculature is visualised and the images are taken. Disruptions in the splenic parenchyma represent splenic injuries, with surrounding hematoma and free intra-abdominal fluid.

Active extravasation of contrast, seen as a high-density blush, contained within a pseudoaneurysm or bleeding into the peritoneal space can be identified occasionally. Injury to the hilar vessels causing complete splenic devascularization or subcapsular hematoma can be other findings.

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.

Angiography has been the recent advance in splenic injury management to evaluate further and also to treat at times. This is used in injuries with active extravasation by CT. As there is much risk of delayed

bleeding in non-operative management, some centres use angiography in all high grade injuries.

Specific sites of bleeding from the splenic parenchyma, segmental or trabecular vessels can be identified by angiography. Though the splenic parenchymal injury cannot be characterized by angiography, still it can be complementary to CT.

The ability to obstruct sites of bleeding endovascularly using angioembolization is the major benefit of angiography. Patients with active extravasation by CT, but candidates for non-operative management are benefited by angiography with embolization to eliminate splenic pseudoaneurysm. With evidences, there is increase in the rate of safety of managing splenic injuries non-operatively [5]. Angiographic evaluation and angioembolic treatment can be considered only in patients who demonstrate hemodynamic stability and not in shock.

Patients with blunt splenic trauma can be managed non-operatively with approppriate patient selection and it should not be overraated. Splenectomy does not have a risk profile when compared to the implications of hemorrhage, and should not be overlooked as a definite treatment for splenic bleeding.

GRADING OF SPLENIC INJURIES

Grade		Description
1	Hematoma	Sub-capsular,<10% surface area
	Laceration	Capsular tear, <1 cm parenchymal depth
2	Hematoma	Sub-capsular, 10-50% surface area Intra-parenchymal, <5 cm in diameter
	Laceration	Capsular tear, 1-3 cm parenchymal depth which does not involve a trabecular vessel
3	Hematoma	Sub-capsular, >50% surface area or expanding Ruptured sub-capsular or parenchymal hematoma Intra-parenchymal hematoma >5 cm or expanding
	Laceration	>3 cm parenchymal depth or involving trabecular vessels
4	Laceration	Laceration involving segemental or hilar vessels producing major devascularization (>25% of spleen)
5	Laceration	Completely shattered spleen
	Vascular	Hilar vascular injury which devascularizes spleen

*** Advance one grade for multiple injuries up to grade III**

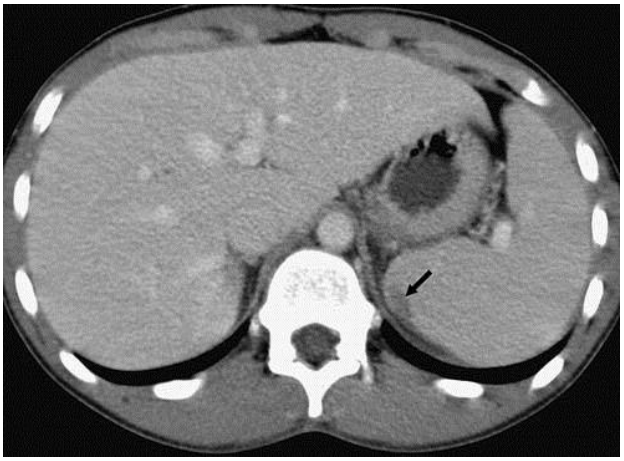


Fig 6- Grade I: sub-capsular fluid involving < 10% of the splenic surface.

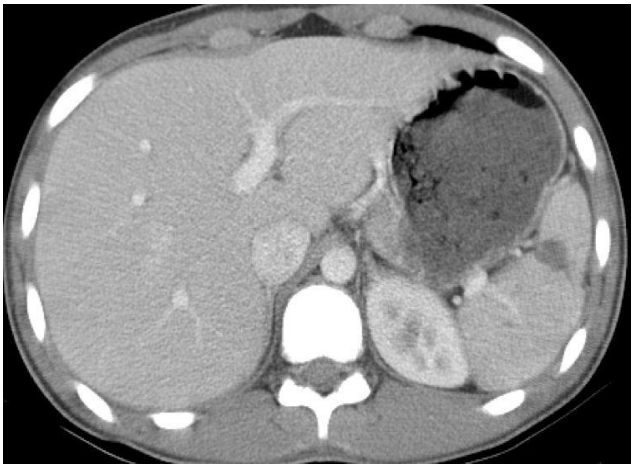


Fig 7- Grade I: sub-capsular fluid involving < 10% of the splenic surface. Capsular tear < 1cm depth.

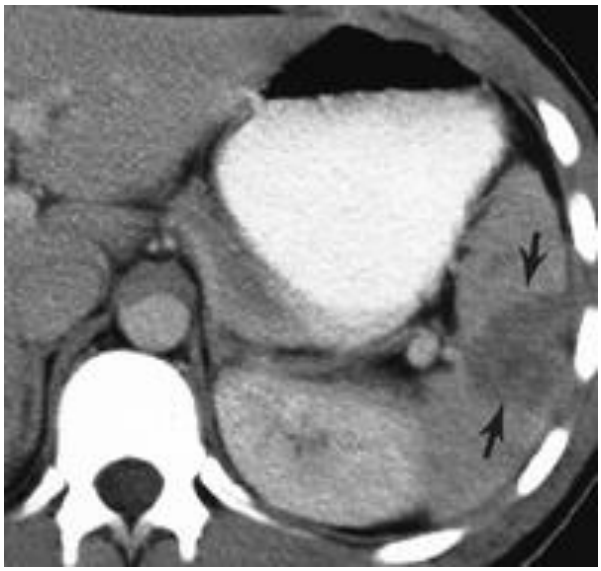


Fig 8- Grade II: sub-capsular hematoma, 10% to 50% surface area; intra-parenchymal hematoma, < 5 cm in diameter

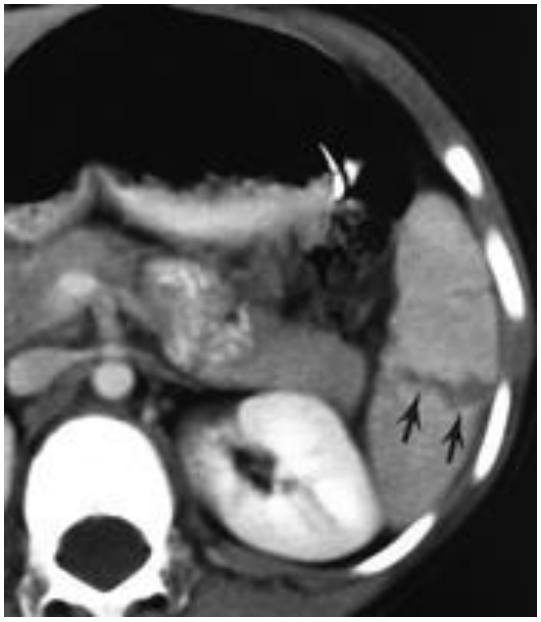


Fig 9- Grade II: capsular tear, 1 to 3 cm parenchymal depth that does not involve a trabecular vessel

Fig 10- Grade III: sub-capsular hematoma, laceration and sub-capsular contrast extravasation.

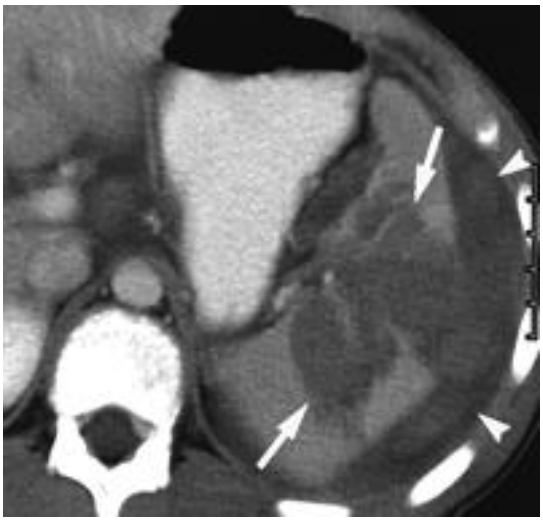


Fig 11- Grade III: laceration of more than 3 cm in depth radiating from the splenic hilum.

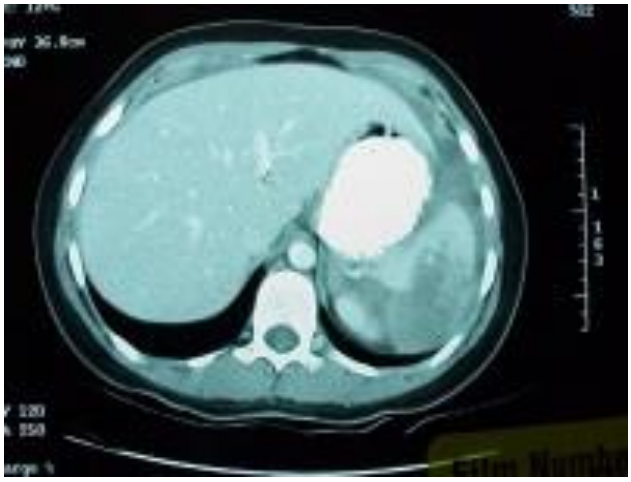


Fig 12- Grade IV: laceration involving segmental or hilar vessels producing major devascularization (>25% of spleen).

Fig 13- Grade V: shattered spleen and hilar vascular injury.

Fig 14- Splenic injury with sub-capsular hematoma. Despite only a 1-cm capsular tear, this injury demonstrated ongoing hemorrhage.

In an attempt to push the non-operative envelope, no patients with bleeding should be managed without splenectomy. It is possible to find appropriate candidates for non-operative management of splenic injury based on patients' physiology. Non-operative management also requires infrastructure to provide the ongoing surveillance.

To manage a patient without surgery, hemodynamic stability is a prerequisite and is indicated by absence of tachycardia, normal blood pressure, no findings suggestive of shock and absence of metabolic acidosis. 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.

There are other factors which have an impact while considering non-operative management, though the patient's physiological status is the most important. On comparing failure rates between groups older and younger than 55 years, two retrospective studies have provided opposite conclusions [6, 7].

A rate of 19% versus 10% failure of non-operative management in patients older than 55 years was demonstrated by larger of these studies [7] but still there is a success rate of 80% among older patients managed non-

operatively. This proves age alone is not a contraindication for managing splenic injuries without surgery.

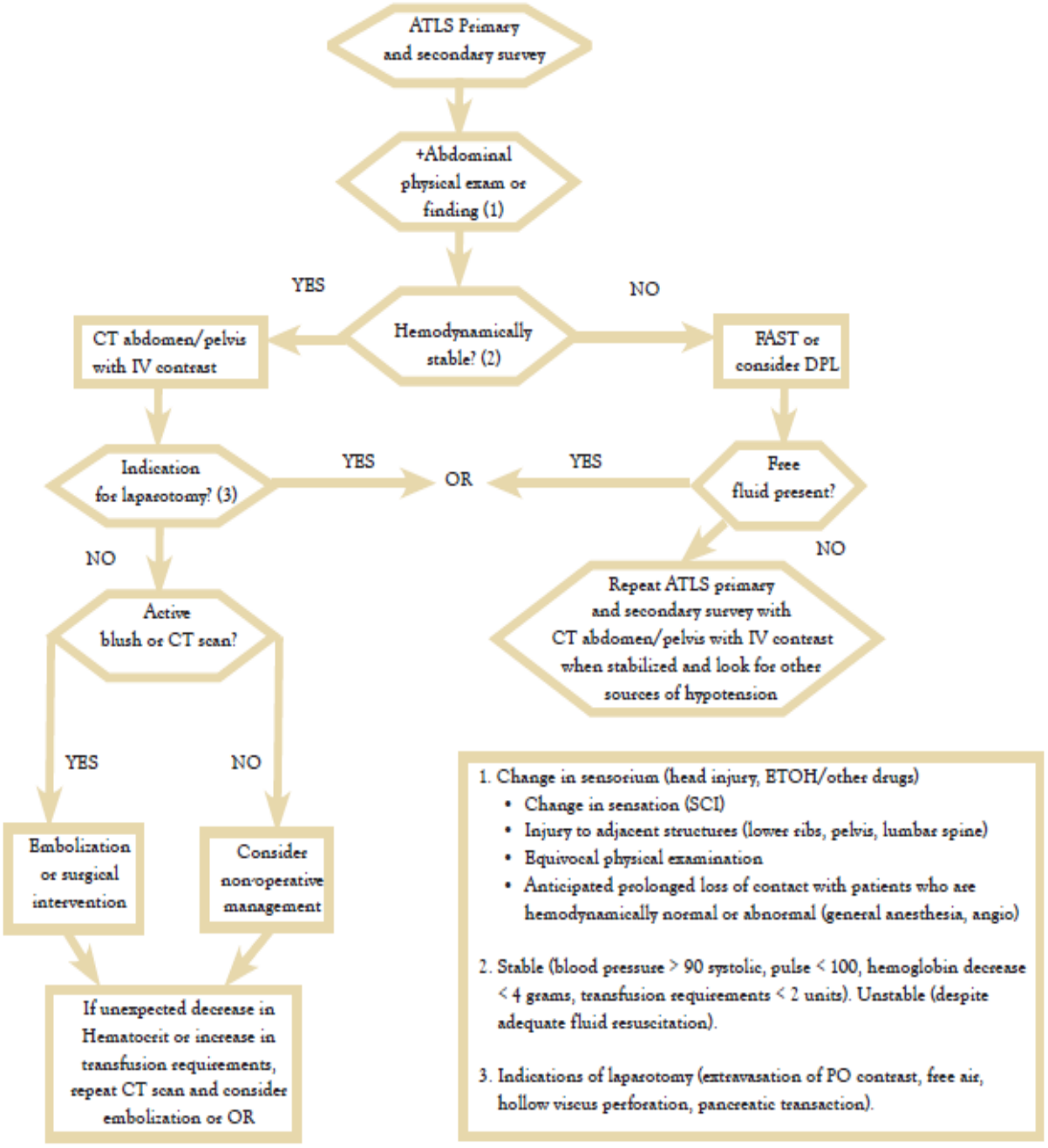


Fig 15 – Non-operative management algorithm for blunt injury to the Spleen

Another factor which has an impact on decision making is the grade of splenic injury identified on imaging during admission, but there is no prospective study to provide guidelines. A failure rate of 33.3% in grade IV and 75% in grade V injuries, with 8% of failures happening more than 9 days after injury has been reported by one multi-institutional retrospective study conducted by EAST. While all high grade injuries failed with non-operative management in another multicenter study.

Some believe that splenectomy does not have a very high morbidity, as there are unacceptably high failure rates with high grade splenic injuries, and almost one in ten after hospital discharge. Others believe it to be managed non-operatively, despite high failure rates. Non-operative management is reserved for grade I, II injuries and isolated grade III injuries. The decision is mainly a personal preference and is guided by surgical intuition.

In case of instability at admission, unknown bleeding site, after failed non-operative management, when spleen is suspected to be the cause preoperatively, operative management is considered.

Splenic injury secondary to penetrating abdominal trauma is usually identified during laparotomy and should be addressed based on the presence

or absence of ongoing bleeding. In damage control, splenic injury can be packed but, more commonly splenectomy is performed.

OPEN SPLENECTOMY

A midline incision with packing of all four quadrants is the best approach when instability is present. Left upper quadrant is exposed with a fixed retractor.

The attachments are exposed by retracting the spleen postero-medially, and dividing the peritoneum laterally marks the beginning of splenectomy. The peritoneum is divided at the white line of Toldt and the dissection begins at the splenocolic ligament extending superiorly, until encountering the short gastric vessels.

After taking down the peritoneum, a blunt plane is created posterior to the spleen in the medial direction that extends behind the tail of pancreas. The spleen is delivered in to the visualized wound after mobilising the entire spleen and distal pancreas.

The short gastric vessels are identified and ligated, avoiding injury to greater curve of stomach. The tail of pancreas is protected while the hilar vessels are clamped and ligated. Drains are placed only if the tail of the pancreas was also injured.

To protect against infections by encapsulated bacteria including Streptococcus pneumoniae, Neisseria meningitidis and Haemophilus influenzae, postsplenectomy vaccines must be provided. Since patients who usually benefit from splenic salvage techniques are managed non-operatively, these techniques are less commonly used now.

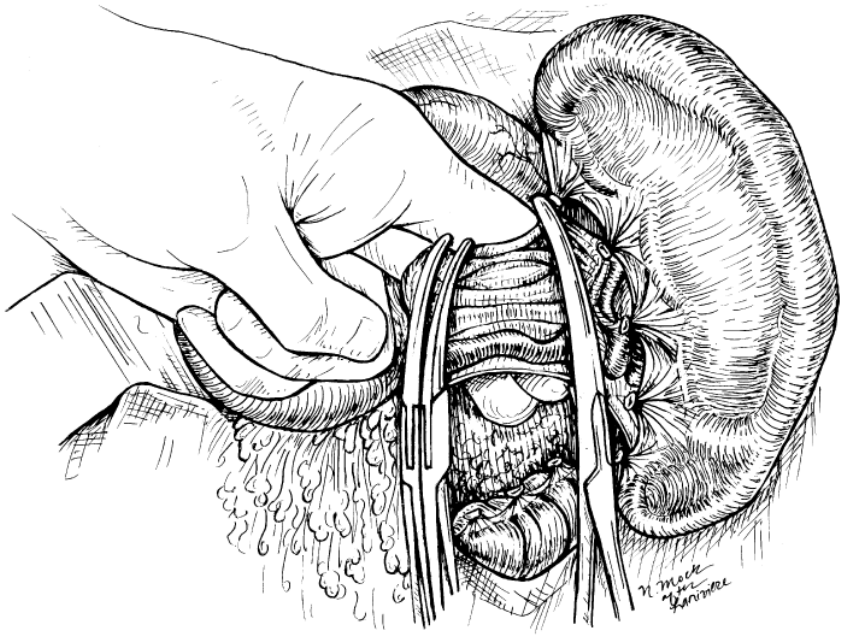


Fig 16- Three-clamp method of Federoff for transecting the splenic hilum. The fingers protect the tail of the pancreas while the clamps are applied.

POST-SPLENECTOMY LYMPHOCYTOSIS

Pathophysiology

The kinetic aspects of lymphocyte trafficking is responsible for post-splenectomy leukocytosis. The mounting of an efficient immune response requires fast mobilization and distribution of lymphocytes. Lymphocytes migrate from the blood cross a specialized endothelium of post-capillary venules into lymph nodes [8, 9] and exit from lymphoid tissue via efferent lymphatics and the thoracic duct. Most lymphocytes circulate through the spleen, where direct access to the marginal zones does not require their migration across a specialized endothelium.

The total lymphocyte pool in is estimated to be approximately $50*10^{10}$ cells out of which only $10\text{-}15*10^{10}$ lymphocytes circulate through lymphoid tissues [41]. At a given time only a small proportion of $1*10^{10}$ lymphocytes circulate in the blood of an average adult with a short transit time of 20-36 minutes for exchangeable cells as shown in fig 17 [10]. This results in a high turnover of migratory blood lymphocytes of 50 times per day and a totally daily exchange of approximately $50*10^{10}$ lymphoid cells between blood and tissues.

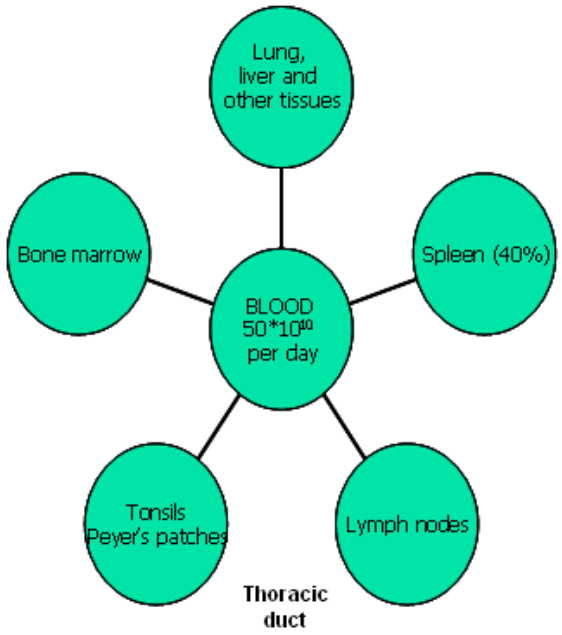


Fig 17- Lymphocyte traffic between blood and organs

As only 6% of the exchanging cell pool passes through the thoracic duct, the majority of cells migrate to tissues other than lymph nodes after leaving the bloodstream.

The spleen is the organ with the highest lymphocyte uptake during the early stages of recirculation. Animal studies have demonstrated a splenic uptake of approximately 40% of radiolabelled cells within the first 2 hours of injection [11, 12]. Between 18 and 24 hours of injection labeled 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, though not quantified, splenectomy may result in neutrophilia. As a result slower clearance of lymphocytes and decreased destruction of neutrophils, there is physiological leukocytosis post-splenectomy.

Post-splenectomy thrombocytosis

Spleen plays major role in regulation of platelets. It is the site where platelets are being destroyed. Hence there is physiologic thrombocytosis after splenectomy predisposing to thrombocytosis in later stages.

COMPLICATIONS OF SPLENECTOMY

- Intra-operative
    - Haemorrhage
    - Injury to tail of pancreas
    - Colon and gastric injuries
    - Diaphragmatic injuries

- Early
    - Haematoma
    - Pancreatic leak
    - Pneumonia
    - Intra-abdominal abscess
    - Wound infections
    - Thrombotic complications
- Late
    - Overwhelming post-splenectomy infection (OPSI)
    - Splenosis

POST_OPERATIVE INFECTIONS

Surgical infections were earlier considered as those that require surgical therapy (e.g., complicated intraabdominal infections) and skin or soft tissue infections. Since patients undergoing surgery suffer from nosocomial infections post operatively, the definition now includes any infection that affects post operative patients. Surgical site infections, central line associated blood stream infections, urinary tract infections, hospital or ventilator acquired pneumonia are some of the post operative infections.

Inherent invasiveness of surgery is responsible for creating portals of entry for the pathogens to break the natural epithelial barriers and invade the host. Surgical illness is an immunosuppressive state similar to the therapeutic suppression following organ transplantation.

Endotracheal intubation and mechanical ventilation following general anaesthesia associated with a period of reduced consciousness poses a risk of aspiration pneumonitis. Since the development of infections post surgery impedes the recovery it is necessary to recognize and minimize the risks and follows an aggressive approach to diagnose and treat post operative infections.

Though infections are preventable it is prudent for every physician in contact with his patients, do their utmost to prevent infections. Since no method is universally effective an ensemble of prevention method is required. It is necessary to take good and strict aseptic care of surgical wounds regularly and dressing done as and when necessary under strict aseptic precautions.

Moreover, one must avoid drains and catheters and if placed it must be removed as early as possible. Role of antibiotics as therapeutic and prophylactic modality must be well understood and used judiciously to prevent the development of multidrug resistant organisms.

RISK FACTORS FOR INFECTION

Host Factors

Innate immunity is responsible for providing epithelial barrier preventing the invasion of foreign antigens.

Potential pathogens are present universally and though colonization may occur even in healthy host, it is necessary for a portal of entry to be present for invasion of organisms to occur which may be seen in surgical patients with surgical wound and intravenous catheters.

Inflammatory response stimulates autonomic nervous system which promotes gluconeogenesis and glycogenolysis, promotes coagulation pathway, and proinflammatory cytokines are stimulated to begin the tissue repair process [13]. Innate and adaptive immunity are depressed by action of cortisol [14].

Age > 64 years is the most important risk factor for adverse outcomes post operatively especially post operative infections which are mainly due to immune senescence [15]. Hyperglycemia induces immune cell dysfunction and thereby causing infection in both diabetic and non diabetic patients.

Factors causing increased risk of post operative infections [4]

- 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

Controversies exist whether there is any sex predilection for post operative infections. Various studies that have been conducted and no study could conclude about sex predilection for post operative infections [15, 16].

Genomic variability may be correlated with disease susceptibility in infections. Nucleotide structures of genes containing single nucleotide polymorphisms (SNPs) and single point mutations related to inflammation (e.g., tumor necrosis factor-α [TNF-α], interleukin [IL]-1, IL-6, and IL-8), the anti-inflammatory response (e.g., IL-10, IL-1 receptor antagonist), the

innate immune response (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 [17].

`Due to heterogeneity in the infection severity and resultant mortality, as well as the immune response and infection predisposition, makes it difficult to make conclusions that a single nucleotide polymorphism will characterize the risk of infection in an individual.

INTERACTION BETWEEN THE THERAPY AND THE HOST

Impairment of host defences, resuscitation and definitive care along with the injury itself increases the risk of infection.

Hypothermia

Hypothermia occurs due to exposure, infusion of huge quantities of unwarmed intravenous fluids and blood products or evaporative losses that occur due to intra-cavitatory surgery, especially when abdomen and chest are opened.

To preserve core heat, peripheral and cutaneous vasoconstriction occur which causes decreased microcirculatory blood flow. This is

aggravated by hypovolemia, inflammatory response, coagulation pathway activation and decreased transfused red blood cell deformability [18]. Thus hypothermia is immunosuppressive, affects cardiovascular performance and increases mortality after trauma and surgery [19, 20].

Tissue hypoxia

Injury to the face, airways, lungs, or chest wall, inability to secure the airway, massive blood loss, cardiovascular instability, disruption of the microcirculation and acute respiratory distress syndrome (ARDS) are few factors that are responsible for tissue hypoxia after trauma. This hypoxia in turn predispose to surgical site infections [21]. Oxygen administration post surgery may reduce the risk of surgical site infection after elective surgery [20].

Manner of Resuscitation

Intravenous fluid resuscitation is very important to restore hemodynamics and microcirculatory perfusion, but the amount and nature of fluid to be administered is still under discussion. Earlier crystalloids were preferred over colloids due to cost effectiveness and almost equivalent results [22]. Delaney and his associates conducted a meta-analysis of

seventeen trials, eight trials were exclusively of crystalloid versus colloid for the treatment of patients in sepsis. Results showed decreased mortality with colloid resuscitation [23]. To sum up resuscitation of immune system is the most important determinant, failure of which may lead to increased risk of nosocomial infections and death [24].

Blood Transfusion

Though blood transfusion is life saving after trauma or hemorrhage, there is increased risk of infection post transfusion. Altered leukocyte antigen presentation and a shift to the T helper 2 cell phenotype causes immuno-suppression following blood transfusion. An exponential relationship between transfusion and infection risk among trauma patients, detectable even after single unit transfusion, and becoming a near certainty after more than 15 units of transfused blood has been suggested by Claridge and his associates [25, 26].

Another study has suggested risk of infection is increased three times in surgical patients following transfusion and five times increased risk in trauma patients following transfusion. Ventilator associated pneumonia, catheter line associated blood stream infections and critically ill patients are also associated with increased risk of infection following transfusion [27].

Prolonged storage of banked blood results in the loss of high energy membrane phosphates which in turn leads to impaired red blood cell deformability, disruption of microcirculation, and impaired oxygen delivery [28]. As a result blood transfusion does not increase oxygen consumption [29] but instead increase organ dysfunction [30]. So it is better to be conservative while deciding on blood transfusion to stable patients in intensive care unit [31].

Control of blood sugar

Hyperglycemia reflects the catabolic state and surgical stress associated insulin resistance along with impairing the immune function of the host. Inadequate glycemic control during the pre-operative and post-operative period increases the risk of infection and worsens the outcome from sepsis in both diabetic and non diabetics.

Hyperglycemia that is more than 200 mg/dl is associated with four times increased risk of surgical site infection in both cardiac and non cardiac surgery. Insulin infusion kept to maintain the blood glucose level less than 110 mg/dl was associated with 40% decrease in mortality among critically ill post-operative patients and also lesser incidence of nosocomial infections and organ dysfunction [32].

Effects of Hyperglycemia upon 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 Metabol*ism**

• Enhanced peripheral glucose uptake

• Hyperlactatemia

• Increased gluconeogenesis

• Depressed glycogenolysis

• Peripheral insulin resistance

Glycemic control in post-operative infection has become controversial because of nonconfirmation of a salutary effect in intensive care patients. Because of the risk of hypoglycemia following insulin therapy, the maintenance blood glucose level has been increased from 110 mg/dl to 140-180 mg/dl [31]. Greisdale and associates conducted a meta-analysis and found that intensive insulin therapy to maintain blood glucose level has been found to greatly reduce the mortality in those patients treated in intensive care unit

regardless of diabetes status. Certain contradicting opinions are still prevailing [21].

Nutritional support in general is very crucial, keeping in mind that restoration of anabolism requires calories and nitrogen in excess of basal requirements of 25 to 30 kcal and 1 g nitrogen/kg/day. It is very difficult to provide adequate calories and protein without producing hyperglycemia.

Parentral feeding has no advantage over other modes of feeding because of inherent central line associated complications [34]. Enteral feeding within 2 days, perhaps as quickly as possible if gut is functional, is beneficial except in those patients where we suspect intestinal ischemia and in those patients where we would like to prevent aspiration pneumonia. In a meta-analysis of 15 randomized control trials it was found that there was 55% reduction in the risk of infection in those patients taking enteral feeding following trauma, surgery or burns [35].

INFECTION CONTROL

It is very important to abide by the general principle of surgical care, critical care and infection control at all times. One must rapidly resuscitate the patients, either over resuscitation or under resuscitation increases the risk of infections. It is also necessary to immediately identify the underlying

pathology and treat it as soon as possible. Central venous catheters must be removed and replaced if inserted under sub optimal conditions (e.g., lack of cap, mask, sterile gown, and sterile gloves for the operator and a full bed drape for the patient). This should be followed by re-insertion in a new site as soon as the patient's condition permits. Avoid placing drains and if placed it must be removed as quickly as possible [36].

Control of infections is both individual and collective responsibility. The most effective way of controlling infection is hand hygiene, compliance of which is very challenging. Universal methods of precaution must be carried out. Alcohol based hand cleansers are the most effective in clearing all organisms except the clostridium spores for which washing with soap and water is found to be very effective [37].

The source of most bacterial pathogens is found to be endogenous flora. Skin surfaces, artificial airways, gut lumen, wounds, catheters, and inanimate surfaces (e.g., bed rails, computer terminals) may become colonized [38]. The most common mode of entry of pathogens is fecal-oral route but health care workers transmit the organisms by their hands.

Isolation of contacts is the most important part of infection control and must be used selectively to prevent pathogen spread such as Methicillin Resistant Staphylococcus Aureus , Vancomycin Resistant Enterococci or

multidrug resistant gram negative bacilli. There should be balance attained because reduced manpower in ICU will result in increased incidence of nosocomial infections [39].

Catheter care

Proper catheter care includes avoidance when not needed, proper skin preparation, protecting the barriers during insertion, appropriate catheter selection (antiseptic or antimicrobial coated), neat and sterile dressing of indwelling catheters and removal of catheters as soon as when no longer needed, or as is practicable, but no longer than 24 hours, after insertion of catheters under less ideal circumstances.

Advantages and disadvantages of catheter insertion must be weighed including the risk of infection. Though all the indwelling catheters carry the risk of infection, non-tunneled central venous catheter and pulmonary artery catheters pose the highest risk for local site infections and central line associated blood stream infections. 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 [40].

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 [41, 42, 43, 44, 45].

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 [46, 47].

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 [48, 49].

SPECIFIC INFECTIONS

Surgical site infections

Surgical procedures are classified into

- Clean procedures
- Clean contaminated procedures
- Contaminated procedures
- Dirty procedures

Clean surgical procedures affect only skin structures and soft tissue. Under controlled setting when a hollow viscus is opened it is termed clean contaminated procedure (e.g., elective aero-digestive or genitourinary tract surgery). Contaminated procedures include those procedures where a large inoculum of microorganism is introduced for a short duration into an otherwise sterile body cavity for an infection to get established during surgery (e.g., penetrating abdominal trauma, enterotomy during adhesiolysis

for mechanical bowel obstruction). In cases of established contamination and infection if a surgery is performed it is termed dirty procedure (e.g., colon resection for perforated diverticulitis).

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. Due to increase in number of ambulatory surgeries the incidence of surgical site infections is under-estimated since the infection in ambulatory surgery is seldom reported.

Risk factors for development of surgical site infections includes

(1) Patient Factors

- Ascites (for abdominal surgery)
- Chronic inflammation
- Corticosteroid therapy (controversial)
- Obesity
- Diabetes
- Extremes of age
- Hypocholesterolemia
- Hypoxemia
- Peripheral vascular disease (for lower extremity surgery)
- Postoperative anemia
- Prior site irradiation
- Recent operations
- Remote infection
- Skin or nasal carriage of staphylococci
- Skin disease in the area of infection (e.g., psoriasis)
- Undernutrition

(2) Environmental Factors

- Contaminated medications

- Inadequate disinfection/sterilization
- Inadequate skin antisepsis
- Inadequate ventilation

(3) Treatment Factors

- Drains
- Emergency procedures
- Hypothermia
- Inadequate antibiotic prophylaxis
- Oxygenation (controversial)
- Prolonged preoperative hospitalization
- Prolonged operative time [50]

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 [51, 52, 53].

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.

A study was conducted in 5031 patients who underwent non-cardiac surgeries and incidence of surgical site infection was found to be 3.2%. Ascitis, diabetes mellitus, post-operative anaemia and recent weight loss were considered independent risk factors responsible for surgical site infections. However chronic obstructive pulmonary disorders, tobacco use and corticosteroid use did not contribute to the above incidence.

In another study conducted prospectively it was found that 12.5% of the 9016 patients included in the study became infected within 28 days of surgery. Analysis revealed that certain factors like low serum albumin, tracheostomy, old age and amputation were responsible for early infections whereas a shunt for dialysis, vessel repair and early infection were responsible for hospital readmission. Hence risk factors for 28 day mortality are increased age, hypoalbuminemia, increased serum creatinine levels and an early infection.

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 [81]. It is said that there is increased incidence of surgical site infections during elective colon surgery and diverse operations due to mild intra-operative hypothermia [54]. Controversies exist whether peri-operative oxygen administration is a boon for infection prevention [55]. 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.

Closure of a contaminated or a dirty wound is associated with very high risk of surgical site infection. Now there has been a rapid increase in open abdomen techniques of temporary abdominal closure for managing trauma and severe peritonitis. From the results of a retrospective analysis it has been found that antibiotic prophylaxis has no role in treating open abdomen [56] but inability to close the abdomen is linked with increased incidence of infections but not mortality [57].

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 [58, 59]. As a result drains must be avoided as much as possible and removed as early as possible. Moreover in cases of retained drains prolonged antibiotic prophylaxis is not recommended to cover the indwelling drains.

Regarding wound irrigation there are controversies regarding their role in reducing the risk of infection. It has been found that compared to routine low pressure saline irrigation high pressure irrigation may be beneficial. Risk of surgical site infections can be reduced by application of intra-operative topical antibiotics. To prevent the development antibiotic resistance it is better to use an anti septic rather than an antibiotic [60, 61, 62].

Diagnosis of surgical site infections is established clinically. Clinical signs and symptoms depend on the depth of infection which may occur as early as post-operative day 4 or 5, though necrotising fascitis of surgical site may occur very early i.e. within 24 hours after surgery. Signs of infection

range from simple induration to pain, warmth, erythema, edema and tenderness, finally ending in wound discharge.

In those patients with deep incisional surgical site infections, tenderness may reach beyond the margins of erythema, and crepitus, vesicles and bullae may develop in the skin. Systemic inflammatory response syndrome which is defined by the presence of two or more of fever, leukocytosis, tachycardia and tachypnea herald the development of sepsis in patients with ongoing sepsis. Symptoms correspond to organ system involved in intracavitary infections i.e. ileus, respiratory distress and failure, altered sensorium.

For superficial surgical site infections it is not necessary to take wound cultures especially in those cases where wound care and drainage are adequate enough to treat the wound site infection. Even if superficial wound swabs are collected, they may be easily contaminated with skin colonists. In patients suffering from deeper infections or hospital acquired infections, it becomes mandatory to send discharge samples for culture and sensitivity as opposed to open wounds which are already colonised.

Immediate surgical attention is needed for severe forms of surgical infection, especially necrotising soft tissue infections. Any delay in management of these patients may result in increased mortality.

Study conducted by Freischlag [63] and his co-workers has shown increase in mortality from 32% to 70% when the treatment is delayed for more than 24 hours. Patients with necrotising soft tissue infection must be subjected for immediate surgical debridement irrespective of identification of the causative pathogen or development of specific symptoms. Sequential debridement might be needed to control the infection.

The initial step in the treatment of surgical site infection is to expose the surgical wound and plan the further management [104]. In cases of superficial infections, providing local wound care is more than enough to treat the patient. Antibiotics are indicated only for those patients where erythema has extended beyond wound margins or in those scenarios where systemic signs of infection are present.

In cases of deeper infections it is necessary to do surgical exploration and wound debridement to achieve local control of wound infection. Surgical site infection must be considered as one of the factors responsible for delayed wound healing.

Organ or space surgical site infections are directly associated with surgical procedures and occur within the body cavity. These infections usually have an occult presentation mimicking incisional surgical site

infection and ending in an inadequate initial treatment. Such infections become apparent only when complications develop.

Organ or space surgical site infections are diagnosed by some form of imaging which confirms the site and extent of infection. Percutaneous or open drainage is necessary to rapidly control and treat the infection.

Vacuum assisted wound closure is a newer technique developed first by Morykwas and his colleagues. Vacuum assist control therapy helps in optimizing the blood flow, reduces the edema and sucks out the accumulated fluid providing clearance of microorganisms causing infection. Also it promotes healing of wound by contraction and promotes cellular proliferation. It is now being used to treat sternal infections following cardiac surgery, laprostomy wounds, complex perineal wounds and management of skin grafts [64, 65].

Various tactics for control of surgical site infections were brought together in a single project known as Surgical Care Improvement Project, the effectiveness of which at present is in doubt.

Previously another program named National Surgical Infection Prevention Project focused mainly on the antibiotic prophylaxis including the agent choice, timing of administration and prophylaxis duration.

An audit found that this antibiotic prophylaxis was inappropriate and timing of administration was suboptimal and about 60% percent of the patients had the antibiotics continued for more than 24 hours. Hence it is advised to start antibiotic prophylaxis 60 minutes prior to incision and must be continued for no longer than 24 hours.

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

1. Antibiotic Prophylaxis

- Proportion of patients who have their antibiotic dose initiated within 1 hour before surgical incision (2 hours for vancomycin or a fluoroquinolone)
- Proportion of patients who receive an approved antibiotic agent for prophylaxis consistent with current recommendations.
- Proportion of patients whose prophylactic antibiotics were discontinued within 24 hours of the surgery end time (48 hours for cardiac surgery)
- Clindamycin use is preferred for patients allergic to β-lactam antibiotics.

- Vancomycin is allowed for prophylaxis of cardiac, vascular, and orthopaedic surgery if there is a physician-documented reason in the medical record or documented β-lactam allergy.

2. Glucose Control (Cardiac Surgery Patients)

- Blood glucose concentration must be maintained <200 mg/dl for the first 2 days after surgery.
- Blood glucose determination closest to 6 am on postoperative days 1 and 2 (surgery end date is postoperative day 0) is monitored.

3. Hair Removal

- No hair removal should be performed; if hair is removed, clippers or a depilatory agent should be used immediately prior to surgery. Razors are not to be used.

4. Normothermia (Colorectal Surgery Patients)

- Core body temperature should be between 96.8° to 100.4°F within the first hour after leaving the operating room.

Though these factors have been enlisted, it does not cover all the factors responsible for controlling infection. Hence to bring about effective surgical site infection control it is necessary to execute an ensemble of prevention tactics apart from those enlisted above.

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. Being one of the most common infections in ICU its incidence is steadily increasing due to increasingly ill patients, incomplete diagnostic criteria, poor antibiotic choice and failure to define therapeutic end point. Presence of multidrug resistant pathogens has made it even more difficult to start empirical therapy and thereby resulting in inadequate antibiotic therapy and thereby increased mortality.

Early onset ventilator associated pneumonia is defined as that occurring within 5 days of intubation. It is most 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 is 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.

The risk of ventilator associated pneumonia is underestimated when clinical criteria alone is taken into consideration. Hence it is necessary to include even microbiological examination. Moreover the risk of infection increases with increase in the number of days of intubation [66, 67]. Risk factors associated with ventilator associated pneumonia are

- 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

It is better to avoid mechanical ventilation as much as possible, instead Non-invasive intermittent positive pressure ventilation should be used whenever possible. Orotracheal intubation is preferred over nasotracheal intubation because of increased risk of sinusitis in the later.

Also 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.

Pharmacologic management includes stress ulcer prophylaxis and selective decontamination of digestive tract with antibiotics (topical or systemic) or antiseptics. Role of selective decontamination of digestive tract to control pneumonia is controversial as there is no proper study to prove this theory. But there are studies to prove that there is significant role for oral decontamination with antiseptics like chlorhexidine [71].

Shorr AF, Duh MS, Kelly KM, et al also found that blood transfusion is also associated with increased risk of pneumonia. Earley AS, Gracias VH, Haut E, et al suggested that there is a 90% decrease in the incidence of pneumonia following anaemia management protocol which insisted on fewer blood transfusions [72].

Diagnosis of ventilator associated pneumonia is difficult for the following reasons

- Abnormal chest x-ray mimicking non infectious processes
- Difficulty in collecting the respiratory secretions from an intubated patient.

- Absence of clinical signs of infection in immunocompromised individuals.
- Diagnosis is incomplete without the identification of the causative agent.

Clinical Pulmonary Infection Score (CPIS) [146] 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.

Various methods of specimen acquisition for culture are

- Non-invasive
    - endotracheal suction aspiration (EA)
    - blinded plugged telescoping catheter (PTC)

- blinded PSB
- mini-BAL

- Invasive
  - bronchoscopic bronchoalveolar lavage (BAL)
  - protected specimen brush (PSB)

Thresholds for specimens collected by various techniques

- 10^3 CFU/ml - protected specimen brush
- 10^4 CFU/ml - bronchoscopic bronchoalveolar lavage
- 10^5 CFU/ml - endotracheal suction aspiration

In case of cultures collected from patients on antibiotics the threshold should be decreased by one order of magnitude [73].

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 Coagulase-negative staphylococci can also cause respiratory dysfunction.

Central Line–Associated Bloodstream Infection

Large-bore central venous access (e.g., femoral, internal jugular, subclavian vein) is more common with critically ill patients as it is more

reliable in the therapeutic access, but still the catheters are prone to infection if kept in-situ on a chronic basis or if proper care is not given. Strict aseptic care, proper insertion technique, and catheter care on a daily basis are important for prevention of central line associated infections (see earlier) as surgical and trauma patients are at a very high risk. When it is inserted for elective (controlled) purposes, optimal technique includes chlorhexidine skin preparation (not povidone-iodine), maximum barrier precautions (i.e., draping the entire bed into the sterile field; donning a cap, mask, and sterile gown and gloves), and following a formal catheter care protocol. If insertion technique is not sterile or the insertion site is contaminated, the risk of infection increases multifold, and the catheter should be removed immediately and replaced (if still needed) with a different one at a different site using strict asepsis and antisepsis as soon as the patient's condition permits, but certainly should be done within 24 hours to avoid further risk of infection. The risk is highest for femoral vein catheters and lowest for catheters placed via the subclavian route [74].

Peripheral vein catheters, peripherally placed central catheters (PICCs), and tunneled central venous catheters (e.g., Hickman, Broviac) pose less risk of infection than percutaneous central venous catheters. Use of proper protocol associated bundles and checklists are effective measures to

decrease the risk of Central Line–Associated Bloodstream Infections (CLABSI) when put to use and adhered to rigorously [75, 76, 77, 78]. Antibiotic- and antiseptic-coated catheters are controversial, but may to help decrease the risk of infection in patients who have a high risk of susceptibility to infections [79].

All intravascular devices and insertion sites must be assessed periodically on a daily basis to determine any ongoing need and to check for signs of local infection (e.g., inflammation or purulence at the exit site or along the tunnel). Contaminated catheter hubs are common portals of entry for organisms colonizing the endoluminal surface of the catheter. Infusate (e.g., fluid, blood products, IV medications) can become contaminated and cause bacteremia or fungemia, which is likely to result in septic shock. Abrupt onset of signs and symptoms of sepsis or shock in patients with an indwelling vascular catheter should arouse the suspicion of catheter infection. Positive blood cultures for staphylococci or Candida species. strongly indicate the infection of a vascular catheter, which should prompt removal and culture of the catheter.

There are studies suggesting the reliability of semi-quantitative or quantitative catheter tip culture procedures for the positive catheter culture is low when there is a low pre-test probability of catheter-related line sepsis,

and catheters removed from ICU patients should only be cultured if there is a strong clinical suspicion of CLABSI.

For patients undergoing evaluation for fever who do not have SIRS, there is usually no need to remove or change all indwelling catheters immediately; however, exceptions to this would be in a patient with a prosthetic heart valve or fresh arterial graft and is strongly recommended for severe sepsis or septic shock, peripheral embolization, disseminated intravascular coagulation, or acute respiratory distress syndrome.

Suppurative phlebitis of a central vein caused by a centrally placed catheter is uncommon. With suppurative phlebitis, bloodstream infection typically originates from a peripheral vein catheter site with an infected intravascular thrombus, which causes a picture of overwhelming sepsis with high-grade bacteremia or fungemia. This syndrome is encountered most often in burns patients or other ICU patients who develop catheter related infection that goes unnoticed, permitting microbes to proliferate.

In patients with persistent *S. aureus* bacteremia or fungemia, echocardiography is appropriate to assess for endocarditis and further therapy can be decided upon after confirming the presence of an infection [80].

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 [81, 82], even in immunocompromised patients [83], unless there is urinary tract obstruction, history of recent urologic manipulation, injury, or surgery, or neutropenia [84, 85]. There has been relatively little emphasis on the prevention of nosocomial UTIs until recently when compared to the combative measures against ventilator associated pneumonia or CLABSIs [86]. As effective prevention tactics, emphasis is now being placed on avoidance or brief duration of catheterization (e.g., <48 hours for elective surgery patients) [78, 86] and on the use of silver alloy–coated catheters [48, 49] 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 catheter-associated bacteriuria or candiduria (>105 CFU/mL) [87, 88]. 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 [86].

Though clinical evaluations suggest the urinary tract as a possible source of fever, a urine specimen 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. Urine collected for culture should reach the laboratory promptly to prevent multiplication of bacteria within the receptacle, which may lead to the misdiagnosis of infection; in case of any delay the specimen should be refrigerated.

Contrary to community-acquired urinary tract infections, in which pyuria is highly predictive of bacteriuria, 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 [88]. 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 [89]. No evidence has shown that higher counts are more likely to represent symptomatic infection.

Whereas it is appropriate to collect urine specimens in the investigation of fever, routine monitoring or surveillance cultures of urine contribute little to patient management. Rapid dipstick tests, which detect

leukocyte esterase and nitrite, are unreliable in the setting of a catheter-related UTI. The leukocyte esterase test correlates with the degree of pyuria, which may or may not be present in a catheter-related UTI. The nitrite test reflects Enterobacteriaceae, which convert nitrate to nitrite, and is therefore unreliable to screen for Enterococcus, Candida, and Staphylococcus species.

Intra-Abdominal Infection

Intra-abdominal infections (IAIs) usually include a varied group of diseases most commonly encountered in surgical practice. Such infections are dichotomized into uncomplicated (uIAI) and complicated (cIAI) [90] and, more recently, as to whether they arose in the community associated (CA-IAI) 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 [91].

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. An abscess is formed as a result of contained infection, where the foreign bodies facilitate to lower the inoculum size and microbial synergy, and creates a low pH environment which would inhibit phagocyte function and impair permeation of immune cells and antibiotics. Diffuse peritonitis occurs as aresult of uncontrolled spread of infrction, a condition that is associated with higher mortality that calls for urgent laparotomy [92]. Most IAIs may be controlled with low associated morbidity, through clearance of the infected focus, antibiotic treatment, and anatomy restoration in cases where resection is done for definitive infection control. However, 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 [93, 94].

Inappropriate initial antimicrobial therapy of high-risk patients with cIAIs results in increased incidences of clinical failure and death [95, 96], often preceded by multiorgan dysfunction syndrome (MODS). There is a mortality rate of 25-35% in patients with abdominal sepsis [97, 98], but may reach upto 70% [99, 100]. 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) [101].

Mortality from HA-IAIs is more than that from CA-IAIs [102, 103]. Health care–associated non-postoperative IAIs, are those arising in patients hospitalized for reasons other than abdominal pathology, present with a poor prognosis [104]. 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 [94] 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].

MATERIALS AND METHODS

Sample size: 50 cases

Study design: retrospective and prospective study

Study population: 50 cases

Study period: December 2013- August 2014

Inclusion criteria: All patients undergoing splenectomy after trauma.

Exclusion criteria: Patients undergoing splenectomy for reasons other than trauma with or without other procedures.

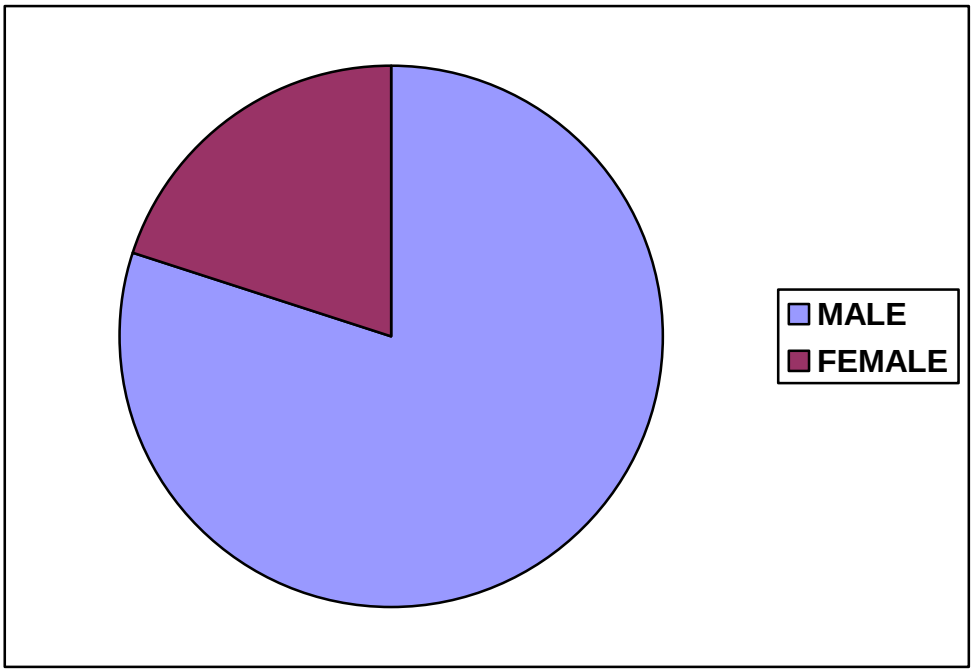
Assessment of parameters:

- Total count
- Platelet count
- Injury Severity Score
- Presence of postoperative infections such as pneumonia, abdominal abscess, septicaemia, urinary tract and surgical site infections.

STATISTICAL ANALYSIS

SEX DISTRIBUTION

SEX	NO OF PATIENTS
MALE	40
FEMALE	10



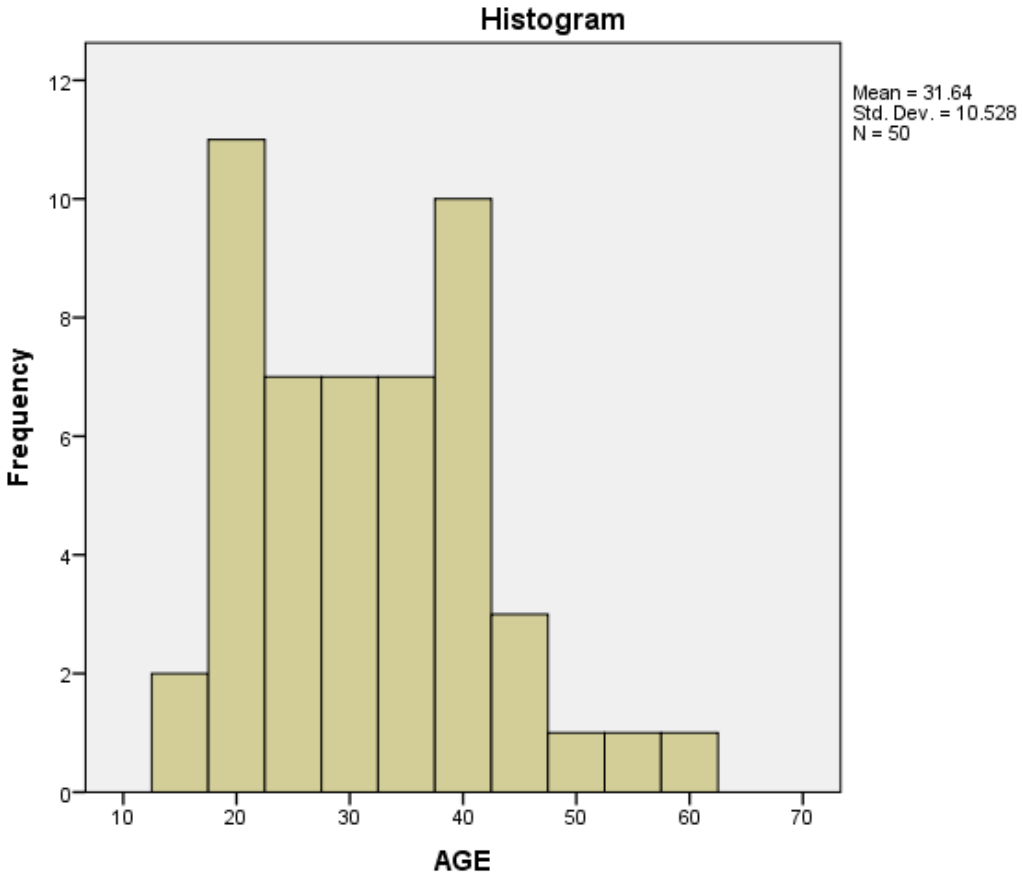
In our study, out of 50 study population 40 were males and 10 were females i.e 80 % were males and 20 % were females.

AGE DISTRIBUTION

AGE

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	15	1	2.0	2.0	2.0
	17	1	2.0	2.0	4.0
	18	4	8.0	8.0	12.0
	19	1	2.0	2.0	14.0
	20	1	2.0	2.0	16.0
	21	3	6.0	6.0	22.0
	22	2	4.0	4.0	26.0
	24	2	4.0	4.0	30.0
	25	4	8.0	8.0	38.0
	26	1	2.0	2.0	40.0
	28	2	4.0	4.0	44.0
	30	1	2.0	2.0	46.0
	31	1	2.0	2.0	48.0
	32	3	6.0	6.0	54.0
	34	1	2.0	2.0	56.0
	35	4	8.0	8.0	64.0
	36	1	2.0	2.0	66.0
	37	1	2.0	2.0	68.0
	38	4	8.0	8.0	76.0
	40	3	6.0	6.0	82.0
	42	3	6.0	6.0	88.0
	43	1	2.0	2.0	90.0
	46	2	4.0	4.0	94.0
	50	1	2.0	2.0	96.0
	55	1	2.0	2.0	98.0
	60	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

	NO OF PATIENTS	MINIMUM	MAXIMUM	MEAN	STD. DEVIATION
AGE	50	15	60	31.64	10.528



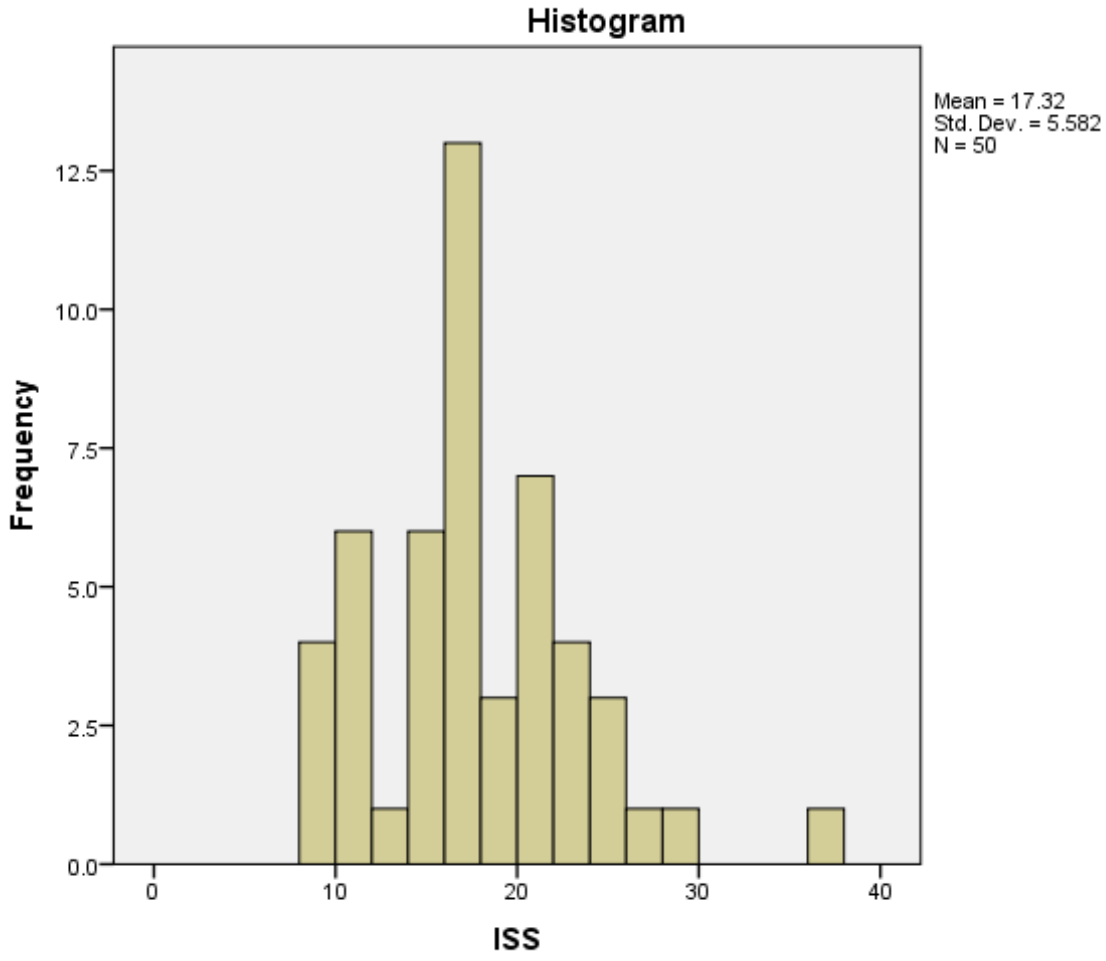
Majority of the study population were between 20-40 years.

DISTRIBUTION OF INJURY SEVERITY SCORE

ISS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	9	4	8.0	8.0	8.0
	11	6	12.0	12.0	20.0
	13	1	2.0	2.0	22.0
	14	6	12.0	12.0	34.0
	16	9	18.0	18.0	52.0
	17	4	8.0	8.0	60.0
	18	1	2.0	2.0	62.0
	19	2	4.0	4.0	66.0
	20	1	2.0	2.0	68.0
	21	6	12.0	12.0	80.0
	22	4	8.0	8.0	88.0
	24	2	4.0	4.0	92.0
	25	1	2.0	2.0	94.0
	27	1	2.0	2.0	96.0
	29	1	2.0	2.0	98.0
	36	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

ISS DISTRIBUTION					
	NO OF PATIENTS	MINIMUM	MAXIMUM	MEAN	STD. DEVIATION
ISS	50	9	36	17.32	5.582



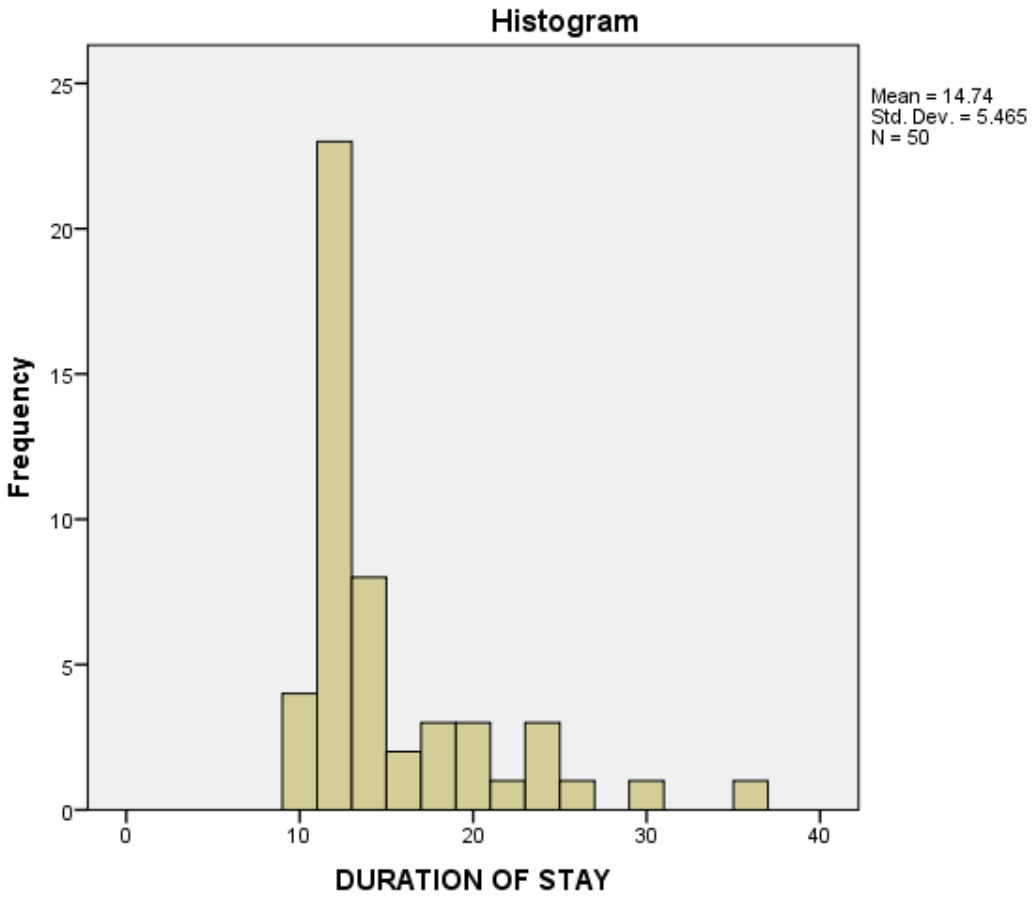
Majority of the study population were having Injury severity score between 16 and 22.

DISTRIBUTION OF DURATION OF STAY

DURATION OF STAY

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	10	4	8.0	8.0	8.0
	11	8	16.0	16.0	24.0
	12	15	30.0	30.0	54.0
	13	7	14.0	14.0	68.0
	14	1	2.0	2.0	70.0
	15	2	4.0	4.0	74.0
	17	1	2.0	2.0	76.0
	18	2	4.0	4.0	80.0
	19	1	2.0	2.0	82.0
	20	2	4.0	4.0	86.0
	21	1	2.0	2.0	88.0
	23	1	2.0	2.0	90.0
	24	2	4.0	4.0	94.0
	25	1	2.0	2.0	96.0
	30	1	2.0	2.0	98.0
	35	1	2.0	2.0	100.0
	Total	50	100.0	100.0	

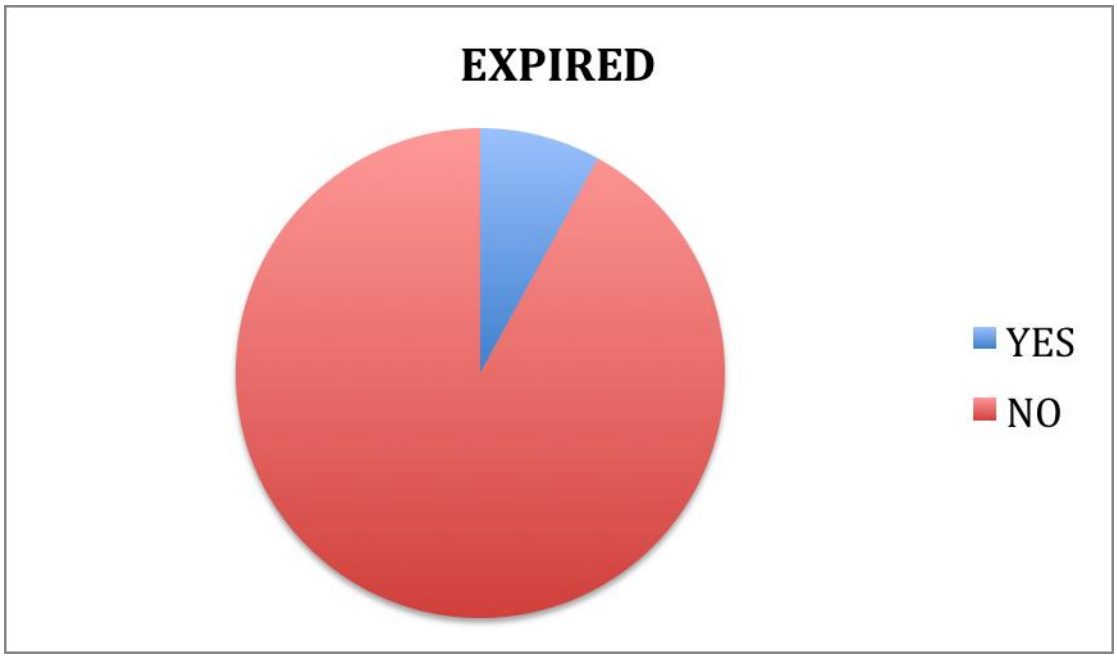
DISTRIBUTION					
	NO OF PATIENTS	MINIMUM	MAXIMUM	MEAN	STD. DEVIATION
DURATION OF STAY	50	10	35	14.74	5.465



Majority of the patients were discharged after 12 days of admission

POST OPERATIVE DEATH

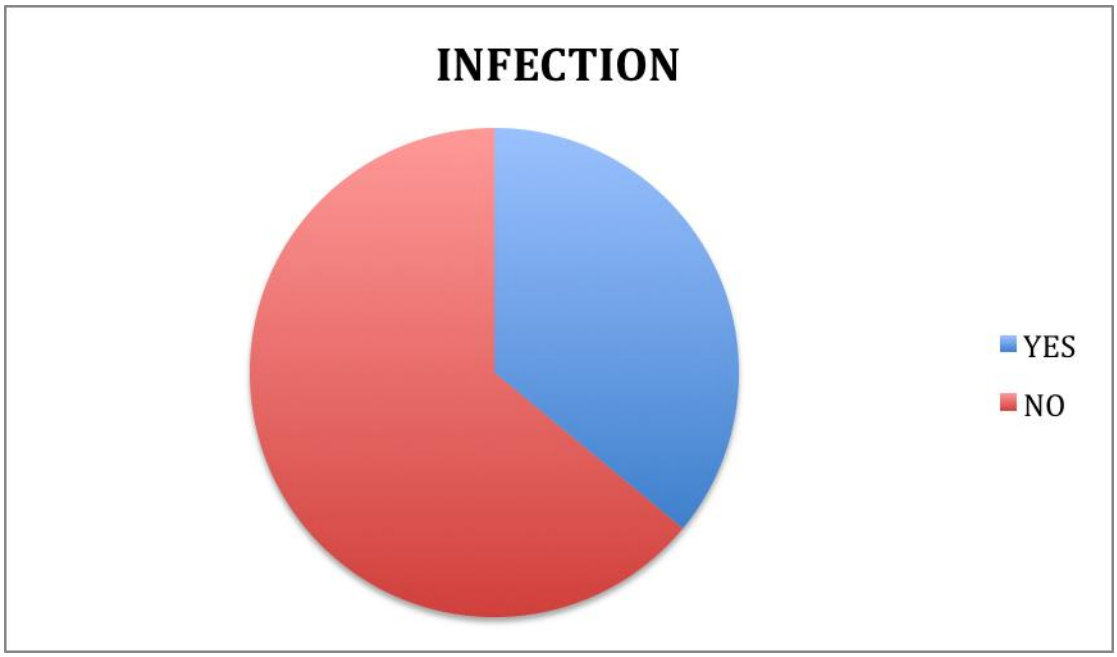
EXPIRED	NO OF PATIENTS
YES	4
NO	46



Out of the 50 patients 4 patients expired of which 2 were from infected group and 2 were from non infected group.

INFECTION

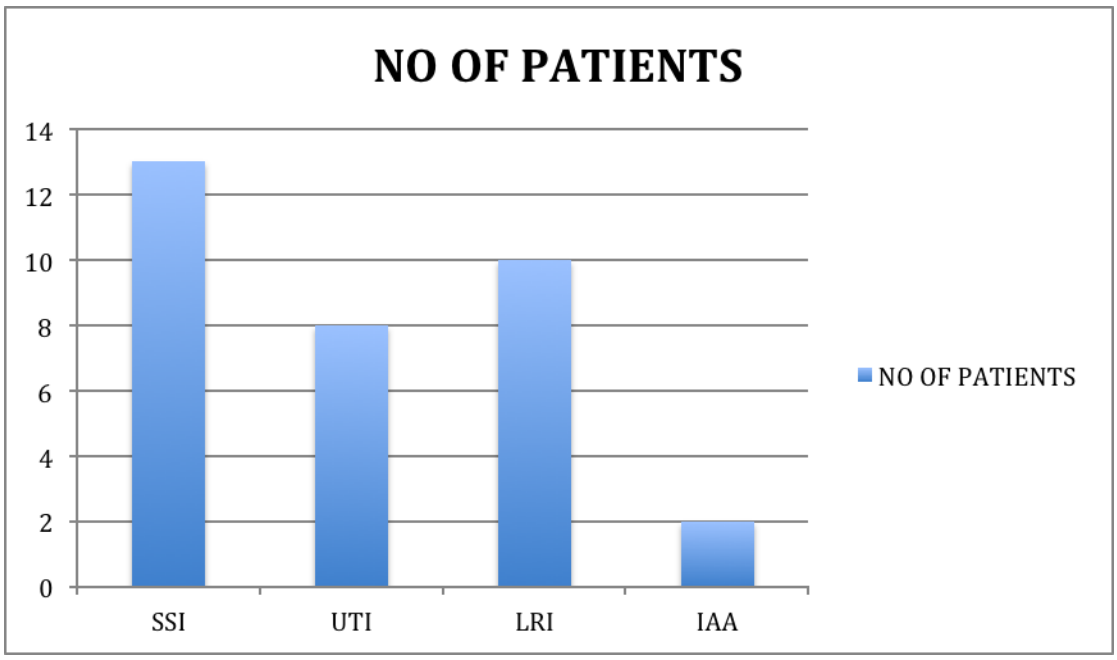
INFECTION	NO OF PATIENTS
YES	18
NO	32



Out of the 50 patients 32 were found to be non infected and 18 were diagnosed to have post operative infections.

NATURE OF INFECTION

NATURE OF INFECTION	NO OF PATIENTS
Surgical site infection	13
Urinary tract infection	8
Lower respiratory tract infection	10
Intra abdominal abscess	2



It is found that surgical site infection is the most common cause of post-operative infection

SIGNIFICANCE OF AGE, SEX, INJURY SEVERITY SCORE, POST OPERATIVE DEATHS AND DURATION OF STAY

AGE

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of age is same among infected and non infected patients	Independent samples Mann-Whitney U test	0.142	Retain the null hypothesis

p value < 0.05 is significant.

From the above test it is inferred that there is no difference in age among infected and non-infected patients.

SEX

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of sex is same among infected and non infected patients	Fischer's exact test	1	Retain the null hypothesis

p value < 0.05 is significant.

From the above test it is inferred that there is no difference in sex among infected and non-infected patients.

INJURY SEVERITY SCORE

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of ISS is same among infected and non infected patients	Independent samples Mann-Whitney U test	0.001	Reject the null hypothesis

p value < 0.05 is significant.

From the above test it is inferred that there is difference in Injury Severity Score among infected and non-infected patients.

POST-OPERATIVE DEATHS

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of deaths is same among infected and non infected patients	Fischer's exact test	0.612	Retain the null hypothesis

p value < 0.05 is significant.

From the above test it is inferred that there is no difference in post-operative deaths among infected and non-infected patients.

DURATION OF STAY

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of duration of stay in the hospital is same among infected and non infected patients	Independent samples Mann-Whitney U test	0.000	Reject the null hypothesis

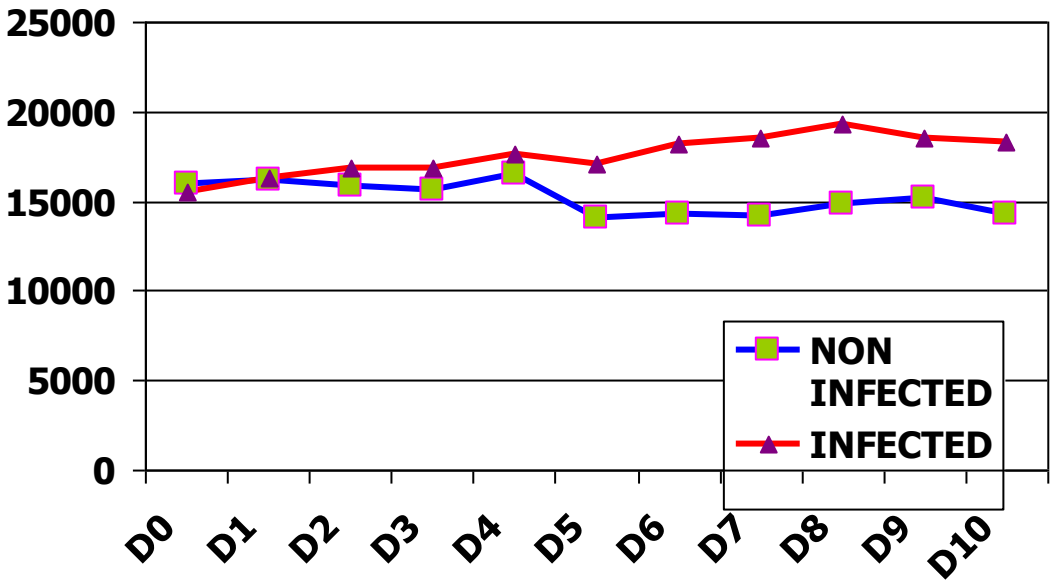
p value < 0.05 is significant.

From the above test it is inferred that there is significant difference in the duration of stay in the hospital among infected and non-infected patients.

COMPARISON OF TOTAL COUNT (TC), PLATELET COUNT (PC) AND PC / TC RATIO

TOTAL COUNT

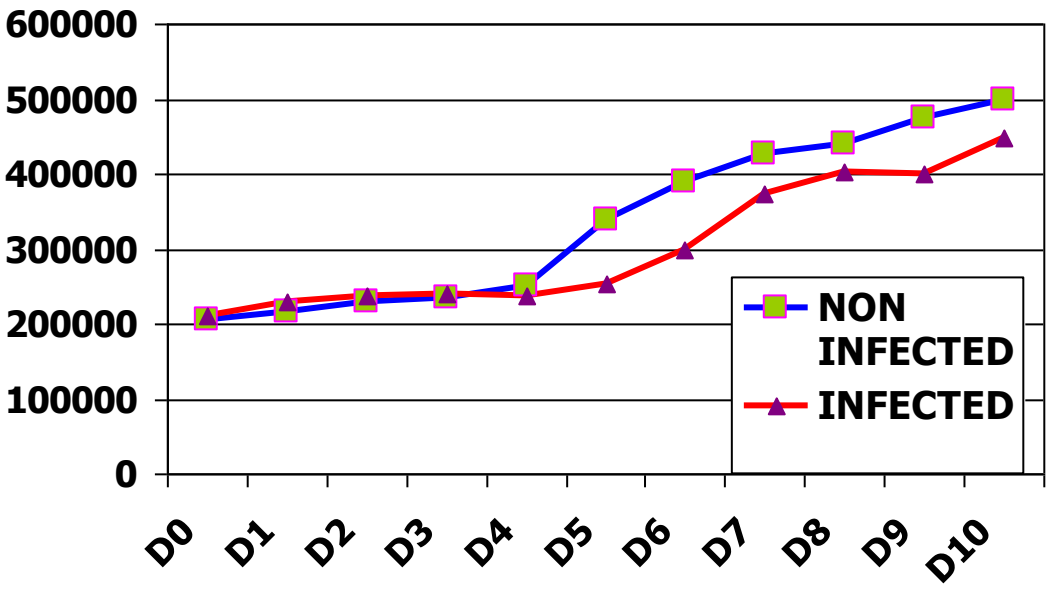
DAY OF SURGERY	MEAN TOTAL COUNT / μL	
	INFECTED	NON-INFECTED
D0	15472	15912
D1	16238	16218
D2	16816	15806
D3	16855	15631
D4	17622	16525
D5	17111	14096
D6	18205	14231
D7	18511	14137
D8	19300	14840
D9	18533	15218
D10	18322	14296



The infected and non-infected patients had similar counts till post-operative 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.

PLATELET COUNT

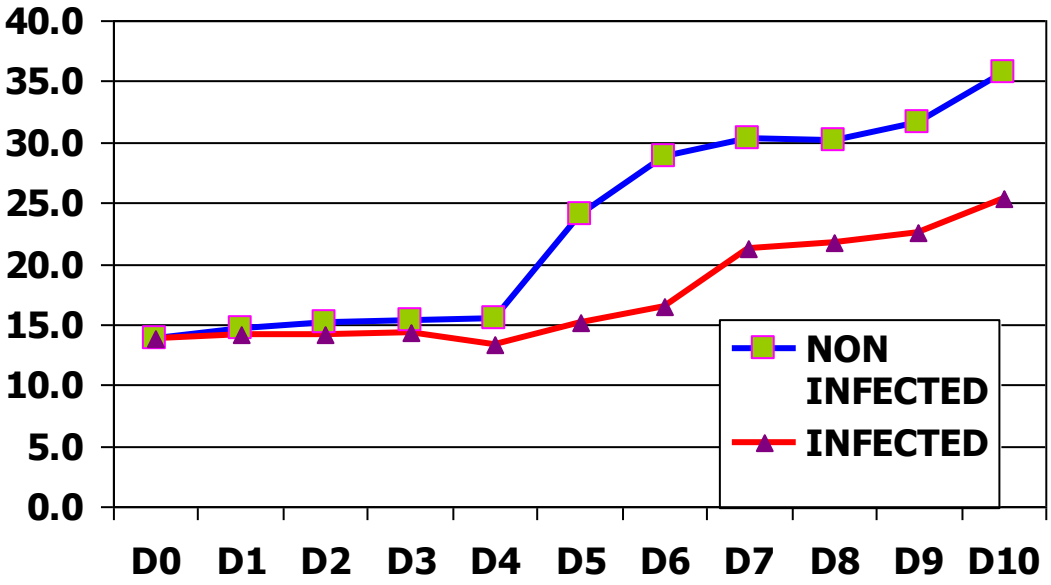
DAY OF SURGERY	MEAN PLATELET COUNT / μL	
	INFECTED	NON-INFECTED
D0	210444	204625
D1	229500	217281
D2	237222	230500
D3	240111	235312
D4	238055	251281
D5	253611	339687
D6	298777	388968
D7	374611	425343
D8	403111	439687
D9	400944	473593
D10	447222	497531



Although the platelet counts rose for both groups throughout the post-operative period, the infected group had a lower platelet count beginning on post-operative day 5.

PC/TC RATIO

DAY OF SURGERY	MEAN PC/TC RATIO	
	INFECTED	NON-INFECTED
D0	13.8	13.8
D1	14.2	14.6
D2	14.2	15.2
D3	14.4	15.3
D4	13.4	15.4
D5	15.1	24
D6	16.4	28.8
D7	21.3	30.3
D8	21.8	30.1
D9	22.6	31.6
D10	25.4	35.7



The PC/TC ratio was lower in the infected group beginning on post-operative day 5. Furthermore the infected group had a lower ratio throughout the post-operative period and did not reach a ratio >20 until post operative day 7, whereas the non-infected group had a ratio >20 by post-operative day 5.

SIGNIFICANCE OF 5TH POST-OPERATIVE DAY VALUES

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of total count on day 5 is same among infected and non infected patients	Student T-Test for 2 independent means	< 0.00001	Reject the null hypothesis

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of platelet count on day 5 is same among infected and non infected patients	Student T-Test for 2 independent means	0.001253	Reject the null hypothesis

NULL HYPOTHESIS	TEST	SIGNIFICANCE	DECISION
The distribution of PC/TC ratio on day 5 is same among infected and non infected patients	Student T-Test for 2 independent means	< 0.00001	Reject the null hypothesis

Post-operative day 5 is the earliest day with significant difference in total count, platelet count and TC/PC ratio among the infected and non-infected patients.

RISK FACTORS FOR INFECTION

Presence of more than one of the following risk factors i.e. TC >15000 on post-operative day 5, PC/TC ratio >20 on post-operative day 5, and ISS >16 is associated with 83% chance of infection.

DISCUSSION

The current study validates the three risk factors for post-splenectomy infections in trauma patients. The results of this study was found to be similar to the prospective study published by Weng J, Brown CV, Rhee P, Salim A, Chan L, Demetriades D, Velmahos GC in the journal of trauma in May, 2005 except for the significance of injury severity score which was significant in the present study unlike the previous study and the incidence of infection which was 83% when >1 risk factor is present as against 79% in the earlier study.

The following are the results of the study

- Injury severity score 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.
- Beyond the first week this ratio becomes insignificant.
- Presence of more than 1 risk factor is associated with 83% chance of infection.

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
  - PC/TC ratio
  - ISS > 16

- Presence of more than one risk factor should prompt clinicians to suspect infection.

BIBLIOGRAPHY

1. Copes WS, Sacco WJ, Champion HR, Bain LW, "Progress in Characterising Anatomic Injury", In Proceedings of the 33rd Annual Meeting of the Association for the Advancement of Automotive Medicine, Baltimore, MA, USA 205-218

2. Baker SP et al, "The Injury Severity Score: a method for describing patients with multiple injuries and evaluating emergency care", J Trauma 14:187-196;1974

3. Peitzman AB, Heil B, Rivera L, et al: Blunt splenic injury in adults: Multi-institutional study of the Eastern Association for the Surgery of Trauma. J Trauma 49:177–189, 2000.

4. Nicholas JM, Parker Rix E, Easley KA, et al: Changing patterns in the management of penetrating abdominal trauma: The more things change, the more they stay the same. J Trauma 55:1095– 1110, 2003.

5. Haan JM, Bochicchio GV, Kramer N, et al: Nonoperative management of blunt splenic injury: A 5-year experience. J Trauma58:492–498, 2005.

6. Cocanour CS, Moore FA, Ware DN, et al: Age should not be a consideration for nonoperative management of blunt splenic injury. J Trauma 48:606–612, 2000.

7. Harbrecht BG, Peitzman AB, Rivera L, et al: Contribution of age and gender to outcome of blunt splenic injury in adults: multicenter study of the Eastern Association for the Surgery of Trauma. J Trauma 51:887–895, 2001.

8. Nieuwenhuis P, Ford WL. Comparative migration of B- and T-lymphocytes in the rat spleen and lymph nodes. Cell Immunol 1976; 23: 254-267

9. Williams GM, Nossal GJV. Ontogeny of the immune response. 1. the development of the follicular antigen-trapping mechanism. J Exp Med; 124: 47-56

10. Schick P, Trepel F, Eder M, et al. Autotransfusion of cytidine labeled blood lymphocytes in patients with hodgkin's disease and non-hodgkin patients. Exchangeable lymphocyte pools. Acta Haematol 1975; 53: 206-218

11. Rannie GH, Donald KJ. Estimation of the migration of thoracic duct lymphocytes to non-lymphoid tissue. Cell Tiss Kinet 1977; 10: 360-371

12. Smith ME, Ford WL. The recirculating lymphocyte pool of the rat: a systemic description of the migratory behaviour of recirculating lymphocytes. Immunology 1983; 49: 83-94.

13. Desborough JP: The stress response to trauma and surgery. Br J Anaesth 85:109–117, 2000.

14. Napolitano LM, Faist E, Wichmann MW, et al: Immune dysfunction in trauma. Surg Clin North Am 79:1385–1416, 1999.

15. Gardner EM, Murasko DM: Age-related changes in Type 1 and Type 2 cytokine production in humans. Biogerontology 3:271–290, 2002.

16. Wichmann MW, Zellweger R, DeMaso CM, et al: Enhanced immune responses in females, as opposed to decreased responses in males following haemorrhagic shock and resuscitation. Cytokine 8:853–863, 1996.

17. Diodato MD, Knoferl MW, Schwacha MG, et al: Gender differences in the inflammatory response and survival following haemorrhage and subsequent sepsis. Cytokine 14:162–169, 2001

18. Gunderson KL, Steemers FJ, Lee G, et al: A genome-wide scalable SNP genotyping assay using microarray technology. Nat Genet 37:549–554, 2005.

19. Machiedo GW, Powell RJ, Rush BF, Jr, et al: The incidence of decreased red blood cell deformability in sepsis and the association with oxygen free radical damage and multiple-system organ failure. Arch Surg 124:1386–1389, 1989.

20. Danks RR: Triangle of death. How hypothermia acidosis and coagulopathy can adversely impact trauma patients. JEMS 27:61–66, 68–70, 2002.

21. Dickinson A, Qadan M, Polk HC, Jr: Optimizing surgical care: A contemporary assessment of temperature, oxygen, and glucose. Am Surg 76:571–577, 2010.

22. Ives CL, Harrison DK, Stansby GS: Tissue oxygen saturation, measured by near-infrared spectroscopy, and its relationship to surgical-site infections. Br J Surg 94:87–91, 2007.

23. Delaney AP, Dan A, McCaffrey J, et al: The role of albumin as a resuscitation fluid for patients with sepsis: A systematic review and meta-analysis. Crit Care Med 39:386–391, 2011.

24. Bochicchio GV, Napolitano LM, Joshi M, et al: Persistent systemic inflammatory response syndrome is predictive of nosocomial infection in trauma. J Trauma 53:245–250, 2002.

25. Nathens AB, Nester TA, Rubenfeld GD, et al: The effects of leukoreduced blood transfusion on infection risk following injury: a randomized controlled trial. Shock 26:342–347, 2006.

26. Claridge JA, Sawyer RG, Schulman AM, et al: Blood transfusions correlate with infections in trauma patients in a dosedependent manner. Am Surg 68:566–572, 2002.

27. Taylor RW, O'Brien J, Trottier SJ, et al: Red blood cell transfusions and nosocomial infections in critically ill patients. Crit Care Med 34:2302–2308; quiz 2309, 2006.

28. Scharte M, Fink MP: Red blood cell physiology in critical illness. Crit Care Med 31:S651–S657, 2003.

29. Fernandes CJ, Jr, Akamine N, De Marco FV, et al: Red blood cell transfusion does not increase oxygen consumption in critically ill septic patients. Crit Care 5:362–367, 2001.

30. Moore FA, Moore EE, Sauaia A: Blood transfusion. An independent risk factors for postinjury multiple organ failure. Arch Surg 132:620–624, 1997.
31. Offner PJ, Moore EE, Biffl WL, et al: Increased rate of infection associated with transfusion of old blood after severe injury. Arch Surg 137:711–716, 2002.

32. van den Berghe G, Wouters P, Weekers F, et al: Intensive insulin therapy in the critically ill patients. N Engl J Med 345:1359– 1367, 2001.

33. Krinsley JS: Understanding glycemic control in the critically ill: 2011 update. Hosp Pract (Minneapolis) 39:47–55, 2011.

34. Heyland DK, MacDonald S, Keefe L, et al: Total parenteral nutrition in the critically ill patient: a meta-analysis. JAMA 280:2013–2019, 1998.

35. Marik PE, Zaloga GP: Early enteral nutrition in acutely ill patients: A systematic review. Crit Care Med 29:2264–2270, 2001.

36. Manian FA, Meyer PL, Setzer J, et al: Surgical site infections associated with methicillin-resistant Staphylococcus aureus: Do postoperative factors play a role? Clin Infect Dis 36:863–868, 2003.

37. Prospero E, Barbadoro P, Esposto E, et al: Extended-spectrum beta-lactamases Klebsiella pneumoniae: Multimodal infection control program in intensive care units. J Prev Med Hyg 51:110– 115, 2010.

38. Dancer SJ: The role of environmental cleaning in the control of hospital-acquired infection. J Hosp Infect 73:378–385, 2009.

39. Evans HL, Shaffer MM, Hughes MG, et al: Contact isolation in surgical patients: A barrier to care? Surgery 134:180–188, 2003.

40. Cook DJ, Walter SD, Cook RJ, et al: Incidence of and risk factors for ventilator-associated pneumonia in critically ill patients. Ann Intern Med 129:433–440, 1998.

41. Milstone AM, Passaretti CL, Perl TM: Chlorhexidine: Expanding the armamentarium for infection control and prevention. Clin Infect Dis 46:274–281, 2008.

42. Darouiche RO, Wall MJ, Jr, Itani KM, et al: Chlorhexidinealcohol versus povidone-iodine for surgical-site antisepsis. N Engl J Med 362:18–26, 2010.

43. Evans HL, Dellit TH, Chan J, et al: Effect of chlorhexidine whole-body bathing on hospital-acquired infections among trauma patients. Arch Surg 145:240–246, 2010.

44. Dixon JM, Carver RL: Daily chlorohexidine gluconate bathing with impregnated cloths results in statistically significant reduction in central line–associated bloodstream infections. Am J Infect Control 38:817–821, 2010.

45. Weber DJ, Rutala WA: Central line-associated bloodstream infections: Prevention and management. Infect Dis Clin North Am 25:77–102, 2011.

46. Bonello RS, Fletcher CE, Becker WK, et al: An intensive care unit quality improvement collaborative in nine Department of Veterans Affairs hospitals: Reducing ventilator-associated pneumonia and catheter-related bloodstream infection rates. Jt Comm J Qual Patient Saf 34:639–645, 2008.

47. Jain M, Miller L, Belt D, et al: Decline in ICU adverse events, nosocomial infections and cost through a quality improvement initiative focusing on teamwork and culture change. Qual Saf Health Care 15:235–239, 2006.

48. Beattie M, Taylor J: Silver alloy versusuncoated urinary catheters: A systematic review of the literature. J Clin Nurs 2011.

49. Johnson JR, Kuskowski MA, Wilt TJ: Systematic review: Antimicrobial urinary catheters to prevent catheter-associated urinary tract infection in hospitalized patients. Ann Intern Med 144:116–126, 2006.

50. Barie PS: Surgical site infections: Epidemiology and prevention. Surg Infect (Larchmt) 3(Suppl 1):S9–21, 2002.

51. National Nosocomial Infections Surveillance (NNIS) System Report, Data Summary from January 1992-June 2001, issued August 2001. Am J Infect Control 29:404–421, 2001.

52. National Nosocomial Infections Surveillance (NNIS) System Report, data summary from January 1992 through June 2004, issued October 2004. Am J Infect Control 32:470–485, 2004.

53. Edwards JR, Peterson KD, Mu Y, et al: National Healthcare Safety Network (NHSN) report: Data summary for 2006 through 2008, issued December 2009. Am J Infect Control 37:783–805, 2009.

54. Hedrick TL, Heckman JA, Smith RL, et al: Efficacy of protocol implementation on incidence of wound infection in colorectal operations. J Am Coll Surg 205:432–438, 2007.

55. Brar MS, Brar SS, Dixon E: Perioperative supplemental oxygen in colorectal patients: A meta-analysis. J Surg Res 166:227–235, 2011.

56. Miller RS, Morris JA, Jr, Diaz JJ, Jr, et al: Complications after 344 damage-control open celiotomies. J Trauma 59:1365–1371, 2005.

57. Vogel TR, Diaz JJ, Miller RS, et al: The open abdomen in trauma: Do infectious complications affect primary abdominal closure? Surg Infect (Larchmt) 7:433–441, 2006.

58. Al-Inany H, Youssef G, Abd ElMaguid A, et al: Value of subcutaneous drainage system in obese females undergoing cesarean section using Pfannenstiel incision. Gynecol Obstet Invest 53:75–78, 2002.

59. Magann EF, Chauhan SP, Rodts-Palenik S, et al: Subcutaneous stitch closure versus subcutaneous drain to prevent wound disruption after cesarean delivery: A randomized clinical trial. Am J Obstet Gynecol 186:1119–1123, 2002.

60. Andersen B, Bendtsen A, Holbraad L, et al: Wound infections after appendicectomy. I. A controlled trial on the prophylactic efficacy of topical ampicillin in non-perforated appendicitis. II. A controlled trial on the prophylactic efficacy of delayed primary suture and topical ampicillin in perforated appendicitis. Acta Chir Scand 138:531–536, 1972.

61. Yoshii S, Hosaka S, Suzuki S, et al: Prevention of surgical site infection by antibiotic spraying in the operative field during cardiac surgery. Jpn J Thorac Cardiovasc Surg 49:279–281, 2001.

62 O'Connor LT, Jr, Goldstein M: Topical perioperative antibiotic prophylaxis for minor clean inguinal surgery. J Am Coll Surg 194:407–410, 2002.

63. Freischlag JA, Ajalat G, Busuttil RW: Treatment of necrotizing soft tissue infections. The need for a new approach. Am J Surg 149:751–755, 1985.

64. Heller L, Levin SL, Butler CE: Management of abdominal wound dehiscence using vacuum-assisted closure in patients with compromised healing. Am J Surg 191:165–172, 2006.

65. Schaffzin DM, Douglas JM, Stahl TJ, et al: Vacuum-assisted closure of complex perineal wounds. Dis Colon Rectum 47:1745– 748, 2004.

66. Fagon JY, Chastre J, Wolff M, et al: Invasive and noninvasive strategies for management of suspected ventilator-associated pneumonia. A randomized trial. Ann Intern Med 132:621–630, 2000.

67. Safdar N, Dezfulian C, Collard HR, et al: Clinical and economic consequences of ventilator-associated pneumonia: a systematic review. Crit Care Med 33:2184–2193, 2005.

68. Drakulovic MB, Torres A, Bauer TT, et al: Supine body position as a risk factor for nosocomial pneumonia in mechanically ventilated patients: A randomised trial. Lancet 354:1851–1858, 1999.

69. Heyland DK, Dhaliwal R, Drover JW, et al: Canadian clinical practice guidelines for nutrition support in mechanically ventilated, critically ill adult patients. JPEN J Parenter Enteral Nutr 27:355– 73, 2003.

70. Berne JD, Norwood SH, McAuley CE, et al: Erythromycin reduces delayed gastric emptying in critically ill trauma patients: A randomized, controlled trial. J Trauma 53:422–425, 2002.

71. van Nieuwenhoven CA, Buskens E, van Tiel FH, et al: Relationship between methodological trial quality and the effects of selective digestive decontamination on pneumonia and mortality in critically ill patients. JAMA 286:335–340, 2001.

72. Earley AS, Gracias VH, Haut E, et al: Anemia management program reduces transfusion volumes, incidence of ventilatorassociated pneumonia, and cost in trauma patients. J Trauma 61:1–5, 2006.

73. Torres A, El-Ebiary M: Bronchoscopic BAL in the diagnosis of ventilator-associated pneumonia. Chest 117:198S-202S, 2000.

74. McGee DC, Gould MK: Preventing complications of central venous catheterization. N Engl J Med 348:1123–1133, 2003.

75. Pronovost P: Interventions to decrease catheter-related bloodstream infections in the ICU: The Keystone Intensive Care Unit Project. Am J Infect Control 36:S171 e171–e175, 2008.

76. Pronovost PJ, Goeschel CA, Colantuoni E, et al: Sustaining reductions in catheter related bloodstream infections in Michigan intensive care units: Observational study. BMJ 340:c309, 2010.

77. Furuya EY, Dick A, Perencevich EN, et al: Central line bundle implementation in US intensive care units and impact on bloodstream infections. PLoS One 6:e15452, 2011.

78. Miller RS, Norris PR, Jenkins JM, et al: Systems initiatives reduce healthcare-associated infections: A study of 22,928 device days in a single trauma unit. J Trauma 68:23–31, 2010.

79. O'Grady NP, Alexander M, Burns LA, et al: Guidelines for the prevention of intravascular catheter-related infections. Clin Infect Dis 52:e162–e193, 2011.

80. Mermel LA, Farr BM, Sherertz RJ, et al: Guidelines for the management of intravascular catheter-related infections. Clin Infect Dis 32:1249–1272, 2001.

81. Tambyah PA, Maki DG: Catheter-associated urinary tract infection is rarely symptomatic: A prospective study of 1,497 catheterized patients. Arch Intern Med 160:678–682, 2000.

82. Golob JF, Jr, Claridge JA, Sando MJ, et al: Fever and leukocytosis in critically ill trauma patients: It's not the urine. Surg Infect (Larchmt) 9:49–56, 2008.

83. Safdar N, Slattery WR, Knasinski V, et al: Predictors and outcomes of candiduria in renal transplant recipients. Clin Infect Dis 40:1413–1421, 2005.

84. Bryan CS, Reynolds KL: Hospital-acquired bacteremic urinary tract infection: Epidemiology and outcome. J Urol 132:494– 498, 1984.

85. Quintiliani R, Klimek J, Cunha BA, et al: Bacteraemia after manipulation of the urinary tract. The importance of pre-existing urinary tract disease and compromised host defences. Postgrad Med J 54:668–671, 1978.

86. Ksycki MF, Namias N: Nosocomial urinary tract infection. Surg Clin North Am 89:475–481, ix–x, 2009.

87. Laupland KB, Bagshaw SM, Gregson DB, et al: Intensive care unit-acquired urinary tract infections in a regional critical care system. Crit Care 9:R60–65, 2005.

88. Schwartz DS, Barone JE: Correlation of urinalysis and dipstick results with catheter-associated urinary tract infections in surgical ICU patients. Intensive Care Med 32:1797–1801, 2006.

89. Schiotz HA: The value of leucocyte stix results in predicting bacteriuria and urinary tract infection after gynaecological surgery. J Obstet Gynaecol 19:396–398, 1999.

90. Solomkin JS, Hemsell DL, Sweet R, et al: Evaluation of new anti-infective drugs for the treatment of intra-abdominal infections. Infectious Diseases Society of America and the Food and Drug Administration. Clin Infect Dis 15(Suppl 1):S33–S42, 1992.

91. Merlino JI, Yowler CJ, Malangoni MA: Nosocomial infections adversely affect the outcomes of patients with serious intraabdominal infections. Surg Infect (Larchmt) 5:21–27, 2004.

92. Nathens AB, Rotstein OD, Marshall JC: Tertiary peritonitis: Clinical features of a complex nosocomial infection. World J Surg 22:158–163, 1998.

93. Pacelli F, Doglietto GB, Alfieri S, et al: Prognosis in intraabdominal infections. Multivariate analysis on 604 patients. Arch Surg 131:641–645, 1996.

94. Roehrborn A, Thomas L, Potreck O, et al: The microbiology of postoperative peritonitis. Clin Infect Dis 33:1513–1519, 2001.

95. Sturkenboom MC, Goettsch WG, Picelli G, et al: Inappropriate initial treatment of secondary intra-abdominal infections leads to increased risk of clinical failure and costs. Br J Clin Pharmacol 60:438–443, 2005.

96. Montravers P, Gauzit R, Muller C, et al: Emergence of antibioticresistant bacteria in cases of peritonitis after intra-abdominal surgery affects the efficacy of empirical antimicrobial therapy. Clin Infect Dis 23:486–494, 1996.

97. Barie PS, Hydo LJ, Shou J, et al: Efficacy and safety of drotrecogin alfa (activated) for the therapy of surgical patients with severe sepsis. Surg Infect (Larchmt) 7(Suppl 2):S77–S80, 2006.

98. Barie PS, Vogel SB, Dellinger EP, et al: A randomized, doubleblind clinical trial comparing cefepime plus metronidazole with imipenem-cilastatin in the treatment of complicated intraabdominal infections. Cefepime Intra-abdominal Infection Study Group. Arch Surg 132:1294–1302, 1997.

99. Farthmann EH, Schoffel U: Principles and limitations of operative management of intraabdominal infections. World J Surg 14:210–217, 1990.

100. Garcia-Sabrido JL, Tallado JM, Christou NV, et al: Treatment of severe intra-abdominal sepsis and/or necrotic foci by an ‘openabdomen’

approach. Zipper and zipper-mesh techniques. Arch Surg 123:152–156, 1988.

101. Marshall JC, Maier RV, Jimenez M, et al: Source control in the management of severe sepsis and septic shock: an evidence- based review. Crit Care Med 32:S513–S526, 2004.

102. Wittmann DH, Aprahamian C, Bergstein JM: Etappenlavage: Advanced diffuse peritonitis managed by planned multiple laparotomies utilizing zippers, slide fastener, and Velcro analogue for temporary abdominal closure. World J Surg 14:218–226, 1990.

103. Ohmann C, Wittmann DH, Wacha H: Prospective evaluation of prognostic scoring systems in peritonitis. Peritonitis Study Group. Eur J Surg 159:267–274, 1993.

104. Gajic O, Urrutia LE, Sewani H, et al: Acute abdomen in the medical intensive care unit. Crit Care Med 30:1187–1190, 2002.

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:

serial no	name	age	sex	ip no	ISS	TC0	P0	PT0	TC1	P1	PT1	TC2	P2	PT2	TC3	P3	PT3
1	JAYARAJ	42	M	1597	9	10800	196000	18.1	9800	186000	19.0	8800	171000	19.4	10700	196000	18.3
2	SURESH	25	M	2359	25	13000	195000	15.0	18000	265000	14.7	16800	165000	9.8	17100	196000	11.5
3	JAYALAKSHMI	50	F	2932	21	15400	198000	12.9	14000	120000	8.6	14200	195000	13.7	18000	190000	10.6
4	RIYAZ	25	M	3698	14	17800	174000	9.8	19400	104000	5.4	17800	346000	19.4	17500	354000	20.2
5	ABAI SHARMA	32	M	4682	22	18000	70000	3.9	6100	60000	9.8	13200	140000	10.6	15200	275000	18.1
6	BANUMATHY	46	F	8545	21	17000	215000	12.6	24000	224000	9.3	16900	230000	13.6	17000	265000	15.6
7	SATHISH	18	M	9593	21	7600	144000	18.9	11000	193000	17.5	11600	275000	23.7	11800	129000	10.9
8	ANAND	20	M	10654	16	17500	241000	13.8	16500	166000	10.1	17400	195000	11.2	16400	265000	16.2
9	KARAN	15	M	13307	16	13200	168000	12.7	9300	268000	28.8	11200	238000	21.3	17400	355000	20.4
10	DHANDAPANI	32	M	13601	17	13000	200000	15.4	19000	265000	13.9	15800	265000	16.8	16000	198000	12.4
11	SADASIVAM	24	M	15117	16	14100	210000	14.9	16600	122000	7.3	17800	218000	12.2	17000	260000	15.3
12	NAGARAJ	18	M	16025	11	19000	187000	9.8	13600	199000	14.6	17500	225000	12.9	16400	245000	14.9
13	BUJIAMMAL	38	F	16976	13	10000	251000	25.1	7400	200000	27.0	8100	156000	19.3	10800	220000	20.4
14	PARAMESHWARAN	46	M	17006	21	12500	345000	27.6	12600	356000	28.3	14500	325000	22.4	13600	256000	18.8
15	PONNIYAMMAL	60	F	20659	17	15400	248000	16.1	16500	113000	6.8	14500	221000	15.2	16500	298000	18.1
16	MOHAN	37	M	25382	9	15900	254000	16.0	20300	310000	15.3	17000	180000	10.6	16900	198000	11.7
17	SAKTHIVEL	42	M	26099	14	14600	326000	22.3	20000	320000	16.0	23500	373000	15.9	18000	265000	14.7
18	RAMESH	43	M	26571	17	16400	321000	19.6	17200	236000	13.7	17800	365000	20.5	18900	190000	10.1
19	RAJESH KUMAR	32	M	26982	16	19000	110000	5.8	16500	298000	18.1	17400	246000	14.1	16500	297000	18.0
20	ARUN KUMAR	18	M	26984	9	17400	320000	18.4	17000	364000	21.4	16500	212000	12.8	17400	224000	12.9
21	PAUL RAJ	19	M	30145	11	18600	142000	7.6	18000	116000	6.4	17500	222000	12.7	16300	223000	13.7
22	ANJI	25	M	30614	14	14900	267000	17.9	15900	345000	21.7	11300	225000	19.9	13600	248000	18.2
23	YOUNIS	28	M	30988	16	24000	162000	6.8	16000	321000	20.1	15600	254000	16.3	16000	300000	18.8
24	KARTHICK	21	M	32656	16	14600	210000	14.4	14300	387000	27.1	15200	175000	11.5	15400	215000	14.0
25	MURUGAN	38	M	33829	14	12500	221000	17.7	17000	132000	7.8	18000	271000	15.1	18600	298000	16.0
26	FAROOQ	38	M	35456	22	15000	241000	16.1	19300	465000	24.1	20100	245000	12.2	16800	275000	16.4
27	MUTHULAKSHMI	31	F	35465	19	17500	198000	11.3	16500	389000	23.6	16900	385000	22.8	18500	359000	19.4
28	MANIKANDAN	21	M	36777	17	14000	113000	8.1	10200	225000	22.1	12000	210000	17.5	12200	225000	18.4
29	PARTHIBAN	21	M	44564	16	14500	365000	25.2	14900	345000	23.2	13600	254000	18.7	13800	172000	12.5
30	SUBRAMANI	40	M	45024	16	9700	165000	17.0	17000	132000	7.8	20500	165000	8.0	16500	210000	12.7
31	SHANKAR	42	M	45343	18	16300	136000	8.3	17100	211000	12.3	15000	197000	13.1	15500	211000	13.6
32	ESWARAN	28	M	45389	36	12000	289000	24.1	21000	241000	11.5	17100	259000	15.1	19000	264000	13.9
33	SAKTHI	17	M	45678	22	17000	452000	26.6	20300	312000	15.4	19800	298000	15.1	18600	245000	13.2
34	DEEPAN	25	M	45751	24	16500	178000	10.8	11900	133000	11.2	18000	143000	7.9	14800	236000	15.9
35	DAWOOD	40	M	46980	11	16500	210000	12.7	17500	195000	11.1	16800	345000	20.5	15600	265000	17.0
36	MAHESHWARI	35	F	47589	14	18500	132000	7.1	19800	174000	8.8	16500	378000	22.9	15900	349000	21.9
37	DINESH	24	M	53656	11	17900	180000	10.1	16400	287000	17.5	15600	221000	14.2	15400	221000	14.4
38	SRIDHARAN	34	M	54276	29	15200	165000	10.9	11900	133000	11.2	14500	234000	16.1	14800	236000	15.9
39	MANIKANDAN	18	M	56549	11	13000	140000	10.8	11400	134000	11.8	14000	183000	13.1	13900	206000	14.8
40	APPUKUTTI	35	F	63292	22	12500	165000	13.2	13000	122000	9.4	11400	138000	12.1	10800	112000	10.4
41	UMA DEVI	38	F	64565	21	20500	321000	15.7	19800	298000	15.1	18500	254000	13.7	18400	242000	13.2
42	SENTHIL	30	M	67327	24	15000	165000	11.0	16400	160000	9.8	19100	210000	11.0	17500	145000	8.3
43	KANNIYAMMAL	55	F	67453	27	15400	79000	5.1	15600	87000	5.6	14400	87000	6.0	15800	186000	11.8
44	NARENDRAN	22	M	112755	16	27000	152000	5.6	38200	110000	2.9	30200	165000	5.5	27300	127000	4.7
45	MOHAN	22	M	114760	20	11000	165000	15.0	11500	166000	14.4	10800	125000	11.6	12500	200000	16.0
46	RAGHUVARAN	40	M	116900	19	19000	265000	13.9	17400	174000	10.0	16500	265000	16.1	15300	321000	21.0
47	JAYAKUMAR	36	M	116998	9	21000	130000	6.2	20600	130000	6.3	16900	161000	9.5	15200	150000	9.9
48	RAJAN	26	M	117467	11	15700	100000	6.4	14500	225000	15.5	14900	265000	17.8	14300	235000	16.4
49	REVATHY	35	F	117504	21	18000	265000	14.7	19500	310000	15.9	18500	321000	17.4	17200	300000	17.4
50	ELUMALAI	35	M	122544	14	16800	190000	11.3	13600	256000	18.8	21000	250000	11.9	19000	200000	10.5

serial no	name	ip no	TC4	P4	PT4	TC5	P5	PT5	TC6	P6	PT6	TC7	P7	PT7	TC8	P8	PT8
1	JAYARAJ	1597	14200	165000	11.6	14400	351000	24.4	10400	562000	54.0	16500	600000	36.4	15400	450000	29.2
2	SURESH	2359	16800	183000	10.9	16800	146000	8.7	20600	406000	19.7	19000	475000	25.0	16000	452000	28.3
3	JAYALAKSHMI	2932	16500	198000	12.0	15600	193000	12.4	15800	213000	13.5	35200	235000	6.7	28800	260000	9.0
4	RIYAZ	3698	18500	321000	17.4	15100	456000	30.2	14900	398000	26.7	14800	365000	24.7	14200	485000	34.2
5	ABAI SHARMA	4682	16300	270000	16.6	16900	309000	18.3	15400	244000	15.8	15400	635000	41.2	28400	632000	22.3
6	BANUMATHY	8545	16300	198000	12.1	16500	257000	15.6	15800	298000	18.9	19700	356000	18.1	22500	456000	20.3
7	SATHISH	9593	14300	150000	10.5	14700	275000	18.7	14500	324000	22.3	14000	256000	18.3	13200	456000	34.5
8	ANAND	10654	15000	398000	26.5	14800	412000	27.8	14200	321000	22.6	13600	432000	31.8	12600	654000	51.9
9	KARAN	13307	16600	240000	14.5	16900	272000	16.1	20600	365000	17.7	17200	452000	26.3	18900	398000	21.1
10	DHANDAPANI	13601	17000	176000	10.4	16500	198000	12.0	16800	265000	15.8	17000	495000	29.1	15400	540000	35.1
11	SADASIVAM	15117	16200	220000	13.6	14500	420000	29.0	13600	422000	31.0	14000	590000	42.1	14700	425000	28.9
12	NAGARAJ	16025	18800	200000	10.6	16000	212000	13.3	16100	320000	19.9	15800	394000	24.9	17000	265000	15.6
13	BUJIAMMAL	16976	11500	165000	14.3	14000	431000	30.8	14300	432000	39.3	15700	600000	38.2	16200	645000	39.8
14	PARAMESHWARAN	17006	16100	387000	24.0	14600	365000	25.0	13500	456000	33.8	12600	387000	30.7	13000	364000	28.0
15	PONNIYAMMAL	20659	16000	375000	23.4	14100	290000	20.6	13500	421000	31.2	13400	356000	26.6	16400	426000	26.0
16	MOHAN	25382	15800	148000	9.4	14400	210000	14.6	14000	613000	43.8	12200	610000	50.0	14400	510000	35.4
17	SAKTHIVEL	26099	20400	365000	17.9	15400	412000	26.8	21100	500000	23.7	17400	465000	26.7	14900	684000	45.9
18	RAMESH	26571	17500	165000	9.4	18600	300000	16.1	16000	324000	20.3	16600	394000	23.7	19800	395000	19.9
19	RAJESH KUMAR	26982	18400	198000	10.8	14900	378000	25.4	14700	478000	32.5	13500	398000	29.5	14900	487000	32.7
20	ARUN KUMAR	26984	28500	398000	14.0	12800	385000	30.1	14000	421000	30.1	14700	384000	26.1	11000	410000	37.3
21	PAUL RAJ	30145	15600	198000	12.7	14600	212000	14.5	14200	463000	32.6	12100	469000	38.8	16000	420000	26.3
22	ANJI	30614	16600	300000	18.1	13700	298000	21.8	14300	337000	23.6	15900	265000	16.7	16500	356000	21.6
23	YOUNIS	30988	18500	321000	17.4	15300	465000	30.4	14600	356000	24.4	13500	345000	25.6	14000	452000	32.3
24	KARTHICK	32656	14900	320000	21.5	14900	354000	23.8	13600	352000	27.9	13500	328000	24.3	16800	554000	33.0
25	MURUGAN	33829	17900	268000	15.0	14900	365000	24.5	14200	421000	29.6	13300	565000	42.5	13600	453000	33.3
26	FAROOQ	35456	17200	210000	12.2	15700	219000	13.9	16500	265000	16.1	17400	398000	22.9	16300	420000	25.8
27	MUTHULAKSHMI	35465	20900	398000	19.0	15000	398000	26.5	16900	387000	22.9	16500	488000	29.6	14600	384000	26.3
28	MANIKANDAN	36777	14500	222000	15.3	12500	300000	24.0	13000	265000	20.4	13100	332000	25.3	17400	468000	26.9
29	PARTHIBAN	44564	12500	398000	31.8	12900	201000	15.6	14200	421000	29.6	13200	374000	28.3	13600	341000	25.1
30	SUBRAMANI	45024	13800	210000	15.2	12900	574000	44.5	14900	554000	37.2	13500	400000	29.6	16500	398000	24.1
31	SHANKAR	45343	19000	298000	15.7	16100	315000	19.6	15600	188000	12.1	16600	335000	20.2	15400	390000	25.3
32	ESWARAN	45389	17000	265000	15.6	17800	198000	11.1	16800	199000	11.8	19500	298000	15.3	18700	342000	18.3
33	SAKTHI	45678	17400	250000	14.4	18700	245000	13.1	15800	398000	25.2	17000	265000	15.6	16400	315000	19.2
34	DEEPAN	45751	17800	298000	16.7	19000	200000	10.5	22500	335000	14.9	17600	380000	21.6	22000	389000	17.7
35	DAWOOD	46980	15500	255000	16.5	15200	252000	16.6	15200	400000	26.3	14500	298000	20.6	13500	574000	42.5
36	MAHESHWARI	47589	16900	312000	18.5	15800	356000	22.5	14900	375000	25.2	14200	397000	28.0	13200	456000	34.5
37	DINESH	53656	17500	225000	12.9	13900	341000	24.5	13200	452000	34.2	13500	587000	43.5	13200	359000	27.2
38	SRIDHARAN	54276	17800	298000	16.7	21800	100000	4.6	22500	335000	14.9	17000	330000	19.4	19000	389000	20.5
39	MANIKANDAN	56549	14500	260000	17.9	14400	428000	29.7	13900	206000	14.8	14900	345000	23.2	16000	320000	20.0
40	APPUKUTTI	63292	14400	115000	8.0	9400	195000	20.7	14500	198000	23.6	14800	597000	40.3	15200	350000	23.0
41	UMA DEVI	64565	16000	156000	9.8	15200	410000	27.0	16500	312000	18.9	15800	421000	26.6	14200	484000	34.1
42	SENTHIL	67327	16000	190000	11.9	15000	412000	27.5	14000	390000	27.9	13500	465000	34.4	17000	425000	25.0
43	KANNIYAMMAL	67453	16200	112000	6.9	16700	254000	15.2	16800	183000	10.9	19500	214000	11.0	17500	280000	16.0
44	NARENDRAN	112755	21000	158000	7.5	10500	115000	11.0	15900	190000	11.9	12900	214000	16.6	18200	256000	14.1
45	MOHAN	114760	14000	150000	10.7	14500	305000	21.0	14400	482000	46.3	12600	530000	42.1	14300	469000	32.8
46	RAGHUVARAN	116900	16400	250000	15.2	12500	365000	29.2	13600	365000	26.8	14900	296000	19.9	13800	374000	27.1
47	JAYAKUMAR	116998	18100	210000	11.6	13700	339000	24.7	11700	353000	30.2	15400	523000	34.0	14300	600000	42.0
48	RAJAN	117467	15200	208000	13.7	15300	360000	23.5	16700	260000	15.6	15500	475000	30.6	16700	218000	13.1
49	REVATHY	117504	17500	214000	12.2	16000	210000	13.1	17400	265000	15.2	16300	200000	12.3	19500	450000	23.1
50	ELUMALAI	122544	24700	337000	13.6	17700	377000	21.3	25000	335000	13.4	19300	341000	17.7	21200	246000	11.6

serial no	name	ip no	TC9	P9	PT9	TC10	P10	PT10	INFECTION	NATURE	EXPIRED	DURATION OF STAY
1	JAYARAJ	1597	14600	648000	44.4	10900	650000	59.6	NO		NO	10
2	SURESH	2359	19300	432000	22.4	20400	425000	20.8	YES	SSI	NO	18
3	JAYALAKSHMI	2932	25300	291000	11.5	24100	358000	14.9	YES	LRI	NO	24
4	RIYAZ	3698	15200	645000	42.4	13200	520000	39.4	NO		YES	12
5	ABAI SHARMA	4682	16500	540000	32.7	19800	525000	26.5	YES	UTI,SSI	NO	20
6	BANUMATHY	8545	15200	415000	27.3	15800	546000	34.6	YES	LRI,SSI	NO	19
7	SATHISH	9593	14200	465000	32.7	13200	545000	41.3	NO		NO	12
8	ANAND	10654	13500	425000	31.5	15400	326000	21.2	NO		NO	12
9	KARAN	13307	20900	412000	19.7	17900	395000	22.1	YES	LRI,SSI, UTI	NO	18
10	DHANDAPANI	13601	19900	451000	22.7	22900	513000	22.4	YES	LRI,SSI	YES	12
11	SADASIVAM	15117	12500	569000	45.5	13000	540000	41.5	NO		NO	12
12	NAGARAJ	16025	15400	360000	23.4	12000	574000	47.8	NO		NO	12
13	BUJIAMMAL	16976	18500	365000	19.7	19400	465000	24.0	NO		YES	13
14	PARAMESHWARAN	17006	13100	432000	33.0	13100	582000	44.4	NO		NO	12
15	PONNIYAMMAL	20659	14300	410000	28.7	19700	625000	31.7	NO		NO	12
16	MOHAN	25382	15900	545000	34.3	12300	560000	45.5	NO		NO	11
17	SAKTHIVEL	26099	18500	640000	34.6	14300	650000	45.5	YES	UTI,SSI	NO	12
18	RAMESH	26571	15600	374000	24.0	15300	458000	29.9	YES	SSI	NO	20
19	RAJESH KUMAR	26982	13700	521000	38.0	17500	465000	26.6	NO		NO	13
20	ARUN KUMAR	26984	17400	698000	40.1	12000	540000	45.0	NO		NO	13
21	PAUL RAJ	30145	13900	398000	28.6	14900	390000	26.2	NO		NO	12
22	ANJI	30614	14200	420000	29.6	12800	567000	44.3	NO		NO	10
23	YOUNIS	30988	19800	354000	17.9	12500	542000	43.4	NO		NO	11
24	KARTHICK	32656	19400	374000	19.3	12800	526000	41.1	NO		NO	11
25	MURUGAN	33829	16900	421000	24.9	13100	452000	34.5	NO		NO	11
26	FAROOQ	35456	16500	425000	25.8	19300	523000	27.1	YES	SSI	NO	21
27	MUTHULAKSHMI	35465	14800	383000	25.9	14200	412000	29.0	NO		NO	12
28	MANIKANDAN	36777	13900	484000	34.8	17400	450000	25.9	NO		NO	11
29	PARTHIBAN	44564	15400	425000	27.6	14000	452000	32.3	NO		NO	13
30	SUBRAMANI	45024	13600	430000	31.6	14600	465000	31.8	NO		NO	13
31	SHANKAR	45343	18500	365000	19.7	21600	395000	18.3	YES	SSI	NO	17
32	ESWARAN	45389	16900	354000	20.9	15900	412000	25.9	YES	UTI,LRI	NO	30
33	SAKTHI	45678	15400	456000	29.6	14900	654000	43.9	YES	SSI	NO	24
34	DEEPAN	45751	19000	450000	23.7	22600	420000	18.6	YES	LRI	N0	15
35	DAWOOD	46980	13800	680000	49.3	12000	398000	33.2	NO		NO	12
36	MAHESHWARI	47589	14800	498000	33.6	13000	500000	38.5	NO		NO	13
37	DINESH	53656	15400	478000	31.0	16900	398000	23.6	NO		NO	11
38	SRIDHARAN	54276	14300	320000	22.4	14900	332000	22.3	YES	LRI,SSI, UTI	NO	12
39	MANIKANDAN	56549	16500	376000	22.8	15400	400000	26.0	NO		NO	10
40	APPUKUTTI	63292	13700	346000	25.3	16500	410000	24.8	NO		NO	10
41	UMA DEVI	64565	14100	542000	38.4	18400	655000	35.6	NO		NO	11
42	SENTHIL	67327	16900	490000	29.0	14600	656000	44.9	NO		NO	11
43	KANNIYAMMAL	67453	18400	274000	14.9	19500	276000	14.2	YES	LRI,UTI,AA	YES	35
44	NARENDRAN	112755	13200	420000	31.8	12100	390000	32.2	NO		NO	14
45	MOHAN	114760	19800	606000	30.6	13200	436000	33.0	NO		NO	12
46	RAGHUVARAN	116900	14200	385000	27.1	13900	465000	33.5	NO		NO	13
47	JAYAKUMAR	116998	14400	562000	39.0	13500	565000	41.9	NO		NO	12
48	RAJAN	117467	16500	398000	24.1	16400	412000	25.1	YES	SSI,AA	NO	15
49	REVATHY	117504	17500	385000	22.0	15400	465000	30.2	YES	UTI,,LRI	NO	25
50	ELUMALAI	122544	29400	235000	8.0	18800	291000	15.5	YES	LRI,SSI, UTI	NO	23

KEY TO MASTER CHART

ip no	:	In Patient Number
ISS	:	Injury severity score
TC0	:	Total count on day 0
TC1	:	Total count on day 1
TC2	:	Total count on day 2
TC3	:	Total count on day 3
TC4	:	Total count on day 4
TC5	:	Total count on day 5
TC6	:	Total count on day 6
TC7	:	Total count on day 7
TC8	:	Total count on day 8
TC9	:	Total count on day 9
TC10	:	Total count on day 10
P0	:	Platelet count on day 0
P1	:	Platelet count on day 1
P2	:	Platelet count on day 2
P3	:	Platelet count on day 3
P4	:	Platelet count on day 4
P5	:	Platelet count on day 5
P6	:	Platelet count on day 6
P7	:	Platelet count on day 7
P8	:	Platelet count on day 8
P9	:	Platelet count on day 9
P10	:	Platelet count on day 10
PT0	:	Platelet total count ratio on day 0

PT1	:	Platelet total count ratio on day 1
PT2	:	Platelet total count ratio on day 2
PT3	:	Platelet total count ratio on day 3
PT4	:	Platelet total count ratio on day 4
PT5	:	Platelet total count ratio on day 5
PT6	:	Platelet total count ratio on day 6
PT7	:	Platelet total count ratio on day 7
PT8	:	Platelet total count ratio on day 8
PT9	:	Platelet total count ratio on day 9
PT10	:	Platelet total count ratio on day 10
SSI	:	Surgical Site Infection
UTI	:	Urinary Tract Infection
LRI	:	Lower Respiratory Tract Infection
AA	:	Abdominal Abscess