TO STUDY THE FETOMATERNAL OUTCOME AND COMPLICATIONS IN PREGNANCY WITH MODERATE AND SEVERE SPLENOMEGALY

DISSERTATION SUBMITTED TO

In partial fulfillment of the requirement for the degree of

DOCTOR OF OBSTETRICS AND GYNAECOLOGY

(Branch II) M. S. (OBSTETRICS AND GYNAECOLOGY)

of

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TIRUNELVELI MEDICAL COLLEGE

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**TO STUDY THE FETOMATERNAL OUTCOME AND COMPLICATIONS IN PREGNANCY WITH MODERATE AND SEVERE SPLENOMEGALY"** to the Tamilnadu Dr. M.G.R Medical University, Chennai, in partial fulfillment of the requirement for the award of M.S. Degree Branch – II (Obstetrics and Gynaecology) is a bonafide research work carried out by her under direct supervision & guidance.

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This is to certify that "**TO STUDY THE FETOMATERNAL OUTCOME AND COMPLICATIONS IN PREGNANCY WITH MODERATE AND SEVERE SPLENOMEGALY"** presented here in by **Dr.G.KOHILA** is an original work done in the Department of Obstetrics and Gynaecology, Tirunelveli Medical College Hospital, Tirunelveli for the award of Degree of M.S. (Branch II) Obstetrics and Gynaecology under my guidance and supervision during the academic period of 2019 -2022.

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DECLARATION

I solemnly declare that the dissertation titled "**TO STUDY THE FETOMATERNAL OUTCOME AND COMPLICATIONS IN PREGNANCY WITH MODERATE AND SEVERE SPLENOMEGALY"** is done by me at Tirunelveli Medical College hospital, Tirunelveli. I also declare that this bonafide work or a 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 or abroad.

The dissertation is submitted to The Tamilnadu Dr. M.G.R.Medical University towards the partial fulfilment of requirements for the award of M.S. Degree (Branch II) in Obstetrics and Gynaecology.

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PROTOCOL TITLE: TO STUDY THE FETOMATERNAL OUTCOME & COMPLICATION IN PREGNANCY WITH MODERATE AND SEVERE SPLEENOMEGALY.
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Dear Dr. G.Kohila, M.S. The Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 20.11.2019.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1. TIREC Application Form ☐
2. Study Protocol ☐
3. Department Research Committee Approval ☐
4. Patient Information Document and Consent Form in English and Vernacular Language ☐
5. Investigator's Brochure ☐
6. Proposed Methods for Patient Accrual Proposed ☐
7. Curriculum Vitae of the Principal Investigator ☐
8. Insurance /Compensation Policy ☐
9. Investigator's Agreement with Sponsor ☐
10. Investigator's Undertaking ☐
11. DCGI/DGFT approval ☐
12. Clinical Trial Agreement (CTA) ☐
13. Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA) ☐
14. Clinical Trials Registry-India (CTRI) Registration ☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
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   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in theoriginal project. (Page no. Clause no. etc.)
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Member Secretary, TIREC
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State of Tamilnadu, South India

CERTIFICATE – II

This is to certify that this dissertation work titled "**TO STUDY THE FETOMATERNAL OUTCOME AND COMPLICATIONS IN PREGNANCY WITH MODERATE AND SEVERE SPLENOMEGALY"** of the candidate **Dr. G.KOHILA** with registration Number **221916356** for the award of **M.S.** Degree in the branch of **OBSTETRICS AND GYNAECOLOGY (II).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **0% percentage** of plagiarism in the dissertation.

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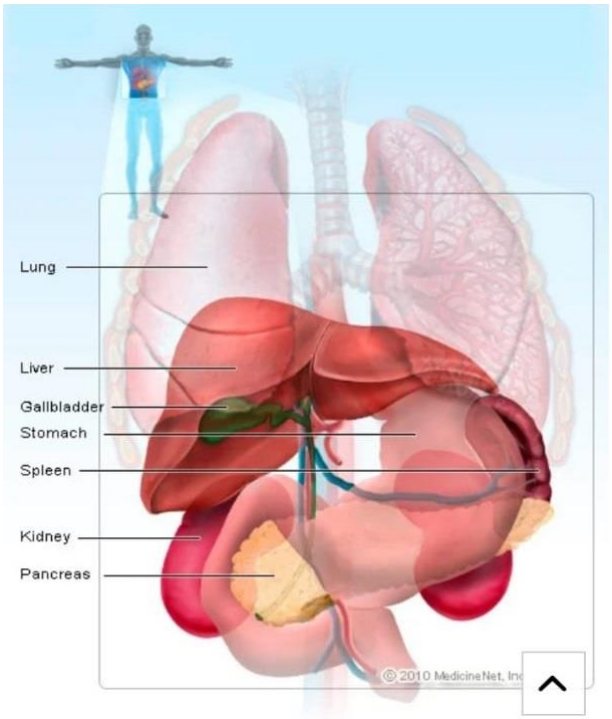
INTRODUCTION

The Spleen is the largest lymphoid organ located in the left hypochondriac region of abdomen. It contains specialised group of lymphocytes and myeloid cells which are arranged in distinct anatomical compartments called as red pulp and white pulp. It has important function of mobilizing body's immune cells to the periphery and scavenging the aging blood cells. It plays important role in various physiological functions like iron metabolism, defense against invading micro organisms and act as a reservoir for various types of blood cells. Spleen is not palpable in normal individuals and a palpable spleen, even if it is asymptomatic is an indication for further evaluation.



Splenomegaly in patients accounts to 0.3% of all hospital admissions. The causes of splenomegaly are extensive , with the most common cause being haematological (16-66%),followed by porto- hepatic causes (9-41%), storage diseases , infectious causes (9-36%),

secondary to systemic diseases / congestive / Inflammatory (4-10%), tropical splenomegaly syndrome , primary splenic causes (1-6%), followed by idiopathic / cryptogenic causes (1-2%). If a definitive etiology cannot be obtained even after extensive non invasive and invasive haematological, radiological and immunological investigations, these patients can be given diagnosis as “Splenomegaly of Indeterminate origin” or “ obscure splenomegaly”.

During Pregnancy there is no physiological hypertrophy or enlargement of spleen described so far. Any splenic enlargement during pregnancy is pathological and have many known diverse etiology, which does not differ much from the etiology of splenomegaly in general population. However, the complications of splenomegaly, and risks to pregnant women and fetus as because of the enlargement are worrisome as compared to general population. Pregnancy may also occur as an aggravating factor to the underlying disease causing splenomegaly. Complications of splenomegaly such as infections due to immune mechanism failure, bleeding risk due to splenic platelet sequesteration , splenic rupture are more detrimental and fatal to pregnant women than general population. Foetal complications like intra uterine growth restriction are also found to be more commonly associated with pregnant women with splenomegaly in the literature. Although various studies account for the etiology of splenomegaly during pregnancy, knowledge of obstetric complications that occurs due to splenomegaly in pregnant women and their management is not fully known. Deterioration of hemodynamic status during labour can occur in pregnant women with splenomegaly, the etiology being the underlying pathology of splenomegaly or physiological changes of pregnancy were not properly studied. Further studies are needed regarding the obstetric complications during intrapartum and postpartum period in pregnant women with splenomegaly as the management needs expertise multidisciplinary approach and is still lacking.

REVIEW OF LITERATURE

The spleen (Latin low spirits, origin from ancient Greek) is a wedge-shaped organ which is located in the left hypochondriac region of abdomen, and partly in the epigastrium. Initially, the function of spleen was not found. Galen believed it was the source of black bile and the idiom " to vent one's spleen" attest to older beliefs that the spleen had an important influence on psyche and emotions. Later, Spleen is found to be the largest lymphoid organ of the body. It is wedged in between the fundus of the stomach and the diaphragm. The spleen is tetrahedral in shape and it is protected by 9^{th}, 10^{th} & 11^{th} rib lying outside. The spleen is very soft in texture, highly vascular and dark purple in color. On an average, a healthy adult human spleen is 1 inch or 2.5 cm thick, 3 inches or 7.5 cm width, 5 inches or 12.5 cm in length and 7 ounces or 150-250 grams in weight. The spleen lies along the long axis of 10^{th} rib.

Normally, the spleen is not palpable. Anatomically, the spleen is directed downwards, forwards and laterally, making an angle of about 45 degrees with the horizontal plane. The spleen has two ends, three borders, two surfaces, two angles and hilum.

- ENDS OF SPLEEN

The anterior or lateral end is expanded and is more like a border. It is directed downwards forwards, and reaches the midaxillary line. The posterior or medial end is rounded. It is directed upwards, backwards and medially, and rests on the upper pole of the left kidney.

- BORDERS OF SPLEEN

Spleen has three borders which are superior, inferior and intermediate. The superior border is characteristically notched near its anterior end. The inferior border is rounded where as the intermediate border is also rounded and directed towards the right.

- SURFACES OF SPLEEN

It has two surfaces namely diaphragmatic and visceral surface respectively. The diaphragmatic surface is convex and smooth. The visceral surface is concave and irregular.

- ANGLES OF SPLEEN

It has two angles which are anterobasal angle forming the junction of superior border with lateral or anterior end. It is the most forward projecting part of spleen. When spleen is enlarged, the spleen in relation to left 9th-11th ribs is felt first, so this is called as "clinical angle of spleen". Another is Posterobasal angle which lies at junction of inferior border with lateral or anterior end of spleen.

- HILUM

Hilum lies between superior and intermediate borders and is represented by a depressed area in the capsule. It is pierced by branches and tributaries of splenic artery, splenic vein, efferent lymphatic vessels, splenic nerve plexus.

LIGAMENTS OF SPLEEN

The spleen is surrounded by peritoneum, and is suspended by many ligaments.

- The gastrosplenic ligament
- The lienorenal ligament

- The phrenicocolic ligament

SURFACES OF SPLEEN – ANATOMICAL RELATIONS

VISCERAL SURFACE

The visceral surface of spleen has important relations to the fundus of the stomach, the splenic flexure of the colon, the left kidney through its anterior surface, and the tail of pancreas.

Gastric impression - fundus of the stomach

Colonic impression - splenic flexure of the large intestine

Pancreatic impression - tail of the pancreas

Renal impression - left kidney

DIAPHRAGMATIC SURFACE

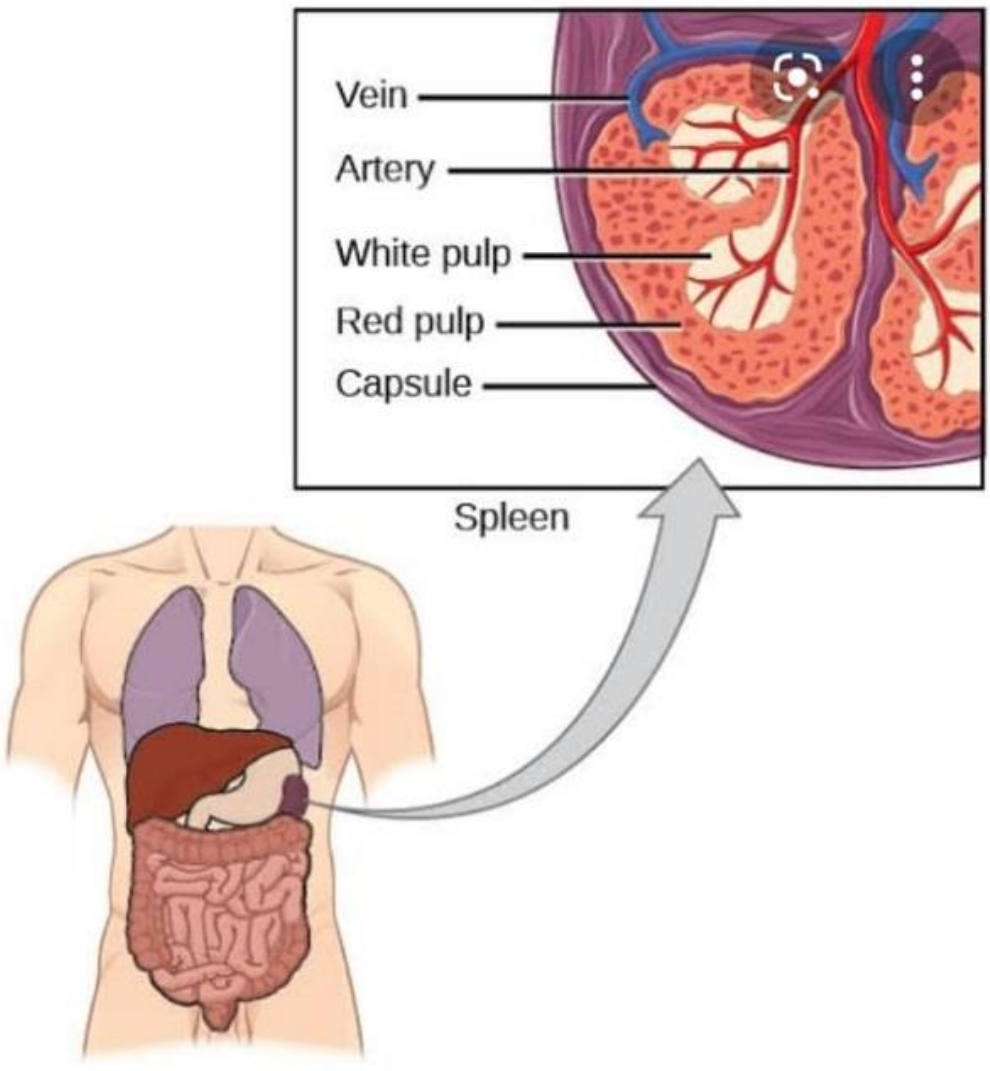
The diaphragmatic surface is smooth and convex related to the diaphragm which separates the spleen from the costodiaphragmatic recess of pleura, lung and 9th, 10th and 11th ribs of the left side.

STRUCTURAL ORGANISATION OF SPLEEN

Anatomically , Spleen is composed of four regions called as

1. Capsule
2. Red Pulp
3. White Pulp
4. Marginal Zone

Each region of spleen has unique structural architecture and have specific functional significance.



CAPSULE

The capsule of spleen is made up of dense connective tissue, elastic and smooth muscle fibres and contains sympathetic nerve fibres from splenic nerve plexus. This splenic capsule invaginates into the parenchyma of spleen to form trabeculae.

RED PULP

It constitutes 70% of the total splenic volume in human adult. Ultrastructurally , Red pulp contains blood filled numerous sinuses separated by spongy cellular cords called as cords of

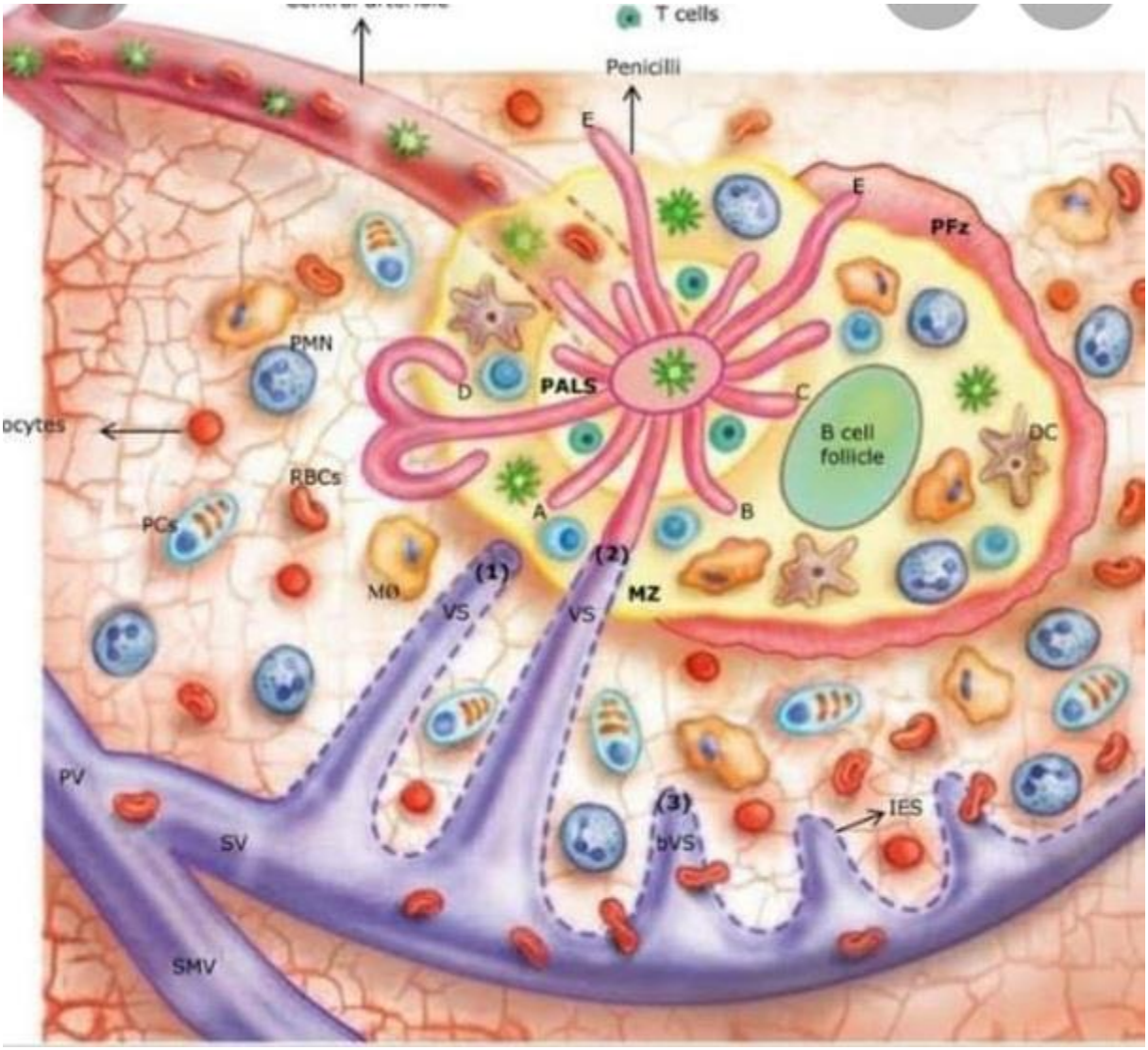
Billroth. Sinuses of red pulp are filled by blood from penicillar arteries and are rich in platelets. Main functions of red pulp of spleen include blood filtration, antigenic stimulation and proliferation of B and T cells and specific production of antibodies. Also, it has been revealed to have a reservoir of monocytes capable of rapidly migrating into injured tissues and mediating local inflammatory response.

WHITE PULP

White pulp is made up of follicles , arterial vessels along with its periarterial lymphatic sheath (PALS) and loose lymphatic tissues. Germinal centre is the center of both primary and secondary follicles. The lymphatic sheath which envelops the central artery and arteriole in white pulp is mainly made up of CD4+ T cells. The follicles contain mainly B cells and T cells near PALS. B cell clonal expansion occurs in germinal centre of B cell follicle following antigenic stimulation. The function of White pulp is mainly immunological in nature.

MARGINAL ZONE

Marginal zone is made up of numerous macrophages and antigen presenting cells along with fewer lymphocytes. Anatomically marginal zone surrounds white pulp and acts as an interface separating the white pulp from the red pulp. The Marginal zone contains the highest concenteration of blood antigens in spleen as because the splenic arterial blood reaches the marginal zone first. Marginal zone B cells show somatic hypermutation, clonal expansion & B cell positive selection.



ARTERIAL SUPPLY

The spleen is supplied by the splenic artery which is the largest branch of the coeliac trunk. The artery is tortuous in its course to allow for movements of the spleen. It passes through the lienorenal ligament to reach the hilum of the spleen where it divides into five or more branches called as trabecular arteries. These trabecular arteries are located within the trabeculae entering the splenic parenchyma. Central artery is a branch of this trabecular arteries. A peculiar feature of this central artery and arteriole is these vessels have rich periarterial lymphatic sheath microcirculation and lymphatic organization. These central arteriole when they leave white pulp

become straight vessels called Penicillar arteries or arteriole. Heavy sheaths of lymphatic tissue or lymphocytes are present around central arteriole at different points. Whereas these penicillar arteries supplying red pulp lack this rich periarterial sheath of lymphocytes.

There are two widely accepted theories regarding circulation of blood in spleen.

- Theory of Closed Splenic Circulation
- Theory of Open Splenic Circulation

According to closed theory of splenic circulation, the capillaries are continuous with the venous sinusoids that lie in the red pulp; the sinusoids join together to form veins. However, according to open theory of splenic circulation, the capillaries end by opening into the red pulp from where the blood enters the sinusoids through their walls. Still others believe in a compromise theory, where the circulation is open in distended spleen and closed in contracted spleen. The splenic circulation is adapted for the mechanism of separation and storage of the red blood cells. Blood flows into the spleen at a rate of about 150 ml/min. Old and damaged erythrocytes are less deformable and are retained in cords of sinuses , where they are destroyed and their components recycled. Red cell – inclusion bodies such as parasites, nuclear residua like howel jolly bodies or denatured hemoglobin called as Heinz bodies are pinched off in the process of passing through the slits , a process called as PITTING. The culling of dead and damaged RBCs and pitting of cells with inclusion bodies appear to occur without significant time delay because the blood transit time through spleen is only slightly slower when compared to other organs.

VENOUS DRAINAGE OF SPLEEN

Venous Drainage of spleen is mainly by splenic vein. Blood from red pulp is collected through venous sinuses into trabecular vein. These trabecular veins converge at hilum to form splenic

vein. It runs a straight course behind the pancreas. It joins the superior mesenteric vein behind the neck of the pancreas to form the portal vein. Its tributaries are the short gastric, left gastroepiploic, pancreatic and inferior mesenteric veins.

VASCULAR SEGMENTS OF SPLEEN

On the basis of its blood supply, the spleen is said to have superior and inferior Vascular segments. The two segments are separated by an avascular plane. Each segment may be subdivided into one to two disc-like middle segments and a cap-like pole segment.

Splenic tissue proper has no lymphatics. A few lymphatics arise from the connective tissue of the capsule and trabeculae and drain into the pancreatico - splenic lymph nodes situated along the splenic artery. Spleen is innervated by Sympathetic fibres which are derived from the coeliac plexus. They are vasomotor in nature. They also supply some smooth muscle which are present in the capsule.

Functions of the Spleen

Spleen has various important physiological role right from the period of organogenesis in the intrauterine life. It is a functionally diverse organ with active roles in immunosurveillance and hematopoiesis. The important functions include

- ✓ Phagocytosis

The spleen is an important component of the reticuloendothelial system. The splenic phagocytes include:

a. The reticular cells and free macrophages of the red pulp

b. Modified reticular cells of the ellipsoids.

c. Free macrophages and endothelial cells of the venous sinusoids, and

d. Surface reticular cells of the lymphatic follicle.

The phagocytes present in the organ remove cell debris and old deformed RBCs, other blood cells and microorganisms, and thus filter the blood and helps to maintain the quality of blood. Spleen plays vital role in prevention of major infectious & hematological diseases like malaria, infectious mononucleosis, EBV infection, sickle cell anaemia, leukemia, pernicious anaemia, hodgkin's disease, sarcoidosis etc…In absence of spleen like surgical removal, it is found in various animal studies that kupffer cells of liver may act as phagocytic cells to remove aging RBCs from the circulation with limited capacity.

- ✓ Hematopoiesis

The spleen is an important haemotopoietic organ during foetal life, particularly upto fifth month of intrauterine life. After birth, erythropoiesis ceases yet lymphopoiesis in spleen continues throughout life. The lymphocytes manufactured in it take part in immune responses of the body. In the adult spleen, haemopoiesis can restart in certain pathologic conditions like chronic myeloid leukaemia and myelosclerosis. Recently, it has been found that the splenic monocytes play a significant role in regeneration of heart tissue following myocardial infarction.

- ✓ lmmune responses

Spleen acts as the lymphoid organ which induces primary immune response against various invading organisms like bacteria, fungi, viruses, prions and other infective

agents. In addition, it also induces immune response against blood antigen (eg) ABO / Rh incompatibility). Thereby, finally it removes the antibody coated micro-organisms and antibody coated blood cells from the circulation. The spleen plays important role in production and sensitization of immune cells to antigenic response and thereby protects other organs from harmful infectious agents/antigens. Majority of T cells in spleen are CD4+ T cells. These cells are found specifically in PALS, splenic cords, marginal zone , sinuses and in the periphery of follicles of spleen. Where as B cells are located richly in Germinal centre of follicles, marginal zone and splenic cords. The B cells of marginal zone are non- migratory and tissue specific. These B cells in marginal zone when encounter blood antigens complementary to their specific antibody receptors develop into plasma cells and these differentiated plasma cells produce antigen specific antibody. CD 4+ T cells found in PALS act as supporting cells for growth and differentiation of B cells.

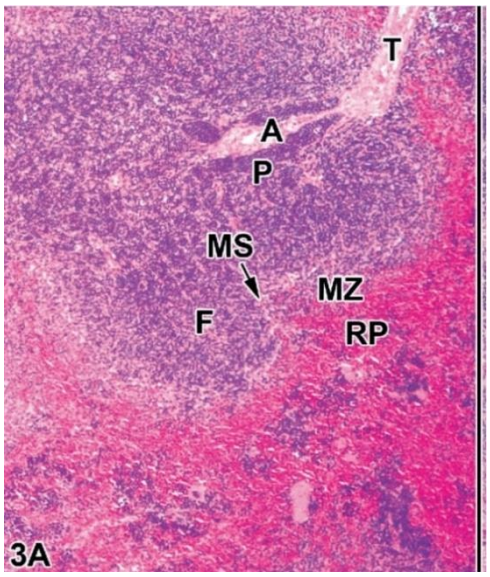
The process specific immune reaction proper starts when T helper cells secrete cytokines in response to antigenic stimulation. These cytokines pave way to the differentiation of T cells into T1 helper and T2 cytotoxic cells. Naïve B cells from bone marrow are transferred through blood into germinal center(GC) of follicles, where they are primed under the influence of cytokines like Interleukin – 21, they become plasmoblasts and finally into mature B cells. Following maturation of B cells, occurs the process Beta cell receptor (BCR) organization and genetic recombination. GC derived B cells are also transferred into memory B cells in addition to high affinity plasma cells which produce specific antibodies. These memory B cells are capable of get transferred into plasma cells of specific antibody type on subsequent exposure to that particular antigen. Apart from IL-21, Activated T helper cells

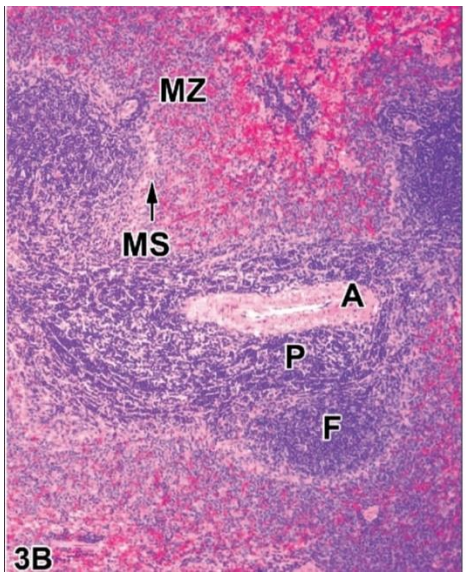
also produce other cytokines like IL-1, IL-4, IL-5, IL-6 which promote B cell proliferation and differentiation.

- ✓ Storage of RBCs

Red blood cells can be stored in the spleen and released into the circulation when needed. It also plays a major role in recycling of iron in iron metabolism. This function is better marked in animals than in man. However, normal human spleen does not sequester or store RBCs and does not contract in response to sympathetic stimuli. The normal human spleen contains approximately one – third of the total body platelets and a significant number of marginated neutrophils. These sequestered cells are available when needed to respond to bleeding or infection.

HISTOTOGY





Histologically, spleen is made up of the following four component parts.

1. Supporting fibroelastic tissue, forming the capsule, coarse trabeculae and a fine reticulum

2. White Pulp consisting of lymphatic nodules arranged around an arteriole called Malpighian corpuscle. These are composed of

a. Lymphoid follicles rich in B- lymphocytes

b. Periarteriolar lymphoid sheaths (PALS) rich in T- lymphocytes.

3. Red pulp is formed by the collection of cells in the interstices of reticulum, in between the sinusoids. The cell population includes:

    a. All types of lymphocytes (small, medium and large),

    b. All three types of blood cells (RBC, WBC and platelets), and

    c. The fixed and free macrophages.

4. Vascular system transverses the organ and penetrates it thoroughly.

DEVELOPMENT

Spleen develops in the mesoderm in the cephalic part of left layer of dorsal mesogastrium. It is one of the abdominal organs which is developed from mesoderm, while most other abdominal organs are derived from endoderm.The development occurs during sixth week of intrauterine life. Number of nodules develop which soon fuse to form a lobulated spleen

Accessory Spleens or Spleniculi

An accessory spleen is a small splenic nodule that has failed to fuse with the splenic tissue during early embryogenesis. Seen in 10% of population. They are typically about 1 cm in diameter. These may be found:

1 In the derivatives of the dorsal mesogastrium, i.e. gastrosplenic ligament, lienorenal ligament, gastrophrenic ligament and greater omentum.

2 In the broad ligament of the uterus.

3 In the spermatic cord.

SPLENOSIS

This is a condition where displaced pieces of splenic tissue autotransplant are present in abdominal cavity as accessory spleens. Occurs usually following or trauma or after splenectomy.

POLYSPLENIA

This is a congenital condition manifested by multiple small accessory spleens, rather than a single full sized normal spleen. Polysplenia may occur as an isolated condition, or sometimes may be associated with other congenital anomalies like intestinal malrotation , biliary atresia, dextrocardia (cardiac anomaly). These accessory spleens are usually non – functional in nature.

ASPLENIA

This refers to a condition characterized by non- functioning spleen which can be congenital or acquired. These patients exhibit increased susceptibility to infections particularly sepsis due to encapsulated polysaccharide bacteria are common.

SPLENOMEGALY

In normal individuals, spleen is usually not palpable and a palpable spleen always an indicator of an underlying disorder. Any increase in normal functions of the spleen can result in splenomegaly. Normal Spleen usually weighs < 250 gram, lies entirely within the rib cage, has a

maximum cephalocaudal diameter of 13 cm by USG or maximum length of 12 cm and width of 7 cm by radionuclide scanning and is usually not palpable on abdominal examination. Spleen also decreases in size with the increase in age of individual. Symptomatic splenomegaly should always be taken into account and should be investigated thoroughly to establish the etiology. The degree of splenomegaly varies according to the etiology causing it. For example, most of the acute conditions like infectious etiology causes mild splenomegaly whereas chronic etiology like hematological causes produces massive splenomegaly. In spite of intensive invasive and non-invasive investigation to establish the etiology, in a subset of patients diagnosis is difficult to ascertain and being labelled as "Splenomegaly of Indeterminate origin".

Splenomegaly may be symptomatic or asymptomatic. Symptoms can be either due to splenomegaly per se or it can be due to underlying etiology which is causing the splenomegaly. Symptoms associated with splenomegaly include

- Pain / Heaviness/ vague discomfort in the upper abdomen (left hypochondrium) that may radiate to the back or shoulder – acute pain due to stretching of capsule, splenic infarction , inflammation of the capsule in splenic abscess.
- Early satiety (as enlarged splenomegaly may compress stomach) – massive splenomegaly
- Indigestion
- Hiccups (may occur because of irritation of diaphragm)
- Anaemia (easy fatiguaebility, shortness of breadth)
- Easy bruising/petechiae (due to low platelet count)
- Loss of appetite & weight (infiltrative disorders)
- Constitutional symptoms (infectious diseases)

- Menstrual disturbances
- Sepsis (ineffective opsonisation to encapsulated organisms)

Asymptomatic splenomegaly can be found out by systemic abdominal examination. Inspection may reveal a fullness in left hypochondrium region. Splenomegaly by examination can be made out by both palpable and percussion

Palpation of Spleen

• Normally, spleen should have enlarged 2-3 times to become palpable. Direction of enlargement of spleen is actually diagonally towards right iliac fossa.

• So during Palpation for splenomegaly , we have to start from right iliac fossa and proceed towards left hypochondrium. Bimanual palpation is done usually.

• Wait for one full phase of respiration. At the height of inspiration, release the pressure on the examining hand so that the finger tips slip over the lower pole of spleen, confirming its presence and surface characteristics like notching.

• If spleen is not palpable, move the examining hand upwards after each inspiration until the finger tips are under the costal margin.

• Repeat this process along the entire rib margin as the position of the enlarging splenic tip is variable. When the spleen tip is felt, the finding is recorded as centimeters below left costal margin at some arbitrary point (from midpoint of umbilicus or xiphisternum junction). This allows the examiner to determine changes in size over time.

PALPATION TECHNIQUES FOR MILD SPLENOMEGALY

• **Middleton's manoeuvre**

PERCUSSION OF SPLEEN

Splenic Dullness extends from left lower ribs to the left hypochondrium and left lumbar region. Splenic dullness gives way to the resonance of surrounding bowel. In cases of mild splenomegaly in which enlargement of spleen cannot be established by palpation, splenomegaly can be diagnosed by percussion. Methods of percussion of spleen are

1. Nixon's method

2. Castell's method

3. Percussion of Traube's Semilunar space

Sensitivity and specificity of finding splenomegaly by palpation and percussion methods are 56-71% and 59-82% respectively when compared to USG or scintigraphy. Splenomegaly once established by palpation and percussion , can be graded as mild, moderate and massive as each grade is associated with certain diagnostic and prognostic significance. Most widely acceptable grading of splenomegaly is Hackett's grading.

HACKETT's GRADING OF SPLENOMEGALY

This is a WHO accepted grading method of splenomegaly and has been widely used during Endemic malaria survey and surveillance purposes. It can be used used for routine clinical examination also.

Class 0 – Spleen not palpable even on deep inspiration

Class 1 – Spleen just palpable below costal margin on deep inspiration

Class 2 – Spleen palpable but not beyond a horizontal line half way between the costal margin and umbilicus

Class 3 – Spleen palpable more than half way to umbilicus, but not below a line running horizontally through umbilicus

Class 4 – Spleen palpable below umbilicus but not below a horizontal line between umbilicus and pubic symphysis

Class 5 – Spleen extending lower than class 4

Class 1 & 2 – is regarded as Mild Splenomegaly

Class 3 – is considered as Moderate splenomegaly

Class 4 & 5 – belongs to Massive Splenomegaly

MASSIVE SPLENOMEGALY

Massive splenomegaly is viewed as a stressful problem usually due to its overtly symptomatic nature, fear of serious underlying etiology, and accompanied complications. Massive splenomegaly is defined by various definitions in literature. They are

- Clinically palpable spleen > 15 cm or 15 finger widths below ribcage or at umbilicus level or lower
- The longest diameter being > 18 cm on radiological imaging or described as greatly enlarged (MRI, CT, Ultrasound)
- Wet weight > 1500 g at excision or autopsy

SPLENOMEGALY - ETIOLOGY

Causes of splenomegaly can be established according to the size of spleen as certain diseases are more commonly associated with mild or massive splenomegaly. Most common causes associated with various grades of splenomegaly are given

Mild splenomegaly (up to 5 cm) – Causes

- Congestive cardiac failure
- Acute malaria
- Typhoid
- Infective endocarditis
- Septicaemia
- Systemic lupus erythematosus
- Rheumatoid arthritis
- Thalassaemia minor
- Miliary tuberculosis
- Leptospirosis
- HIV.

Moderate Splenomegaly (5-8 cm) – Causes

- Viral hepatitis
- Cirrhosis
- Lymphomas
- Leukaemias
- Infectious mononucleosis
- Haemolytic anaemias
- Splenic infarcts
- Splenic abscess
- Amyloidosis
- Haemochromatosis
- Polycythaemia.

Massive Splenomegaly (> 8 cm) – Causes

- Chronic myeloid leukaemia
- Myeloid metaplasia
- Myelofibrosis
- Hairy cell leukaemia
- Gaucher's disease
- Niemann-Pick disease
- Sarcoidosis
- Thalassaemia major
- Chronic malaria
- Kala-azar
- Congenital syphilis
- Extrahepatic portal vein obstruction
- Schistosomiasis
- Diffuse splenic haemangiomatosis
- Lymphoma
- Polycythaemia.

MECHANISM OF SPLENIC ENLARGEMENT

Pathophysiologic mechanism underlying splenic enlargement are as follows:

- Hyperplasia or hypertrophy related to a particular splenic function such as reticuloendothelial hyperplasia (work hypertrophy) in diseases such as hereditary spherocytosis or thalassemia syndromes that require removal of large number of defective red blood cells or immune response

to various systemic infections like infectious mononucleosis, subacute bacterial endocarditis, or due to immunological diseases like immune thrombocytopenia, SLE, Felty's syndrome

- Passive Congestion due to decreased blood flow from the spleen in conditions that produce portal hypertension eg): Cirrhosis, Budd- chiari syndrome, Congestive cardiac failure (CCF)
- Infiltrative diseases of spleen - like lymphomas, metastasis, amyloidosis, Gaucher's disease, myeloproliferative diseases & extramedullary hematopoiesis

CLASSIFICATION OF SPLENOMEGALY – ACCORDING TO ETIOPATHOGENESIS

- Enlargement due to increased demand for splenic function
- Enlargement due to abnormal splenic/ portal blood flow
- Infiltration of spleen
- Unknown etiology

SPLENOMEGALY IN PREGNANCY

Pregnancy with splenomegaly is challenging condition because of underlying diverse etiology and potentially adverse outcomes. Splenomegaly during pregnancy is a testing situation for the managing obstetrician as it is associated with a myriad of medical complications both to the mother and the foetus. According to the underlying etiology of splenomegaly, outcome of the pregnancy varies. For example pregnancy with splenomegaly due to infectious diseases that have been adequately treated carries good prognosis where as related to chronic conditions like hematological malignancy results in poor prognosis for both mother and foetus. Also physiological changes of pregnancy alon with the pathology causing splenomegaly can cause maternal deterioration of hemodynamic status. A multidisciplinary approach in tertiary care

centre with involvement of multiple specialities like gastroenterologist , hematologist, intensivist can yield successful maternal and fetal outcomes

Most commonly associated causes of splenomegaly in pregnancy was studied from literature. They include infectious diseases such as Chronic malaria(tropical splenomegaly syndrome), tuberculosis, kala- azar, hemolytic anaemia, hemoglobinopathies like thalassemia, myelofibrosis, cirrhotic or non-cirrhotic portal hypertension(non- cirrhotic portal fibrosis), extra- hepatic portal vein obstruction, collagen vascular disorders (SLE), other myeloproliferative disorders, banti's disease, splenic tumour(rarest)etc…

TROPICAL SPLENOMEGALY SYNDROME

Hyper-reactive malarial splenomegaly or tropical splenomegaly is a common cause of splenic enlargement in pregnant females in malaria endemic areas. Patients usually have anaemia, hepatosplenomegaly. Spontaneous rupture of spleen can occur during acute presentation. Certain diagnostic criteria is followed for diagnosis of tropical splenomegaly syndrome. They include

- Residence in malaria endemic areas
- Gross splenomegaly > 10cm
- Elevated serum Ig M level for malarial antigen – 2 SD or more
- Hypersplenism
- Clinical or immunologic response to antimalarial therapy
- Hepatic sinusoidal lymphocytosis in liver biopsy.

Anti-malarials stay main mode of treatment for this condition. Mefloquine is the most common anti-malarial drug used. Prognosis is good and recovery is monitored by splenic size improvement and symptomatic relief.

EXTRA HEPATIC PORTAL HYPERTENSION

Another common cause of splenomegaly in pregnant female is extra hepatic portal hypertension which may be caused by various congestive, infectious, storage, myeloproliferative, hemolytic and structural etiologies.. Splenomegaly may be the only manifestation. Hypersplenism is common and severe with pronounced anaemia. Ascites is a common feature of non- cirrhotic portal fibrosis. Bleeding from esophageal varices are relatively common (40%) and is the most common complication encountered during 2^{nd} & 3^{rd} trimester of pregnancy because of the overlap of physiological changes in circulatory system during pregnancy and portal hypertensive changes. Thrombocytopenia occurs yet with functionally normal platelets accounting for lesser incidence of bleeding manifestations. Variceal haemorrhage can be treated symptomatically and blood products are usually needed during delivery. Severe anaemia and near fatal variceal haemorrhage are the indication for splenectomy. Pregnancy is usually well tolerated and prognosis is good as because of preserved liver functions.

HEMOGLOBINOPATHIES

Thalassemia is most common hemoglobinopathy encountered during pregnancy with splenomegaly patients followed by Hb E disease and sickle cell anaemia. Hb E disease is usually asymptomatic and goes unnoticed. Thalassemia trait/ minor may have severe anaemia and can require blood transfusion during pregnancy. Some patients may manifest with anaemia with splenomegaly first time during pregnancy and may require frequent blood transfusions for good maternal & fetal outcome.

Etiology should be ascertained as management of disease per se and expected complications differ in each case. Successful medical treatment of the primary disorder in cases of splenomegaly can lead to regression of hypersplenism without the need for surgery.

MATERNAL COMPLICATIONS OF SPLENOMEGALY

Most of the pregnant women with splenomegaly presents with anaemia, thrombocytopenia and subsequent bleeding tendency due to splenic sequesteration of platelets, increased risk of infection etc..

- HYPERSPLENISM

Hypersplenism often develops in parallel with splenomegaly. Triad of hypersplenism includes Pancytopenia, Cellular bone marrow & improvement of pancytopenia after splenomegaly. Pathophysiologic mechanisms underlying hypersplenism depends upon the etiology underlying it. Splenic blood volume increases due to the increased venous pressure leading to congestive splenomegaly, or because of the increased splenic arterial blood flow induced by a variety of diseases, resulting in hyperemic splenomegaly. As a consequence, there is retention of a large number of leukocytes, erythrocytes and platelets in the spleen. The number of retained blood cells can be 5.5-20 times higher than the normal level, thus facilitating capture, phagocytosis or destruction of blood cells by phagocytes resulting in peripheral pancytopenias.

In 1965, Aster found by using 51Cr-labeled platelets that under normal circumstances, approximately one-third of platelets are stored in the human spleen, and the remaining two-thirds in the blood circulation. In hypersplenism, 50-90% of platelets are retained in the enlarged spleen resulting in a reduction of platelets in the circulating blood. Furthermore, Aster demonstrated that the distribution of platelets returned to normal after splenectomy, which eliminated the retention of platelets by the splenic blood pool.

In comparing the spleen in hypersplenism with a normal spleen, Ma et al found many differentially expressed cytokines, 21 of which were significantly upregulated in hypersplenism, including cytokines related to monocyte chemotaxis and Macrophage activation, such as

macrophage colony‑stimulating factor (M-CSF), tumor necrosis factor (TNF)-β, interferon (IFN)-γ, interleukin (IL)-10, MDC/CCL22, MCP-2/CCL8, and SDF-1/CXCL12. Moreover, M-CSF, TNF-β, and IFN-γ promoted transformation of blood monocytes to Macrophages and maintained activation of Macrophagess .

Five cytokines have been demonstrated to be significantly downregulated during hypersplenism, including IL-1β, BLC/CXCL13, MCP-1, EGF, and BDNF.

Under normal circumstances, a mutual restriction and a dynamic balance are present between cytokine or gene upregulation and cytokine or gene downregulation. Significant changes were observed in the expression of cytokines or genes in the spleen of patients with hypersplenism . The differentially expressed cytokines or genes may play an important role in immune cell activation in the spleen and the changes in the immune function in patients with hypersplenism.

The spleen is the largest lymphoid organ, and is an important site for the production of antibodies. Antigens unprocessed by the liver may enter the periphery of splenic lymphoid follicles (splenic nodules), where reactions of immature lymphocytes and plasma cells occur after antigen stimulation, thus producing antibodies, which may destroy blood cells.. Platelets entering the spleen are cleared rapidly from the blood stream in the hypersplenic state.

PAIgG is a class of platelet autoantibodies bound to the platelet surface glycoprotein and mainly produced by the spleen. PAIgG-bound platelets were easily captured and phagocytized by Macrophages while flowing through the spleen with mediation by antibodies. In addition, PAIgG can also bind to and destroy megakaryocytes and their precursors, thus inhibiting their differentiation and platelet formation.

ACUTE SPLENIC SEQUESTERATION CRISIS

Acute splenic sequesteration crisis is more common in patients with sickle cell disease and hereditary hemolytic anaemia. It is characterized by sudden enlargement of spleen due to trapping of significant proportion of blood volume, followed by rapid drop of hematocrit and resultant hypovolemia and thrombocytopenia. Infection and high altitude are pre disposing factors. It is very rare complication in adults.

SPLENIC RUPTURE

Splenic rupture is the most feared complication of splenomegaly. Pregnant patients with Splenomegaly are more prone to develop rupture following blunt abdominal trauma or low thoracic trauma. Patients are advised to avoid high-impact or contact sports to minimize this risk. Spontaneous splenic rupture have also been reported during pregnancy in literature.

FOETAL COMPLICATIONS

Foetal complications associated with maternal splenomegaly were less studied in literature. Few of the complications mentioned in literature with strong association are

- Increased risk of abortions (15- 30%)
- Prematurity (25%)
- Meconium stained liquor with fetal distress
- Still births
- Intrauterine growth restriction - due to chronic maternal anaemia and restricted peritoneal space for uterine enlargement

OBSTRETIC COMPLICATIONS

Approach to pregnant patients with splenomegaly during labour is still considered as a potential obstetric issue and information regarding proper management of all stages of labour in pregnant

female with splenomegaly is still lacking in literature. Best mode of delivery for these patients is not established with certainity. Risk of variceal bleeding are more with normal vaginal delivery with high grade varices. Also, profuse bleeding from abdominal wall collaterals can also be faced when going with the option of deliver through lower segment cesarean section. Vaginal deliver with epidural analgesia and second stage cut short by forceps or ventouse can be considered as a better safer option of delivery. However , obstetric complications such as Atonic Postpartum hemorrhage, Disseminated intravascular coagulation are more common with splenomegaly mothers. Hence, mode of delivery should be chosen according to individual clinical condition of the patient.

INVESTIGATIONS

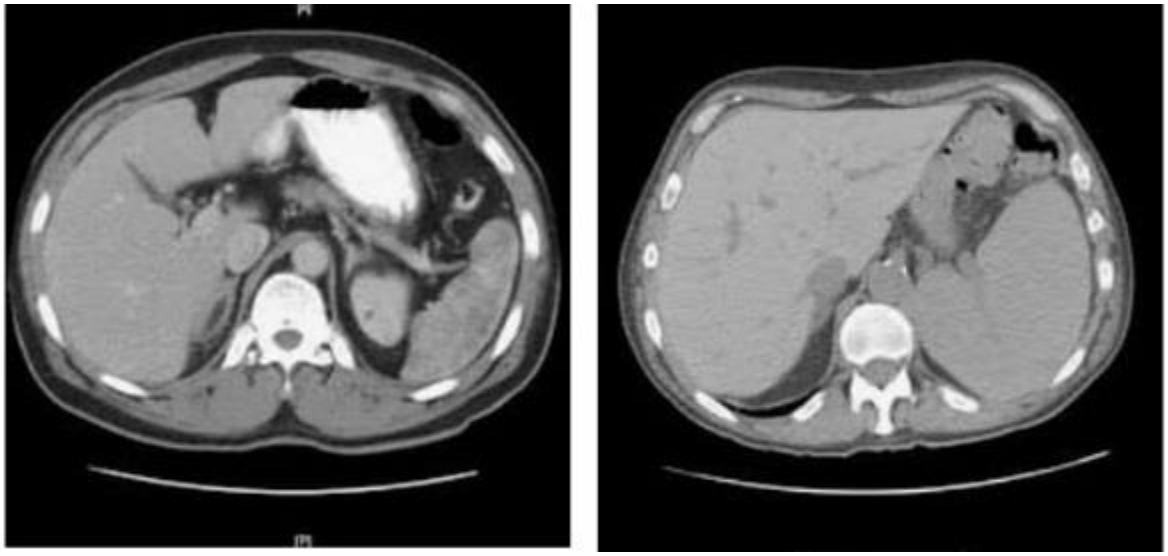
A combination of blood investigations and imaging studies may definitively diagnose splenomegaly and the underlying cause.

BLOOD INVESTIGATIONS

Derangement in the complete blood (cell) counts and morphology including WBC, RBC, and platelets will vary based on the underlying disease state. Anaemia with peripheral smear findings spherocytosis, sickle cells, elliptocytosis, electrophoretic band showing Hb SC would indicate the splenomegaly may be due to hereditary hemoglobinopathies. Anemia with high LDH and bilirubin may indicate a hemolytic etiology or may be due to liver disease. Abnormalities in liver function tests, coagulation profile will confirm the etiology as an associated liver disease with portal hypertension. Peripheral smear for malarial parasites should carry importance particularly in endemic areas.Abnormal lipase, rheumatologic panels, direct antiglobulin test , urine for bence jones protein and disease-specific infectious testing also aids in the diagnosis of causative disease. Abnormal leukocytosis may point to myeloproliferative disorder and warrants further

bone marrow aspiration and biopsy studies. Bone marrow studies also helps us to differentiate pancytopenia due to primary myelofibrosis and hypersplenism. Fluorescence in situ hybridization or polymerase chain reaction should be done for further evaluation of myeloproliferative disorders.Hypersplenism shows characteristic leukopenia, anemia, and thrombocytopenia(pancytopenia) in complete blood count analysis.

IMAGING



Imaging may be used to diagnose splenomegaly and elucidate its underlying cause. Also it is confirmed and quantified using imaging studies. USG is a noninvasive, highly sensitive and specific imaging technique for evaluation of splenic size. Ultrasound is a useful imaging modality in measuring the spleen and spares the patient radiation from CT imaging. Normal spleen size measured via ultrasound is less than 13 cm superior to the inferior axis, 6 cm to 7 cm in medial to lateral axis and 5 cm to 6 cm in anterior to the posterior plane. The spleen has a similar attenuation as the liver when measured on CT imaging. In addition to diagnosing splenomegaly (a splenic measurement of greater than 10 cm in craniocaudal length), abdominal CT may detect splenic abscess, hilar lymph nodes, accessory spleens, mass lesions, vascular abnormalities, cysts, inflammatory changes, traumatic injury, intra-abdominal lymphadenopathy,

or liver abnormalities. Also, CT remains most useful investigation to measure splenic volume. Angiographic findings are used to differentiate splenic cysts from tumours. Splenoportography helps us to diagnose portal vein patency and distribution of collateral vessels.

In specific diseases such as Niemann – Pick disease showing sphingomyelin and cholesterol accumulation within large foamy cells, amyloidosis involving spleen showing large hyaline masses occupying white pulp are specific histologic findings. Two forms of spleen involvement is seen in amyloidosis – "sago spleen" in which amyloid deposits are limited to follicles and " lardaceous spleen" in which amyloid is deposited in walls of splenic sinusoids.

TREATMENT

Peripheral cytopenias caused by hypersplenism often affects prognosis, and should therefore be closely monitored during treatment.

Non-surgical treatment. This category of treatment primarily includes etiological treatment and treatment of concomitant diseases.

Etiological treatment. Hypersplenism has many causes, and treatment should be administered in light of the specific cause. In clinical practice, any deficiencies should be supplemented; for example, transfusion of erythrocytes and platelets should be given for anaemia and thrombocytopenia, respectively, and transfusion of whole blood should be carried out for leukopenia. Kalambokis and Tsianos observed an increased incidence of peripheral cytopenias in patients with liver cirrhosis, which may be related to hypersplenism, and the activation of monocytes and promotion of the release of pro-inflammatory cytokines, such as serum IL-1, leukocyte IL-6, TNF-α, and IFN-γ, by endotoxin produced by intestinal bacteria. In this case, antibiotic therapy should be prescribed for endotoxemia in patients with cirrhosis to increase

blood cell counts. Zuchini et al found that electromagnetic hyperthermia was effective in treating thrombocytopenia in a rat model of cirrhotic hypersplenism. Chernykh et al reported satisfactory outcomes achieved by autologous stem cell transplantation in the treatment of peripheral cytopenias due to cirrhotic Portal hypertension induced hypersplenism.

External irradiation and ablation.

Kenawi et al treated eight patients with liver cirrhosis, splenomegaly and hypersplenism by externally irradiating the spleens using radioactive Co-60. Laboratory data showed that the hemogram returned to completely normal in two patients and partially normal in three patients. Moreover, remission of chronic splenic pain was obtained in all patients and no significant complications were reported (40). Also Low dose radiotherapy has been used in splenomegaly patients with myelofibrosis, CLL as palliative care.Feng et al (42) treated patients with hypersplenism using radiofrequency ablation which gave effective symptom relief while maintaining normal blood Tuftsin levels essential for the anti-infective and antitumor functions of the body.

Partial splenic artery embolization. In 1979, Spigos et al used partial splenic artery embolization for the first time to treat hypersplenic patients with success . Thereafter, it has been applied in the treatment of hypersplenism. This procedure not only increases platelet and leukocyte counts, but also reduces splenic size, improves pancytopenia , and stimulates the immune system . Despite some clinical success in treating splenomegaly and hypersplenism, the indications for partial splenic artery embolization are limited due to serious complications, such as splenic infarction and abscess, which could result in a high risk of death .

Total splenectomy. In secondary hypersplenism, the underlying disease must be treated to prevent further sequestration or destruction of blood cells, and possible spleen enlargement. These therapies should be tested prior to splenectomy removal of the target organ of hypersplenism (48). In severe cases of splenomegaly and hypersplenism, splenectomy is performed to correct the effects of low blood cell and platelet counts . As well, it can effectively improve liver function (51). In particular, laparoscopic splenectomy has several advantages and is superior to partial splenic artery embolization (53). Although splenectomy is commonly used and effective for the treatment of hypersplenism , some risk factors and side effects do exist. For instance, the treatment of all future infections in patients who underwent a complete splenectomy became more complicated, as a key component of the body's normal defense system is not longer present . These individuals will be more susceptible to sepsis and other infections, and should receive, pre-operatively, proper immunization for pneumococcus, influenza, hepatitis, and meningococcemia. For this reason, total splenectomy should be avoided if possible.

Treatment of Postsplenectomy Infection

Fulminant, life-threatening infection represents a major long-term sequela after splenectomy in patients with splenomegaly. Splenic macrophages play a major role in filtering and phagocytizing bacteria and parasitized blood cells from the circulation. In addition, the spleen is a significant source of antibody production.

Overwhelming postsplenectomy infection (OPSI), also known as postsplenectomy sepsis syndrome, begins as a nonspecific, flulike prodrome that is followed by a rapid evolution to full-blown bacteremic septic shock—accompanied by hypotension, anuria, and clinical evidence of disseminated intravascular coagulation—making OPSI as a medical emergency. Despite

appropriate antibiotics and intensive therapeutic intervention, the overall mortality rate in published studies of established cases of OPSI ranges from 50-70%. However, if early intervention was made, the mortality rate may be reduced to approximately 10%. Mortality due to OPSI was claimed to be very high during the first 48 hours of hospital admission. The precise incidence of OPSI remains controversial. Overall, the most reliable data related to incidence estimate approximately 1 case occurring per 500 person-years of observation. Asplenic children younger than 5 years, especially infants splenectomized for trauma, may have an infection rate of greater than 10%.

Most instances of serious infection are due to encapsulated bacteria, such as pneumococci (eg, Streptococcus pneumoniae). Because these organisms are encapsulated and the spleen is integral in the removal of opsonized bacteria, affected patients are at increased risk for sepsis. Pneumococcal infections account for 50-90% of cases of OPSI and accounts to the mortality rate of up to 60%. Hemophilus influenza type B, meningococci, and group A streptococci are the other common organisms accounting for 25% of infection among OPSI patients.

Splenectomy performed for a hematologic disorder, such as thalassemia, hereditary spherocytosis, or lymphoma, appears to carry a higher risk than splenectomy performed as a result of trauma. A major contributing factor is the frequent existence of splenic implants or accessory spleens in traumatized patients, although accessory spleens can also be seen as a developmental anomaly.

Prevention of OPSI

Preventative strategies for OPSI fall into 3 major categories: education, immunoprophylaxis, and chemoprophylaxis.

Vaccination is appropriate in the prevention of OPSI. This has best been defined for S pneumoniae. Unfortunately, the most virulent pneumococcal serotypes tend to be the least immunogenic, and evidence indicates that the efficacy of the vaccine is poorest in younger patients, who would be at higher risk. However, under ideal conditions in a healthy, immunocompetent host, the vaccine offers a 70% protection rate. The pneumococcal vaccine should be administered at least 2 weeks before an elective splenectomy. If the time frame is not practical, the patient should be immunized as soon as possible after recovery and before discharge from the hospital or, at the latest, 24 hours following the procedure.

Most authorities recommend antibiotic prophylaxis for asplenic children, especially for the first 2 years after splenectomy. Some investigators advocate continuing chemoprophylaxis in children for at least 5 years or until age 21 years. However, the value of this approach in older children or adults has never been adequately evaluated in a clinical trial.

AIMS AND OBJECTIVES OF THE STUDY

- **To study the mode of delivery and maternal complications like**
  - ❖ Thrombocytopenia
  - ❖ preterm labour
  - ❖ Postpartum hemorrhage
  - ❖ High dependency unit admission
  - ❖ Portal hypertension
- **Fetal complications like**
  - ❖ IUGR
  - ❖ Low birth weight
  - ❖ Meconium stained liquor
  - ❖ Respiratory distress syndrome,
  - ❖ Hyperbilirubinemia
  - ❖ SNN Admissions

MATERIALS AND METHODS

SOURCE OF THE DATA: Pregnant women those admitted in antenatal ward diagnosed with splenomegaly by ultrasonography from the period of September 2019 to September 2021 in Tirunelveli Medical College Hospital are included in the study.

STUDY DESIGN AND SAMPLING

Cohort study of antenatal mother with splenomegaly included in inclusion criteria for the period of 2 years.

METHOD OF COLLECTION OF DATA

- Ultrasonography findings
- Diagnosis mainly by the
- Symptomatology
- Basic blood investigations

INCLUSION CRITERIA

- Antenatal women diagnosed with splenomegaly either during pregnancy or even prior to pregnancy
- Singleton pregnancy
- Splenomegaly ≥13cm

EXCLUSION CRITERIA

- Anaemia
- Multiple Pregnancy
- Placenta previa
- Preeclampsia
- Abruptio placenta

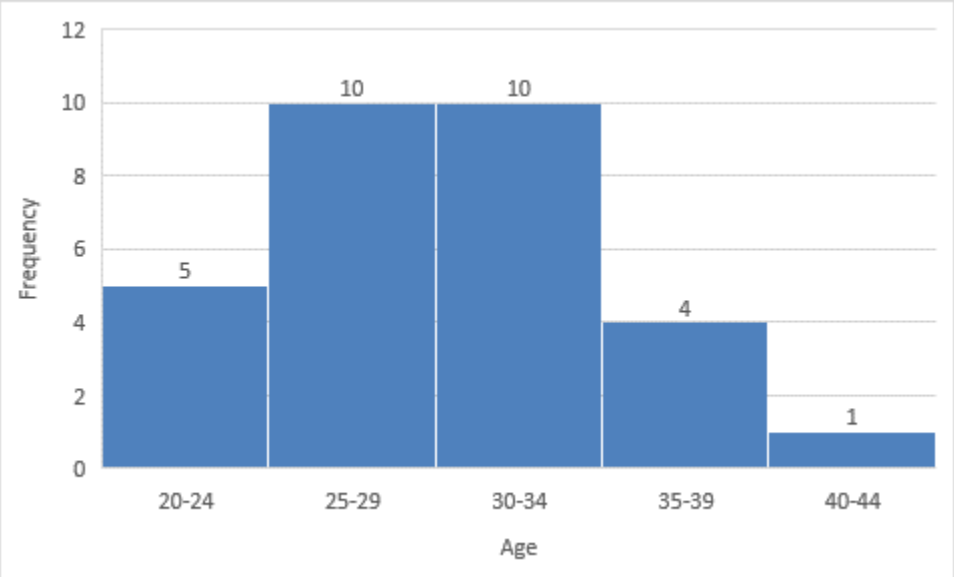
RESULTS

In our study we recruited 30 pregnant patients with splenomegaly and we analysed the demographic profile, characteristic features of splenomegaly and its presentation, etiological profile, associated hematological features with the maternal and fetal outcome.

DEMOGRAPHIC PROFILE

I AGE DISTRIBUTION

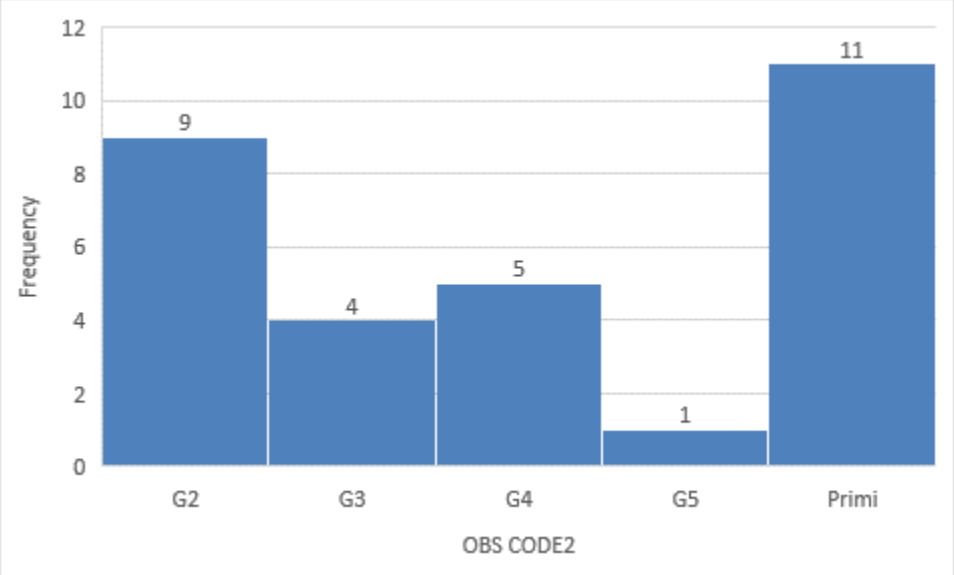
Age	Frequency	Percentage
20-24	5	16.67
25-29	10	33.33
30-34	10	33.33
35-39	4	13.33
40-44	1	3.33
Grand Total	**30**	100.00

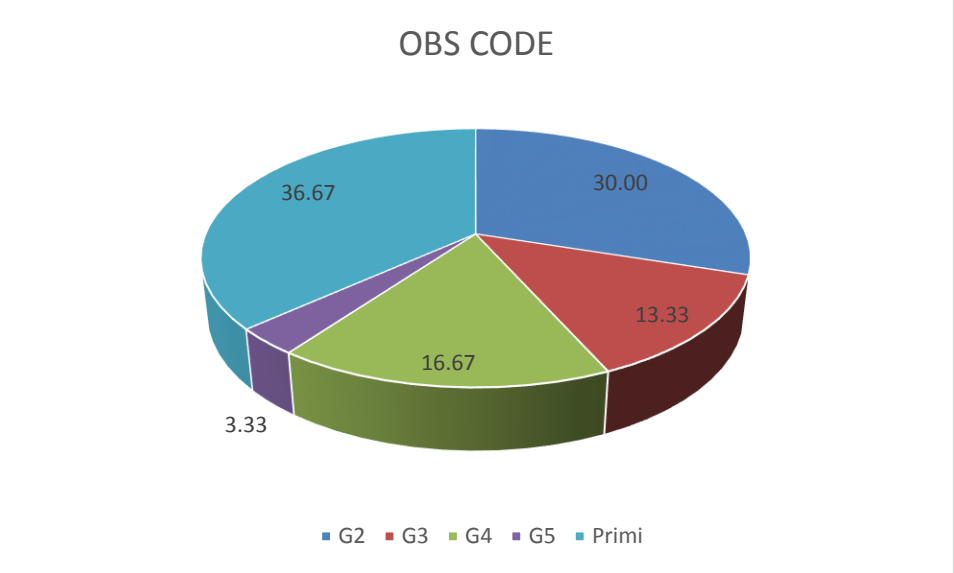


Among our study group, most common age group is 25-39years of age. Least common is above 40 years of age.

II DISTRIBUTION OF GRAVIDA AMONG OUR STUDY GROUP

OBS CODE	Frequency	Percentage
Primi	11	36.67
G2	9	30.00
G3	4	13.33
G4	5	16.67
G5	1	3.33
Grand Total	**30**	100.00

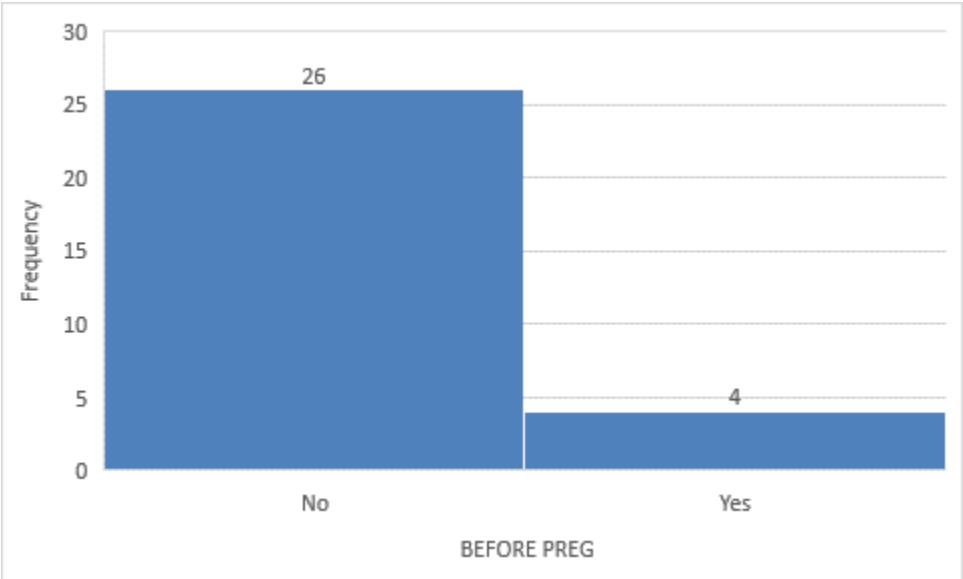




Among our study population, maximum number of patients were primi (36.67%) and minimum number of patients were multi gravida (3.33%)

III DISTRIBUTION OF PARITY AMONG OUR STUDY GROUP

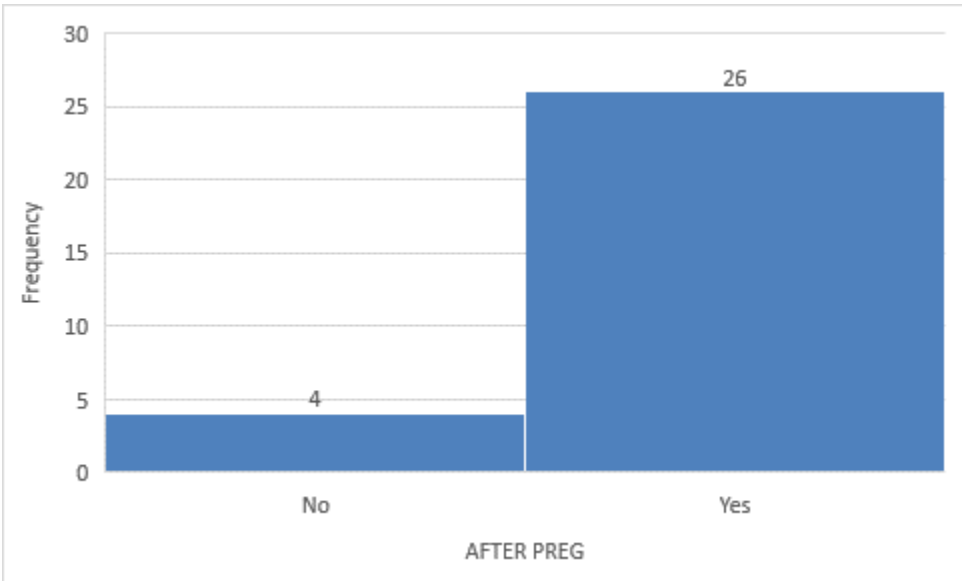
DIAGNOSIS BEFORE PREGNANCY	Frequency	Percentage
No	26	86.67
Yes	4	13.33
Grand Total	**30**	100.00



Among our study, 86.67% did not have splenomegaly previous to pregnancy.

IV DISTRIBUTION OF SPLENOMEGALY AFTER PREGNANCY

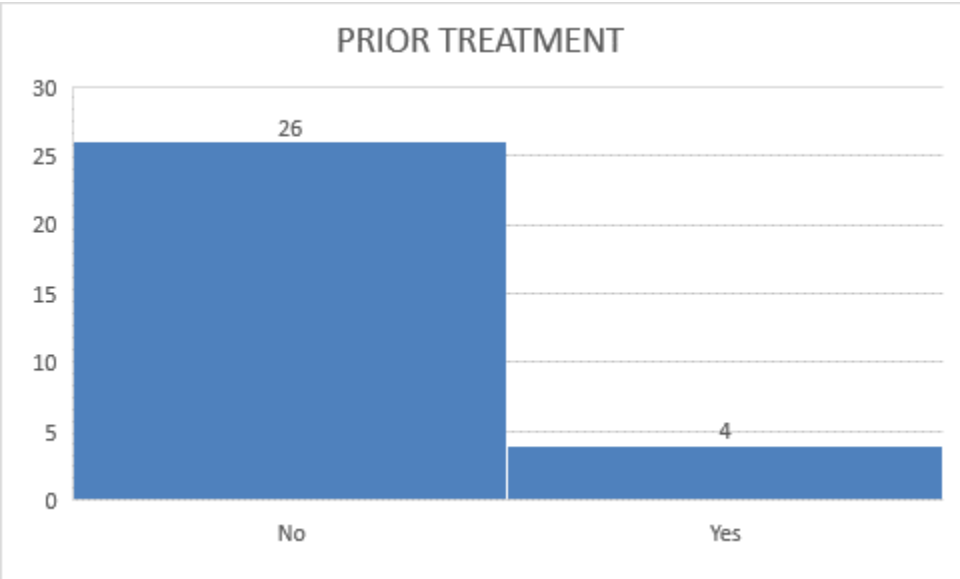
DIAGNOSIS AFTER PREGNANCY	Frequency	Percentage
No	4	13.33
Yes	26	86.67
Grand Total	**30**	100.00



Among our study group, 86.87% had persistence of splenomegaly after pregnancy.

V DISTRIBUTION OF PRIOR TREATMENT AMONG OUR STUDY GROUP

PRIOR TREATMENT	Frequency	Percentage
No	26	90.00
Yes	4	10.00
Grand Total	**30**	100.00

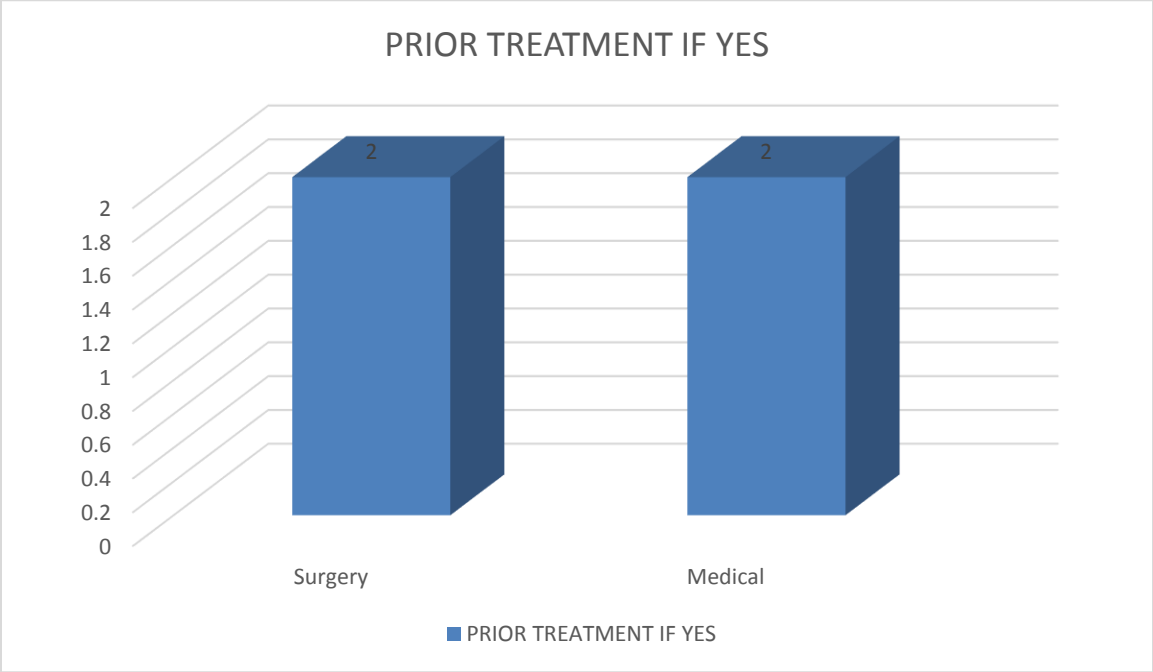


Among our study group, prior treatment was initiated in only 10% of population (before pregnancy)

VI DISTRIBUTION OF TYPE OF TREATMENT OUR STUDY GROUP

PRIOR TREATMENT IF YES	Frequency
Surgery	2
Medical	2

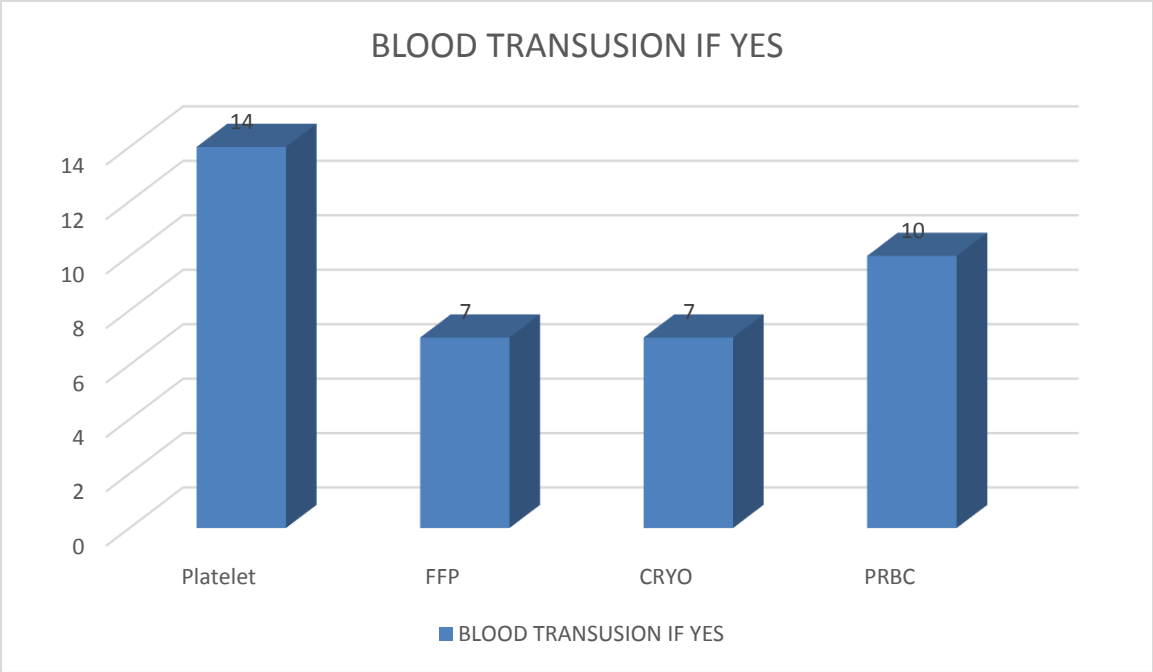
Among our study group, 2 patients got surgical treatment and 2 patients took medical treatment



VII DISTRIBUTION OF TRANSFUSION AMONG OUR STUDY GROUP

BLOOD TRANSUSION IF YES	Frequency
Platelet	14
FFP	7
CRYO	7
PRBC	10
Grand Total	**38**

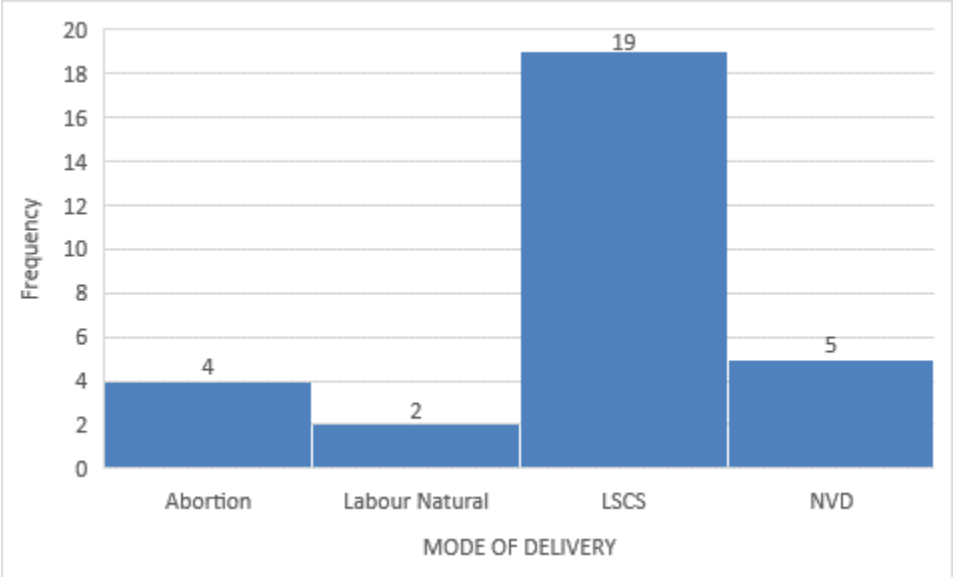
Among our study group, 14 patients received platelet transfusion, 7 patients received fresh frozen plasma and cryoprecipitate transfusion each respectively and 10 patients received packed red blood cell transfusion.



VIII DISTRIBUTION OF MODE OF DELIVERY

MODE OF DELIVERY	Frequency	Percentage
Abortion	4	13.33
Labour Natural	2	6.67
LSCS	19	63.33
NVD	5	16.67
Grand Total	**30**	100.00

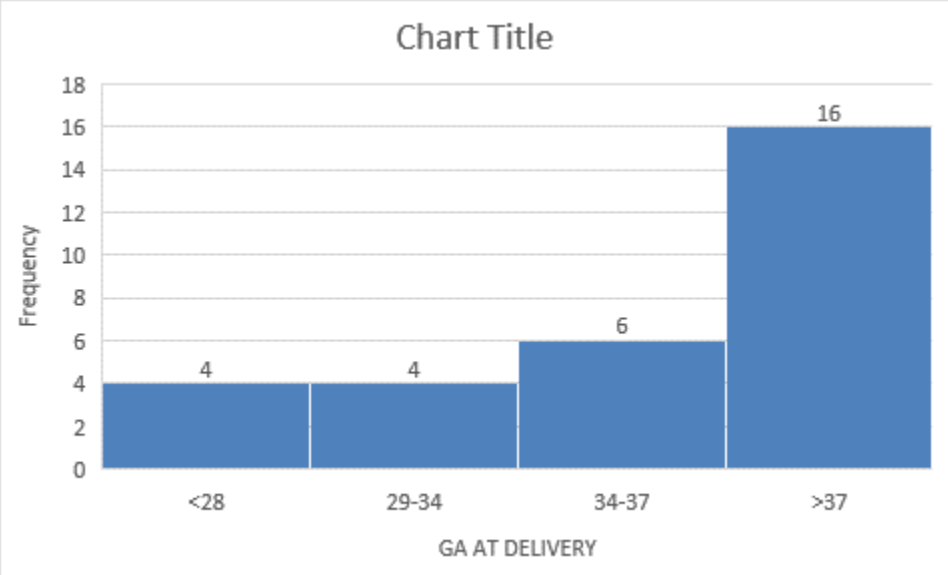
Among our study group, 63.33% of patients delivered by LSCS. Whereas 13.33% of patients got spontaneous abortion. Rest of the patients delivered by normal vaginal delivery.



IX DISTRIBUTION OF GESTATIONAL AGE AT DELIVERY

GA AT DELIVERY	Frequency	Percentage
<28	4	13.33
29-34	4	13.33
34-37	6	20.00
>37	16	53.33
Grand Total	30	100.00

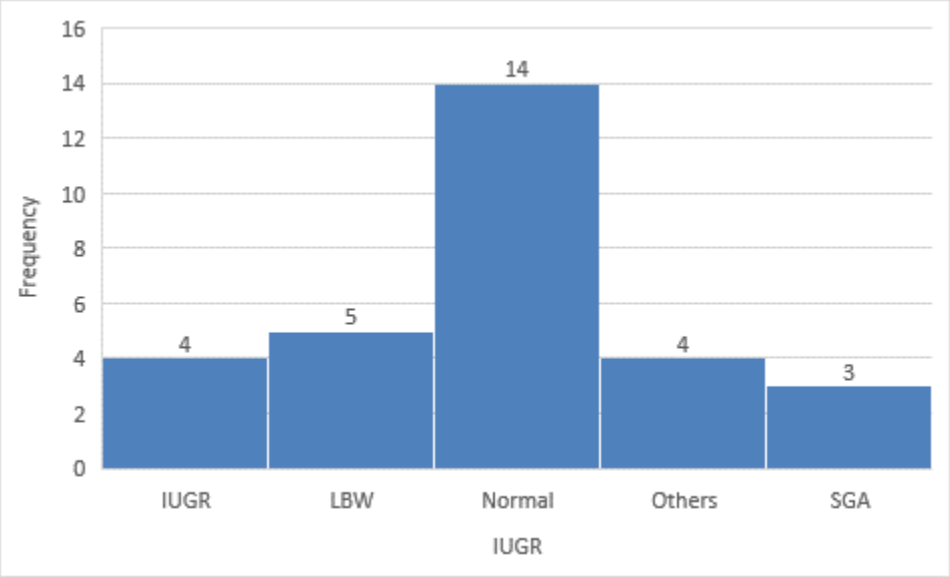
Maximum number (53.33%) delivered above 37 weeks of gestation. Only 13.33% of patients had preterm delivery before 28 weeks of gestation



X DISTRIBUTION OF SNN ADMISSION OF NEONATE

SNN Admission at delivery	Frequency	Percentage
IUGR	4	13.33
LBW	6	16.67
SGA	3	10.00
Normal	13	46.67
Others	4	13.33
Grand Total	**30**	100.00

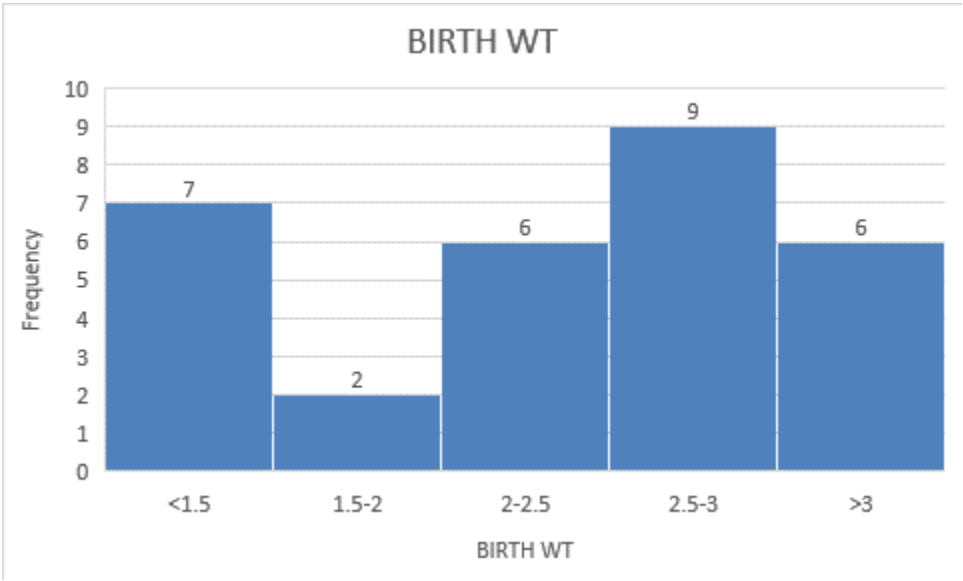
46.67% of neonates born to mother with splenomegaly were normal 16.67% of neonates were low birth weight babies and 13.33% of babies had intrauterine growth restriction and got admitted in SNN post delivery period.



XI DISTRIBUTION OF BIRTH WEIGHT

BIRTH WT	Frequency	Percentage
<1.5	7	23.33
1.5-2	2	6.67
2-2.5	6	20.00
2.5-3	9	30.00
>3	6	20.00
Grand Total	**30**	100

Among our study group, 23.33% of neonates were very low birth weight < 1.5 kg, 50% of neonates were in normal weight, 30% in 2.5-3 kg and > 3 kg contributes to 20% of neonates.



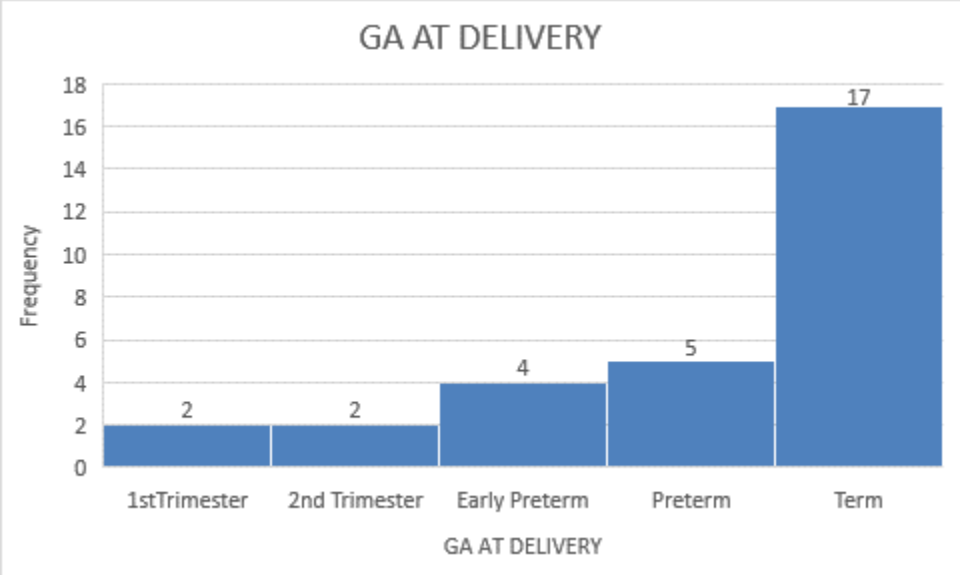
XII DISTRIBUTION OF GESTATIONAL AGE AT DELIVERY

GA AT DELIVERY	Frequency	Percentage
1stTrimester	2	6.67
2nd Trimester	2	6.67
Early Preterm	4	13.33
Preterm	5	16.67
Term	17	56.67
Grand Total	**30**	100.00

Term delivery occurred in 56.67% of mothers with splenomegaly

Preterm delivery happened in 16.67% and early preterm delivery contributes to 13.33% of our study population

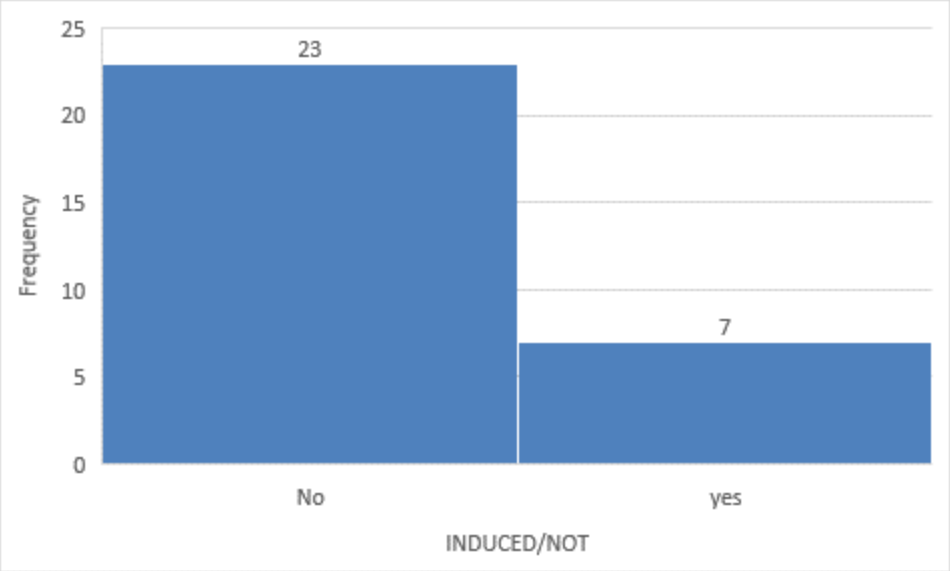
1st trimester and 2nd trimester abortion contribute to 6.67% of study population each respectively.



XIII DISTRIBUTION OF INDUCED DELIVERY

LABOUR INDUCED/NOT	Frequency	Percentage
No	23	76.67
yes	7	23.33
Grand Total	**30**	100.00

23.33% of patients had termination of pregnancy by induced labour in our study group.

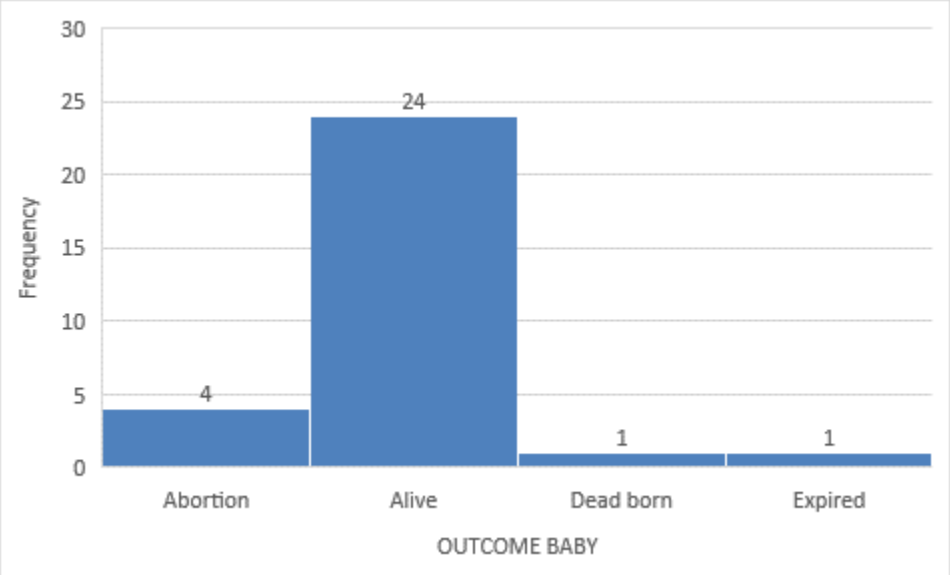


XIV DISTRIBUTION OF OUTCOME OF BABY

OUTCOME OF BABY	Frequency	Percentage
Abortion	4	13.33
Alive	24	80.00
Dead born	1	3.33
Expired	1	3.33
Grand Total	**30**	100.00

Among our study group, 80% of babies were alive, where 6.66% of babies were dead born /expired.

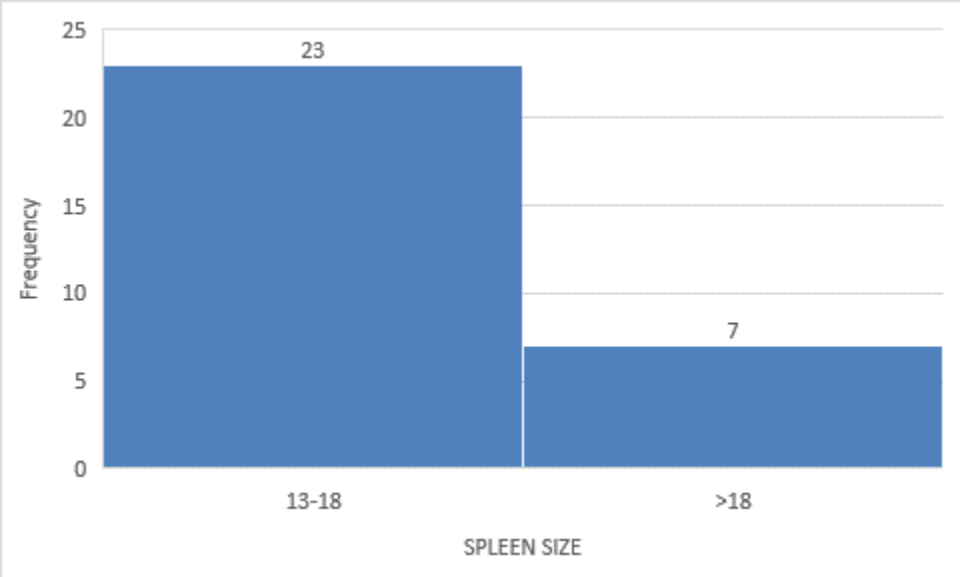
13.33% of pregnancy outcome is abortion in our study group.

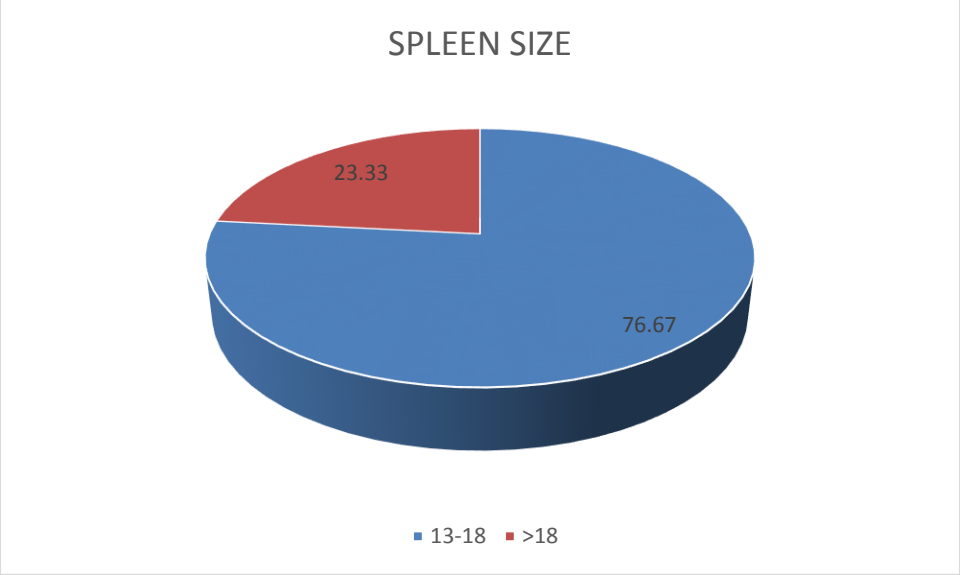


XV DISTRIBUTION OF SPLEEN SIZE

SPLEEN SIZE(USG)	Frequency	Percentage
13-18	23	76.67
>18	7	23.33
Grand Total	**30**	100.00

76.67% of patients had spleen size in 13-18 cm size and 23.33% had > 18 cm spleen size.

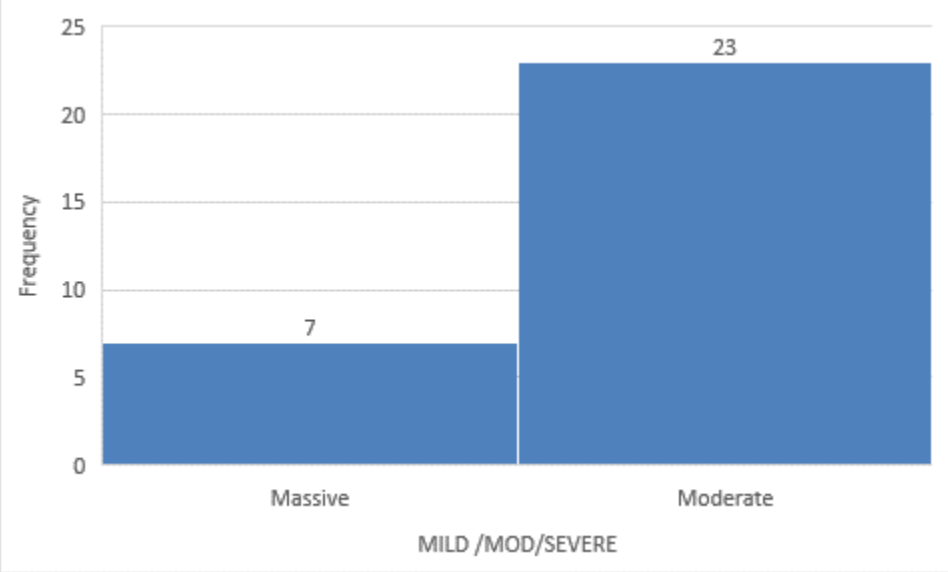


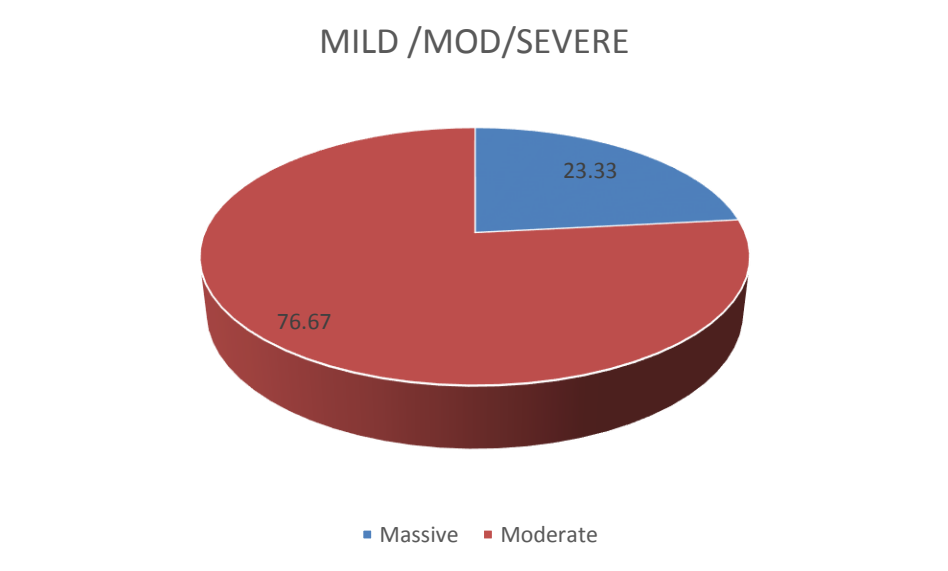


XVI DISTRIBUTION OF GRADING OF SPLENOMEGALY

MODERATE/MASSIVE	Frequency	Percentage
Massive	7	23.33
Moderate	23	76.67
Grand Total	**30**	100.00

76.67% had moderate splenomegaly and 23.33% had massive splenomegaly

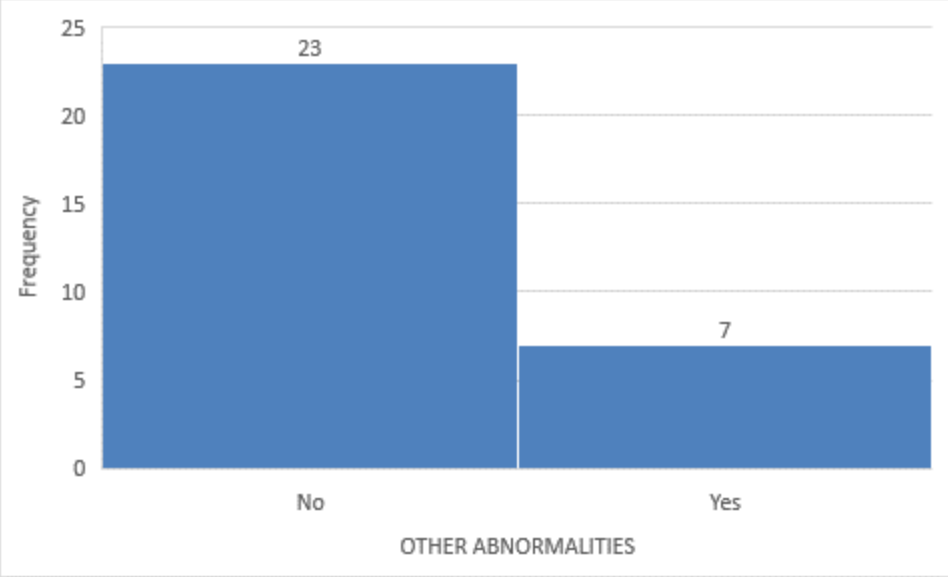


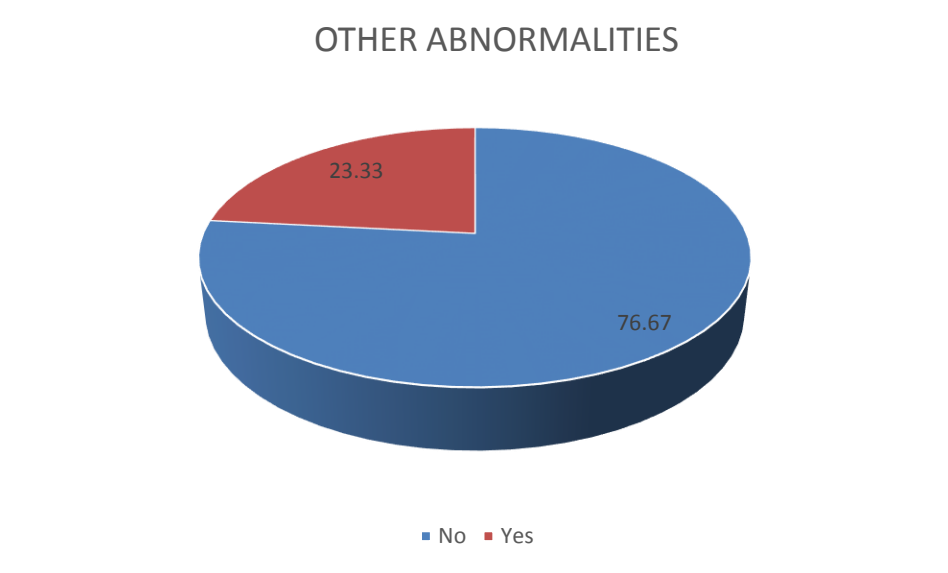


XVII DISTRIBUTION OF OTHER ABNORMALITIES

OTHER ABNORMALITIES	Frequency	Percentage
No	23	76.67
Yes	7	23.33
Grand Total	**30**	100.00

Other abnormalities like hepatomegaly etc.. were found in 23.33% of our study group



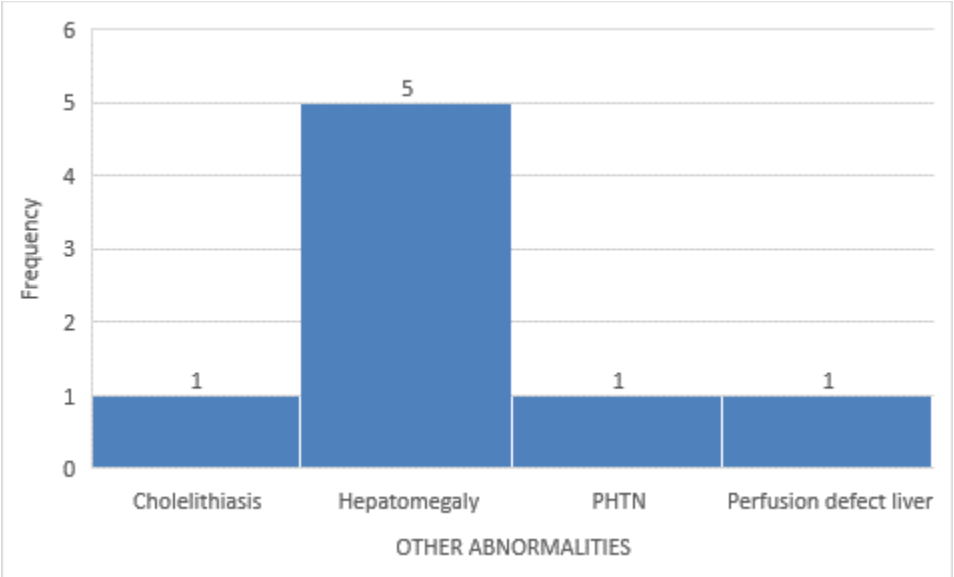


XVIII DISTRIBUTION OF TYPE OF OTHER ABNORMALITIES

OTHER ABNORMALITIES IF YES	Frequency
Cholelithiasis	1
Hepatomegaly	5
PHTN	1
Perfusion defect liver	1

Among our study group, hepatomegaly was more common seen in 5 patients.

Cholelithiasis and pulmonary hypertension was seen in 1 patient each respectively.

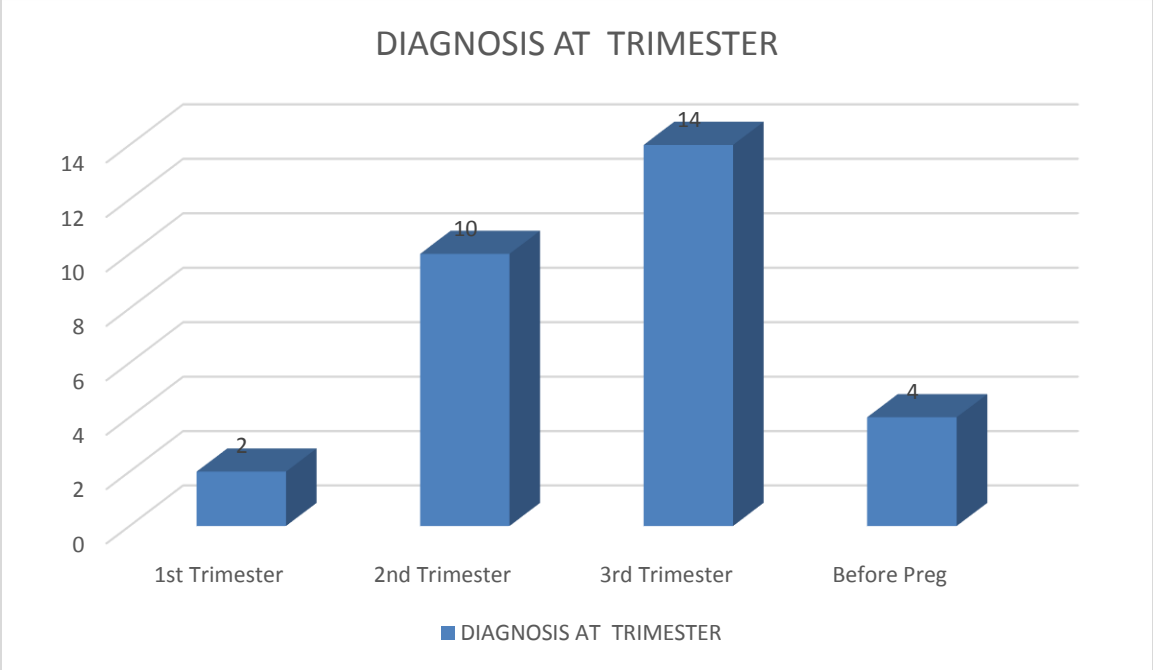


XIX DISTRIBUTION OF TIME OF DIAGNOSIS

DIAGNOSIS AT TRIMESTER	Frequency	Percentage
1st Trimester	2	6.67
2nd Trimester	10	33.33
3rd Trimester	14	46.67
Before Preg	4	13.33
Grand Total	**30**	100.00

Splenomegaly was diagnosed before pregnancy in 13.33% and after pregnancy in 3.33% of population

Diagnosis was made in 1st, 2nd, 3rd trimester in 6.67%, 33.33% and 46.67% of patients respectively.



XX DISTRIBUTION OF PLATELET COUNT

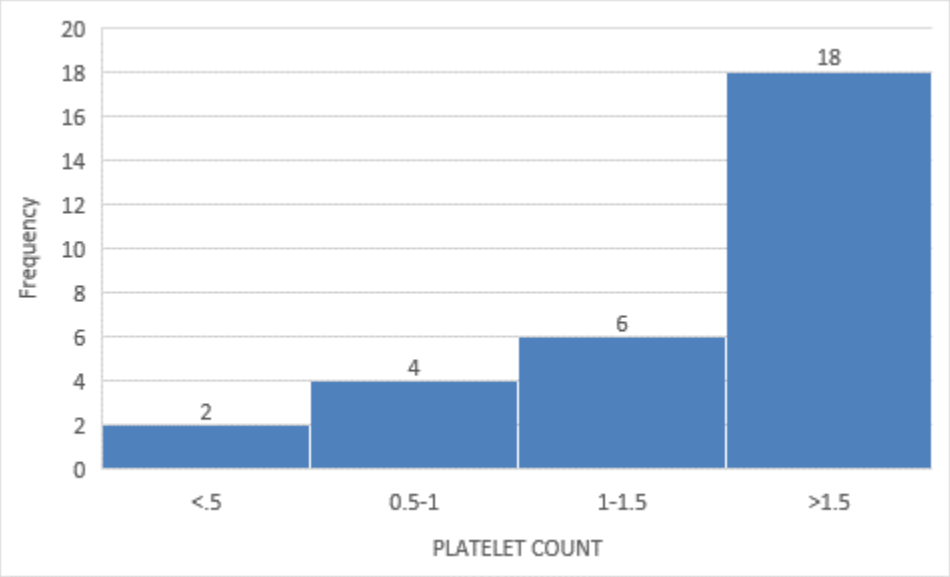
PLATELET COUNT	Frequency	Percentage
<.5	2	6.67
0.5-1	4	13.33
1-1.5	6	20.00
>1.5	18	60.00
Grand Total	30	100.00

Normal platelet count was seen in 60% population

Thrombocytopenia in range of 1-1.5 lakhs was observed in 20% of population

13.33% of population had platelets in range of 50000 – 1 lakhs

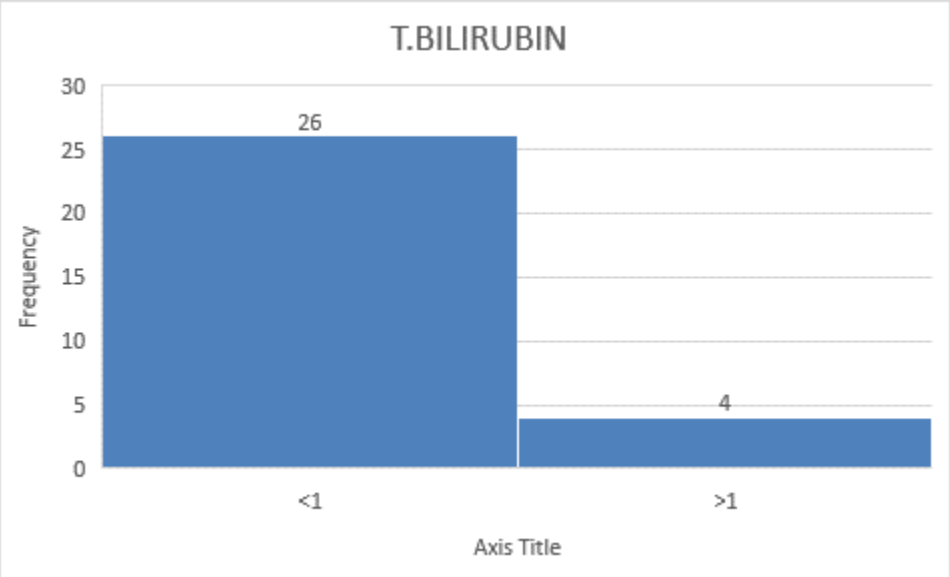
And 6.67% had less than 50000 platelets



XXI DISTRIBUTION OF BILIRUBIN

T.BILIRUBIN	Frequency	Percentage
<1	26	86.67
>1	4	13.33
Grand Total	**30**	100.00

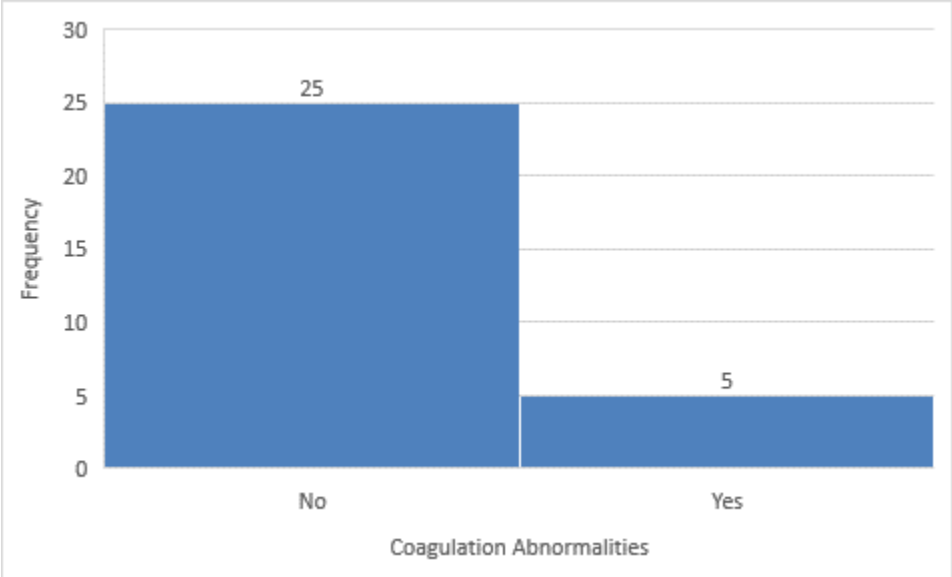
Among our study group, normal bilirubin was seen in 86.67% of patients



XXII DISTRIBUTION OF COAGULATION ABNORMALITIES

Coagulation Abnormalities	Frequency	Percentage
No	25	83.33
Yes	5	16.67
Grand Total	**30**	100.00

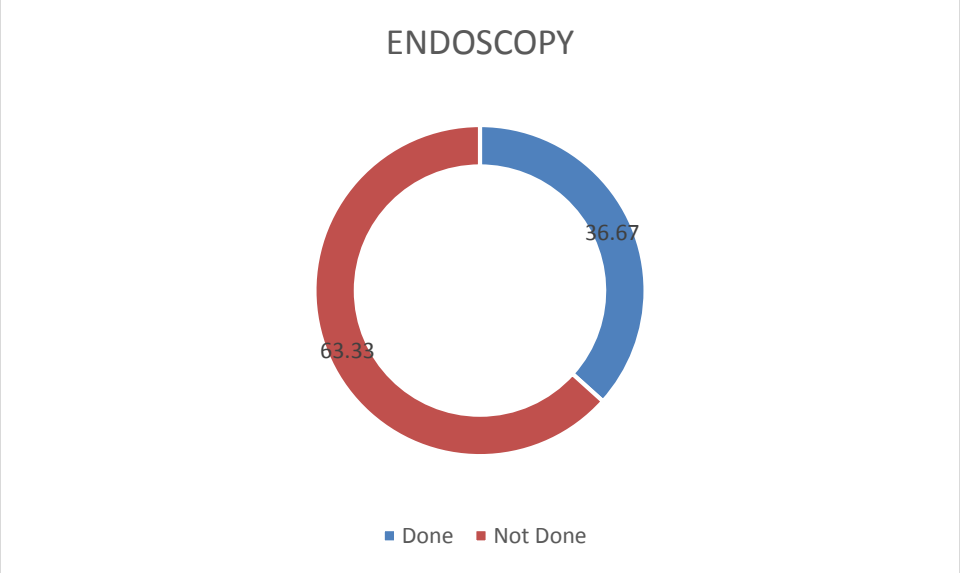
Only 16.67% of population had coagulation abnormalities in our study group.



XXIII DISTRIBUTION OF ENDOSCOPIC FINDINGS

ENDOSCOPY	Frequency	Percentage
Done	11	36.67
Not Done	19	63.33
Grand Total	**30**	100

UGI Endoscopy was done for 36.67% of patients in our study group.



XXII DISTRIBUTION OF ENDOSCOPIC FINDINGS

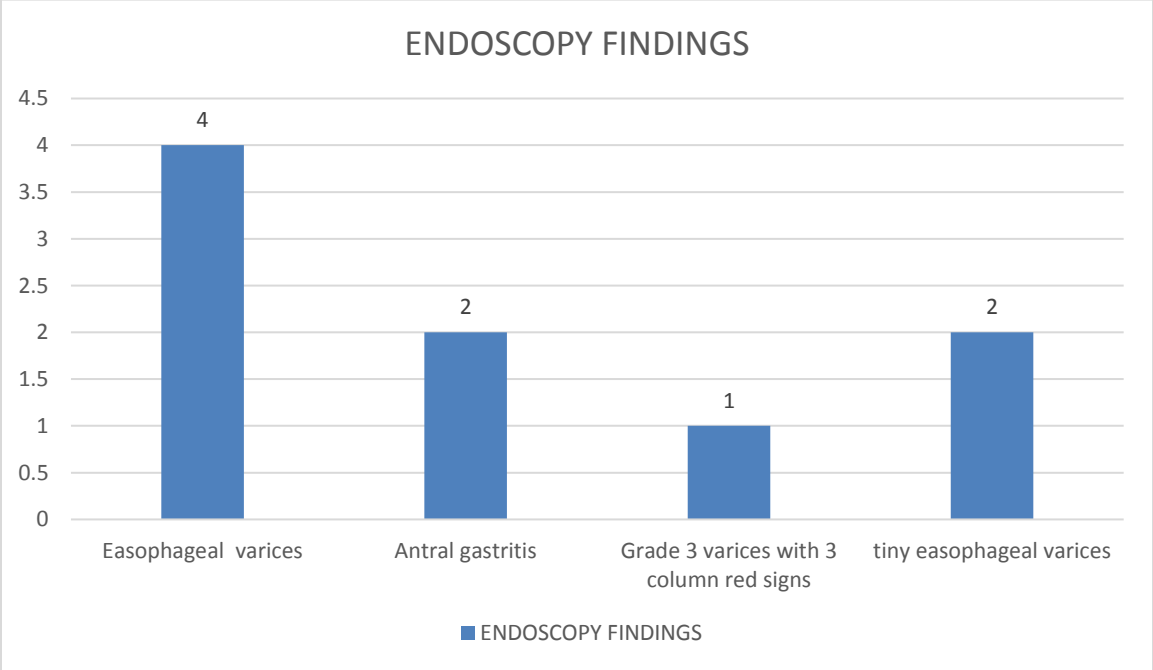
ENDOSCOPY FINDINGS	Frequency
Easophageal varices	4
Antral gastritis	2
Grade 3 varices with 3 column red signs	1
tiny easophageal varices	2

To the maximum, esophageal varices was seen in 4 patients

Antral gastritis was the second common seen in 2 patients

Grade 3 varices with warning sign of 3 column red signs was seen in 1 patient

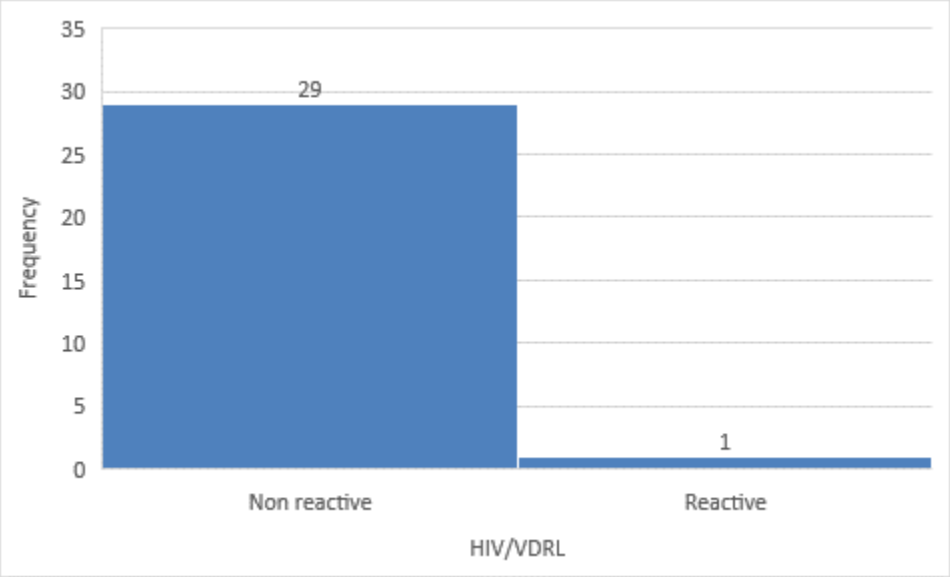
Tiny oesophageal varices was also seen in 1 patient.



XXIII DISTRIBUTION OF HIV/VDRL AMONG OUR STUDY GROUP

HIV/VDRL	Frequency	Percentage
Non reactive	29	96.67
Reactive	1	3.33
Grand Total	**30**	100.00

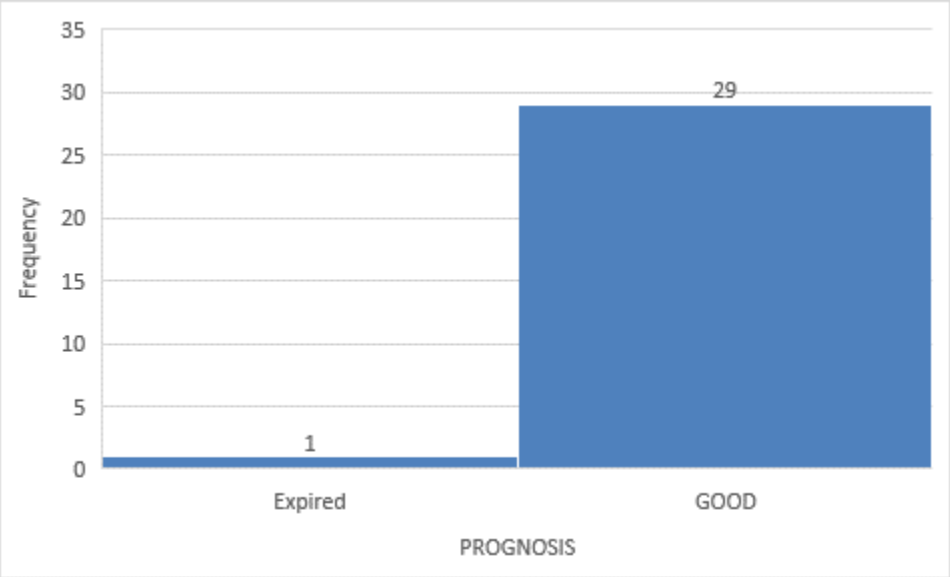
Only 1 patient was HIV positive in our study group.



XXIV DISTRIBUTION OF PROGNOSIS

PROGNOSIS	Frequency	Percentage
Expired	1	3.33
GOOD	29	96.67
Grand Total	**30**	100.00

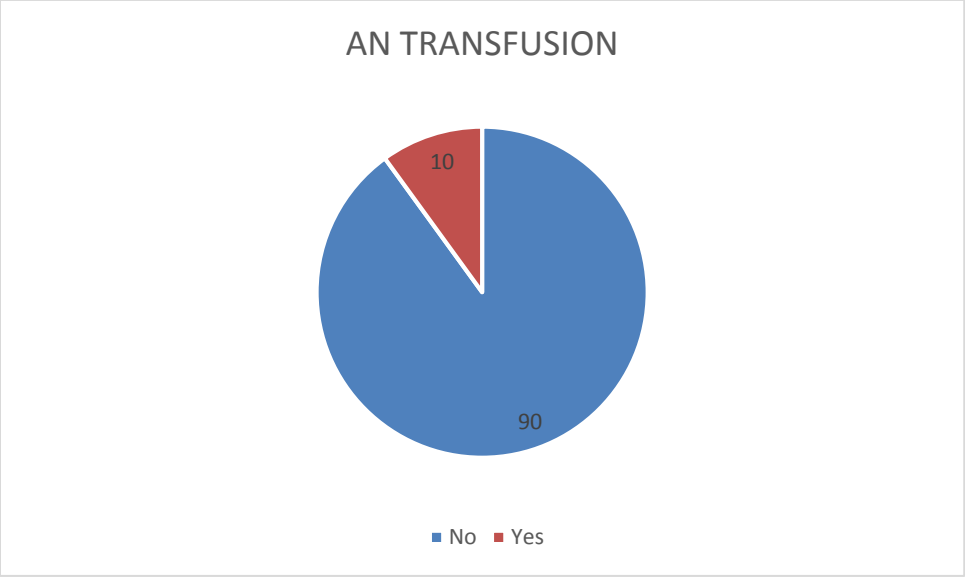
Only 1 patient expired in our study group



XXV DISTRIBUTION OF ANTENATAL TRANSFUSION

AN TRANSFUSION	Frequency	Percentage
No	27	90
Yes	3	10
Grand Total	30	100

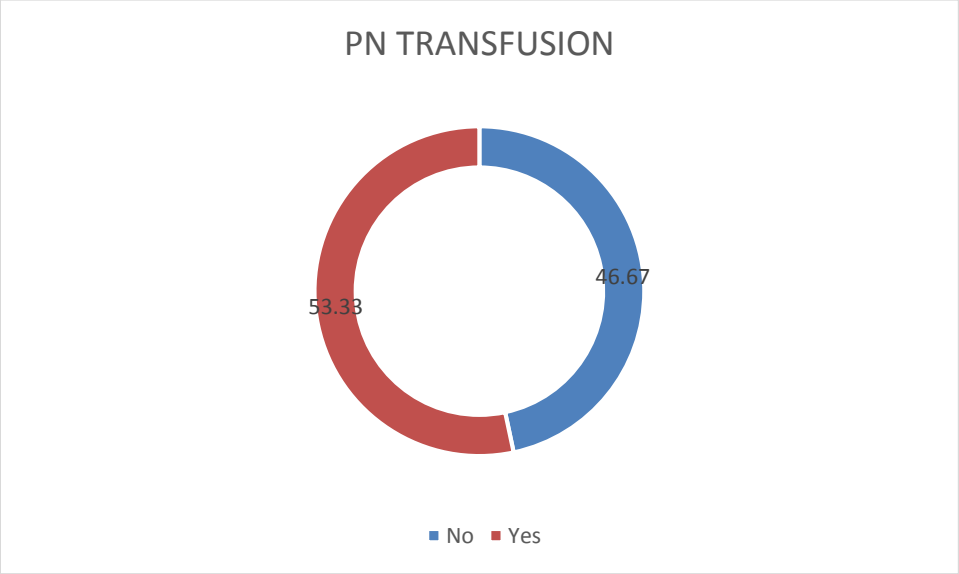
Only 3 patients received (10%) antenatal transfusion in our study group



XXVI DISTRIBUTION OF POSTNATAL TRANSFUSION

PN TRANSFUSION	Frequency	Percentage
No	14	46.67
Yes	16	53.33
Grand Total	30	100

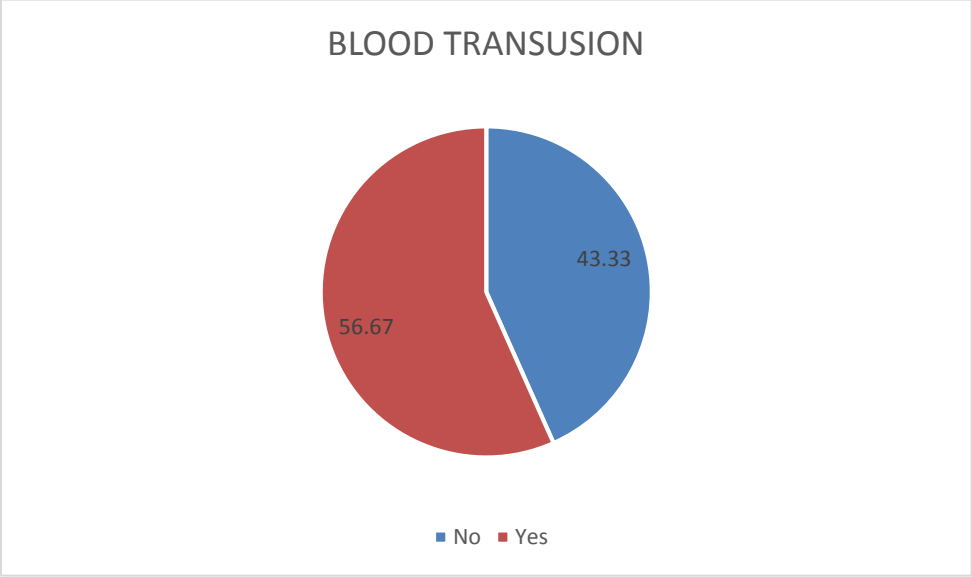
53.33% of patients received postnatal transfusion among our study group



XXVII DISTRIBUTION OF TRANSFUSION AMONG OUR STUDY GROUP

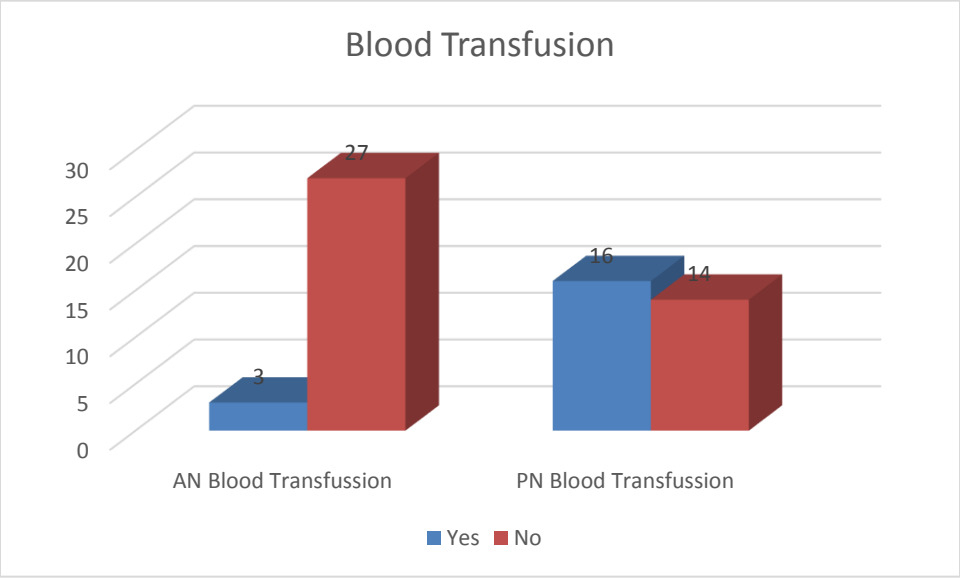
TOTAL TRANSUSION	Frequency	Percentage
No	13	43.33
Yes	17	56.67
Grand Total	30	100

56.67% of patients received transfusion in our study group irrespective of period of transfusion.



XXVIII COMPARISON OF ANTENATAL vs POSTNATAL TRANSFUSION

Blood Transfusion	AN Blood Transfusion	PN Blood Transfusion
Yes	3	16
No	27	14
Grand Total	30	30
Chi-Square = 13.0167		
Degrees of Freedom = 1		
p = 0.0003		



Postnatal transfusion was done more commonly than antenatal transfusion among mothers with splenomegaly. This association carries statistical significance (p value 0.0003)

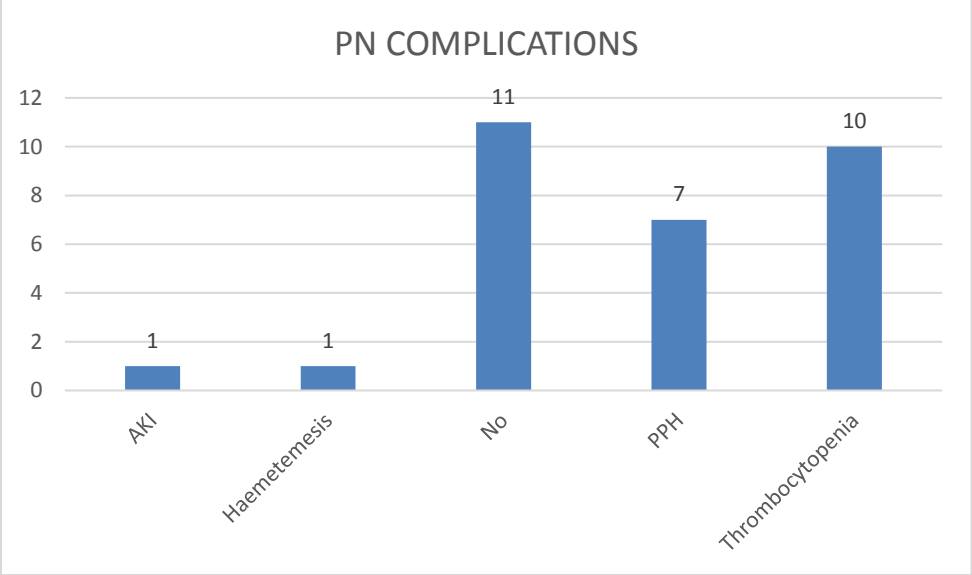
XXIX DISTRIBUTION OF POSTNATAL COMPLICATIONS

PN COMPLICATIONS	Frequency	Percentage
AKI	1	3.33
Haemetemesis	1	3.33
No	11	36.67
PPH	7	23.33
Thrombocytopenia	10	33.33
Grand Total	30	100.00

36.67% of patients does not exhibit any postnatal complications

33.33% of patients had thrombocytopenia as the most common postnatal complication.

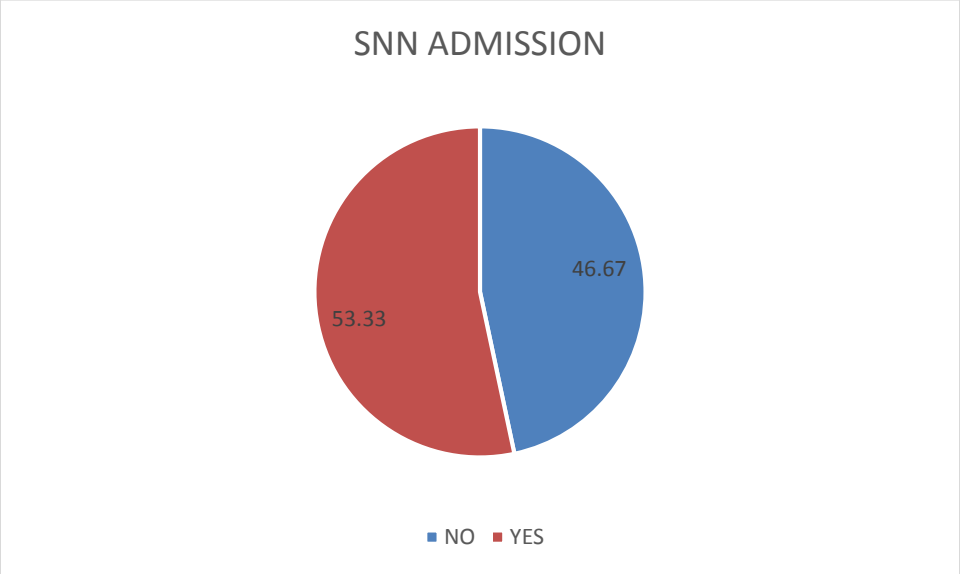
PPH was seen in 23.33% of patients and hematemesis , AKI was observed in 3.33% of patients each respectively.



XXX DISTRIBUTION OF SNN ADMISSION

SNN ADMISSION	Frequency	Percentage
NO	14	46.67
YES	16	53.33
Grand Total	30	100.00

53.33% of babies had SNN admission.



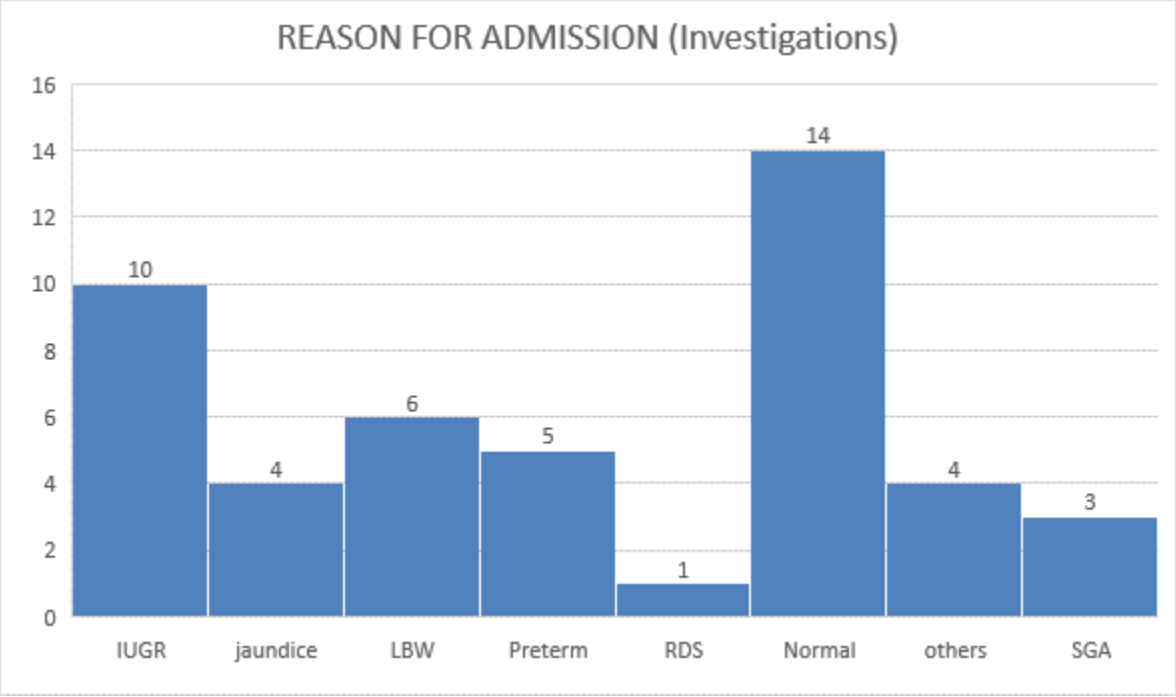
XXXI DISTRIBUTION OF REASON FOR SNN ADMISSION

REASON FOR ADMISSION (Investigations)	Frequency	Percentage
IUGR	5	13.33
jaundice	4	3.33
LBW	6	20
Preterm	5	6.67
RDS	1	10
IUGR	5	16.67
Normal	14	36.67
others	4	13.33
SGA	3	10

Among our study, low birth weight(20%) was the most common reason for SNN admission followed by IUGR (16.67%)

Preterm delivery was the reason for 6.67% of babies and jaundice was observed in 3.33% of babies

RDS was seen in 1 baby alone in our study group.



XXXII DISTRIBUTION OF ANTENATAL RISK FACTOR

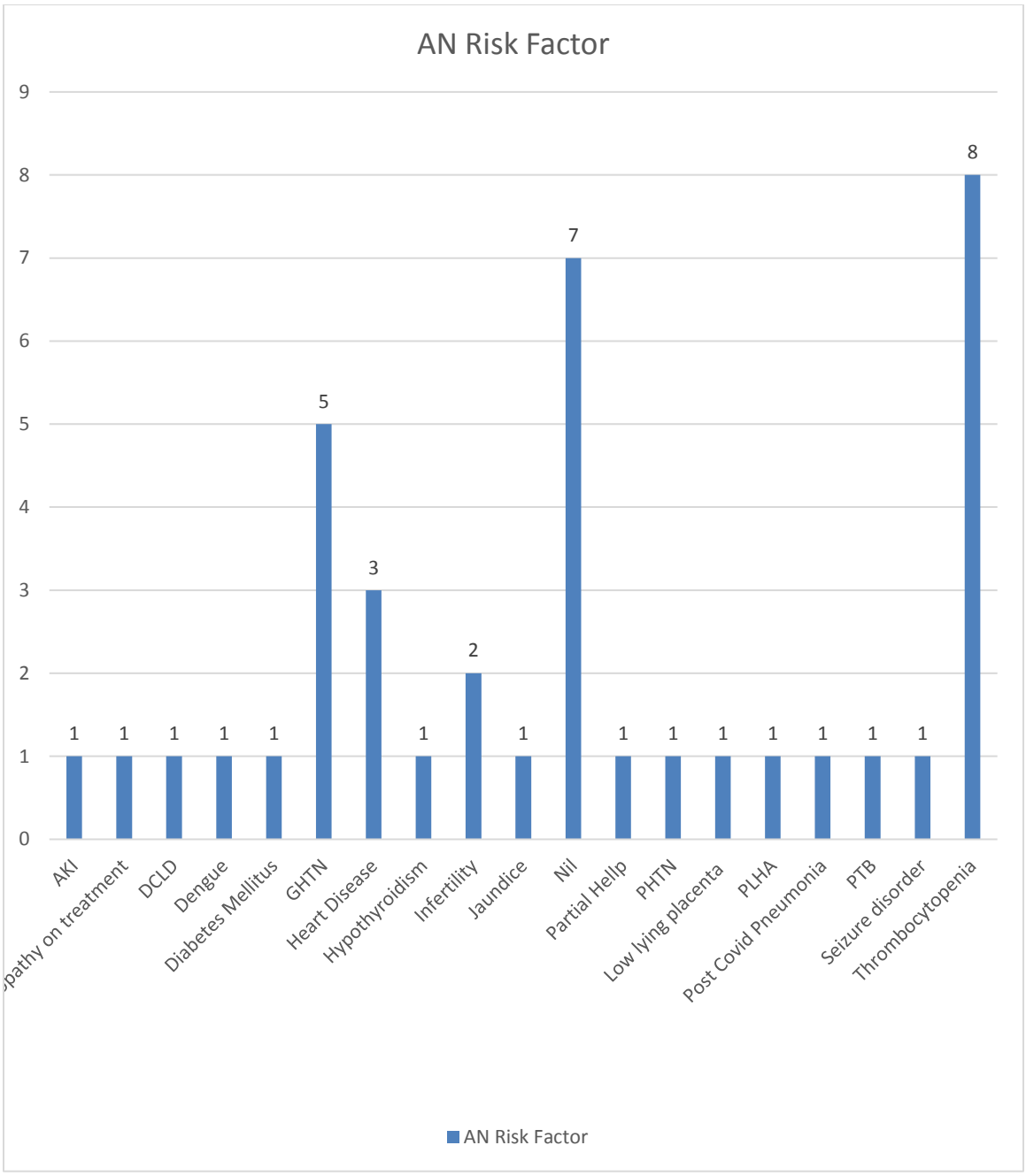
AN Risk Factor	Frequency	Percentage
AKI	1	3.33
Congestive Gastropathy	1	3.33
DCLD	1	3.33
Dengue	1	3.33
Diabetes Mellitus	1	3.33
GHTN	5	16.67
Heart Disease	3	10.00
Hypothyroidism	1	3.33
Infertility	2	6.67
Jaundice	1	3.33
Nil	7	23.33
Partial Hellp	1	3.33
PHTN	1	3.33
Low lying placenta	1	3.33
PLHA	1	3.33
Post Covid Pneumonia	1	3.33
PTB	1	3.33
Seizure disorder	1	3.33
Thrombocytopenia	8	26.67

Gestational hypertension was the most common risk factor seen in 16.67% of patients

Heart disease was the second most common (10%)

No risk factor was observed in 23.33% of patients

Co-existing antenatal thrombocytopenia was seen in 26.67% of patients.


AN Risk Factor
9
8
7
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pathy on treatment
DCLD
Dengue
Diabetes Mellitus
GHTN
Heart Disease
Hypothyroidism
Infertility
Jaundice
Nil
Partial Hellp
PHTN
Low lying placenta
PLHA
Post Covid Pneumonia
PTB
Seizure disorder
Thrombocytopenia
AN Risk Factor

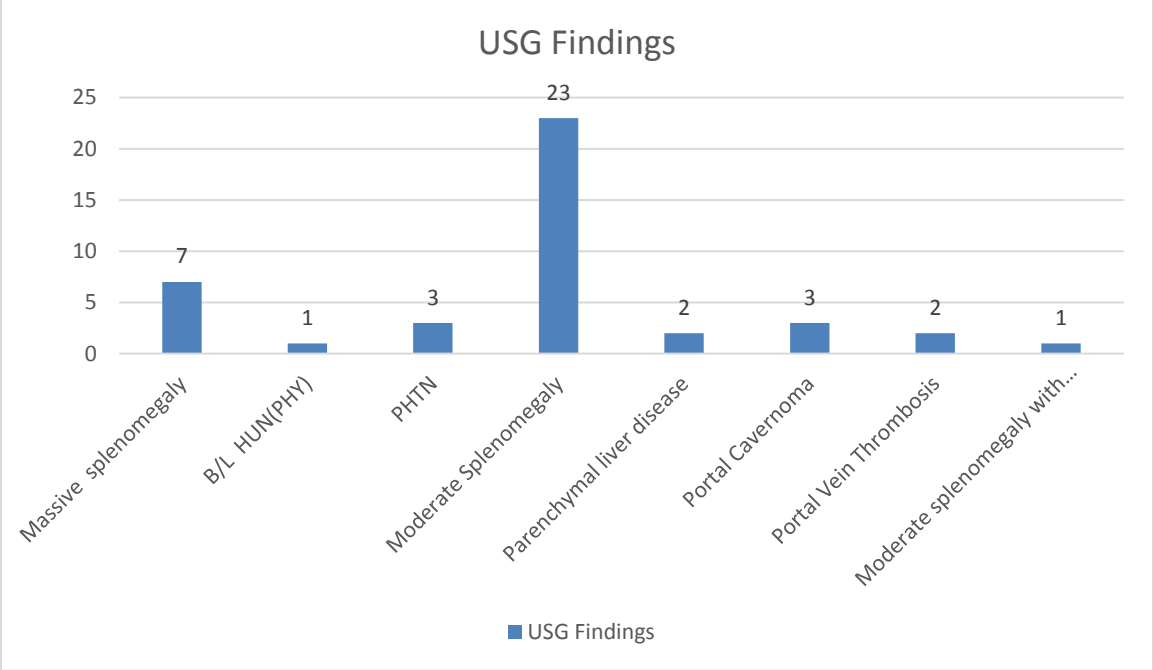
XXXIII DISTRIBUTION OF USG FINDINGS

USG Findings	Frequency	Percentage
Massive splenomegaly	7	23.33
B/L HUN(PHY)	1	3.33
PHTN	3	10.00
Moderate Splenomegaly	23	76.66
Parenchymal liver disease	2	6.67
Portal Cavernoma	3	10.00
Portal Vein Thrombosis	2	6.67
Moderate splenomegaly with caudate lobe hypertrophy	1	3.33

Moderate splenomegaly was the most common USG finding seen in 83.33% of patients.

Massive splenomegaly was seen in 16.67% of patients and portal hypertension was observed in 10% of patients.

Portal vein cavernoma was seen in 10% of patients and parenchymal liver disease was seen in 6.67% of patients.



XXXIV DISTRIBUTION OF PORTAL VEIN DOPPLER FINDINGS

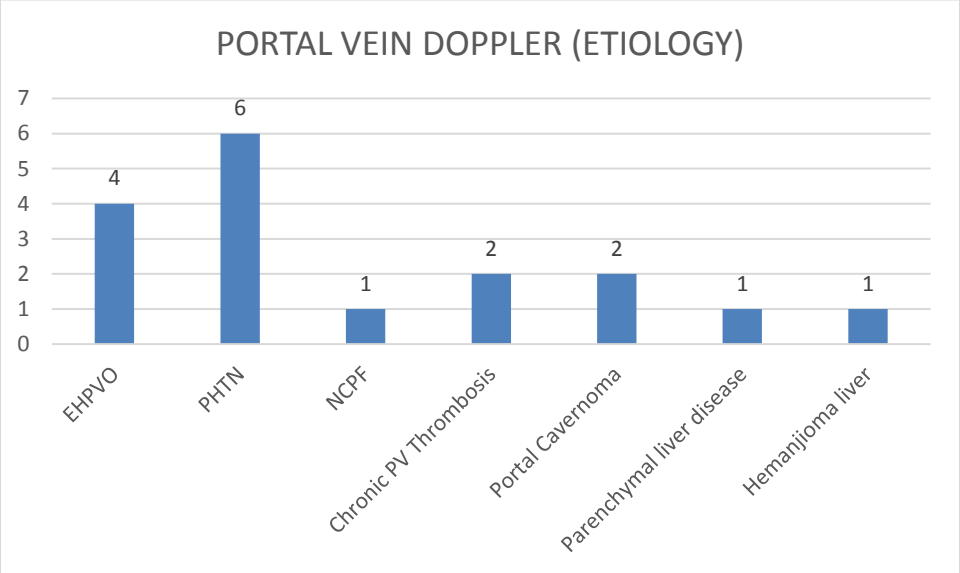
PORTAL VEIN DOPPLER (ETIOLOGY)	Frequency	Percentage
EHPVO	4	13.33
PHTN	6	20.00
NCPF	1	3.33
Chronic PV Thrombosis	2	6.67
Portal Cavernoma	2	6.67
Parenchymal liver disease	1	3.33
Hemanjioma liver	1	3.33

Portal hypertension was the most common finding seen in 20% of patients

Chronic portal vein thrombosis and portal cavernoma was seen in 6.67% of patients each respectively.

Extrahepatic portal vein obstruction was seen in 13.33% of patients

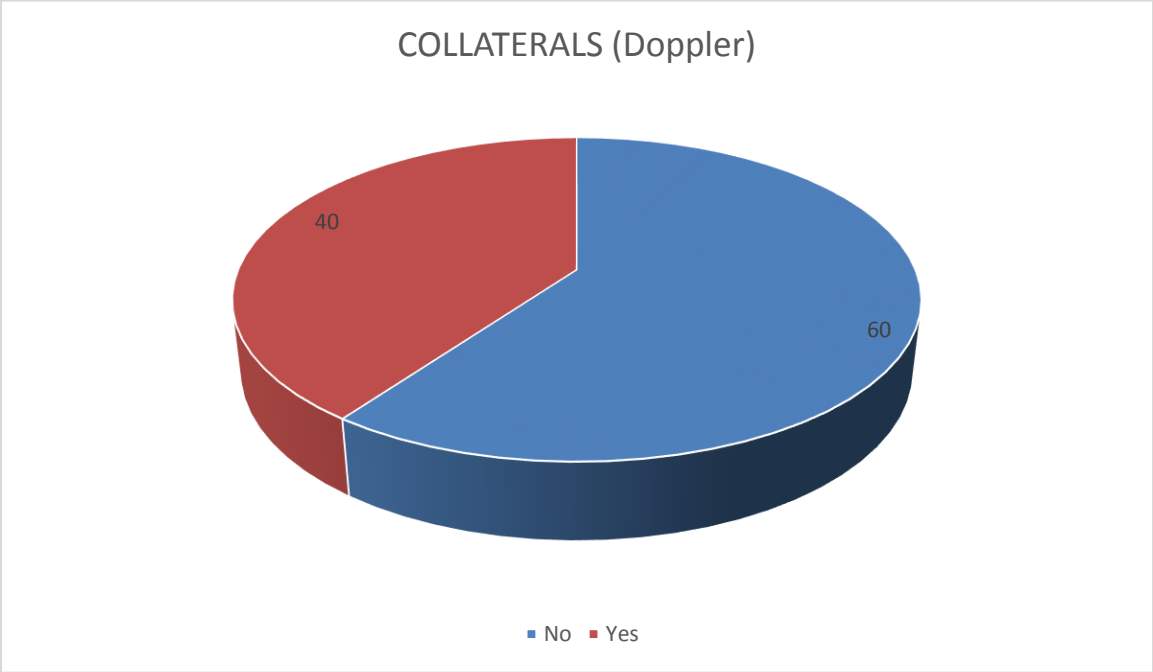
And Non cirrhotic portal fibrosis , parenchymal liver disease and hemangioma liver was seen in 3.33% of patients each respectively.



XXXV DISTRIBUTION OF COLLATERALS IN DOPPLER

COLLATERALS (Doppler)	**Frequency**	**Percentage**
No	18	60.00
Yes	12	40.00
Grand Total	**30**	100.00

Only 36.67% of patients had collaterals in Doppler study

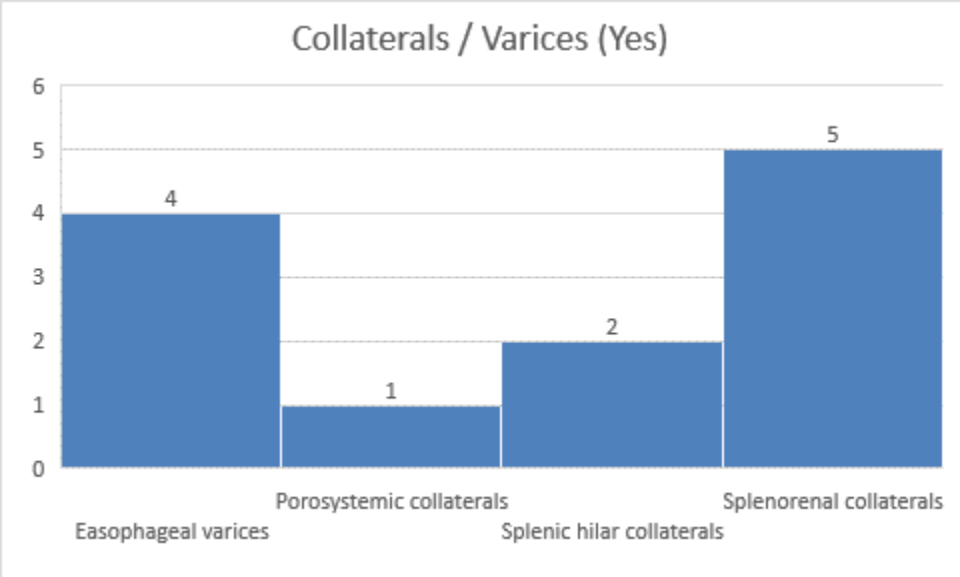


XXXVI DISTRIBUTION OF COLLATERALS AT DIFFERENT SITE

Collaterals / Varices (Yes)	Frequency	Percentage
Easophageal varices	4	13.33
Porosystemic collaterals	1	3.33
Splenic hilar collaterals	2	6.67
Splenorenal collaterals	5	16.67

16.67% of patients were found to have splenorenal collaterals and 13.33% of patients were found to have esophageal varices

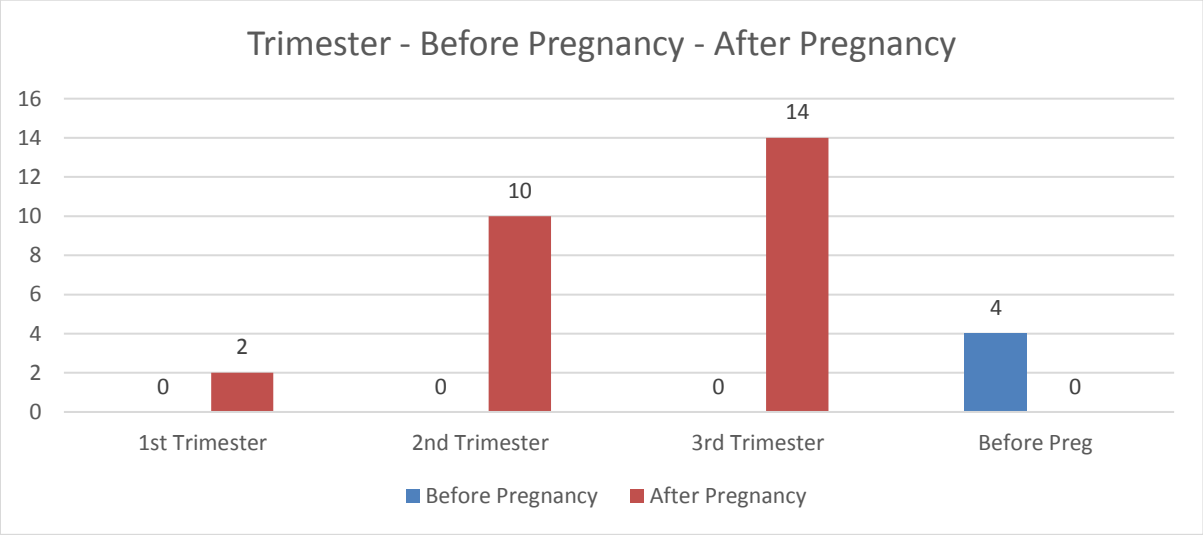
6.67% and 3.33% had splenic hilar and portosystemic collaterals respectively.



XXXVII COMPARISON OF BEFORE vs AFTER PREGNANCY SPLENOMEGALY

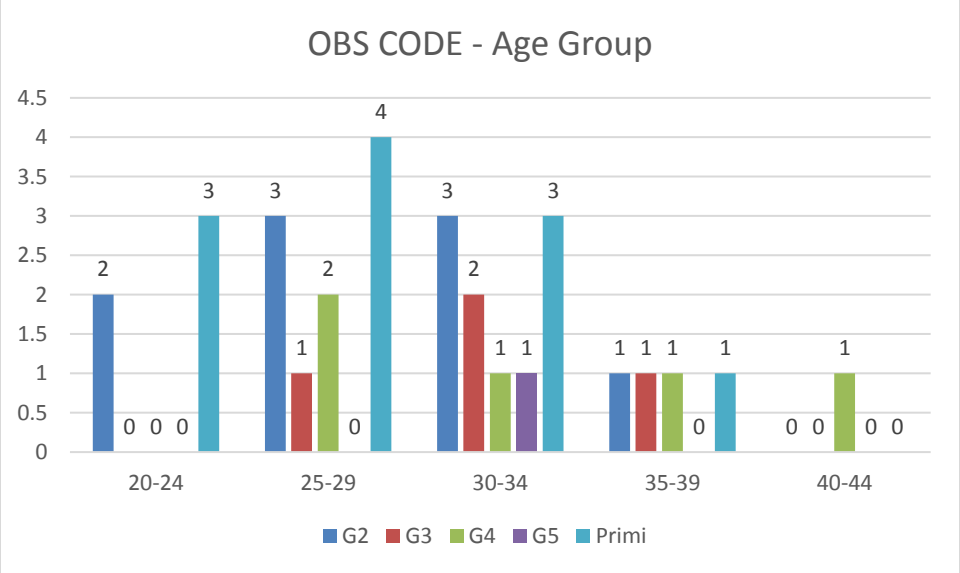
Trimester	Before Pregnancy	After Pregnancy	
1st Trimester	0	2	2
2nd Trimester	0	10	10
3rd Trimester	0	14	14
Before Preg	4	0	4
Grand Total	**4**	**26**	**30**
Chi-Square = 30			
Degrees of Freedom = 3			
p = 0.000001			

Only 4 patients had splenomegaly before pregnancy , whereas 26 patients were diagnosed to have splenomegaly during or after pregnancy. This association also had statistical significance with a p value of 0.000001



XXVIII DISTRIBUTION OF AGE AMONG SPLENOMEGALY MOTHERS

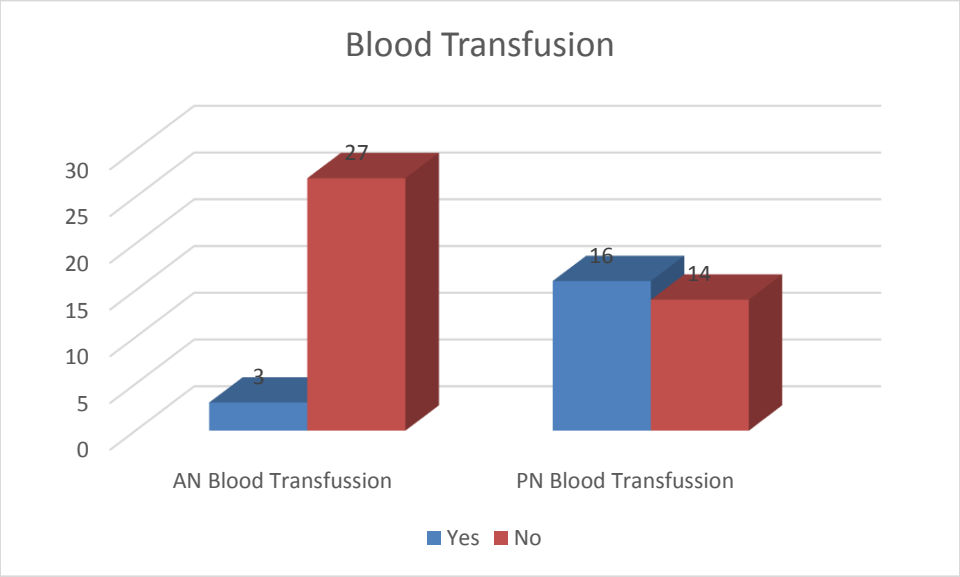
OBS CODE	Age Group					Grand Total
	20-24	**25-29**	**30-34**	**35-39**	**40-44**	
G2	2	3	3	1	0	9
G3	0	1	2	1	0	4
G4	0	2	1	1	1	5
G5	0	0	1	0	0	1
Primi	3	4	3	1	0	11
Grand Total	**5**	**10**	**10**	**4**	**1**	**30**
Chi-Square = 11.0341						
Degrees of Freedom = 16						
p = 0.807376						



Age distribution does not have significant association with the prevalence of splenomegaly in our study group (p value 0.807376)

XXXIX DISTRIBUTION OF TRANSFUSION

Blood Transfusion	AN Blood Transfusion	PN Blood Transfusion
Yes	3	16
No	27	14
Grand Total	30	30
Chi-Square = 13.0167		
Degrees of Freedom = 1		
p = 0.0003		

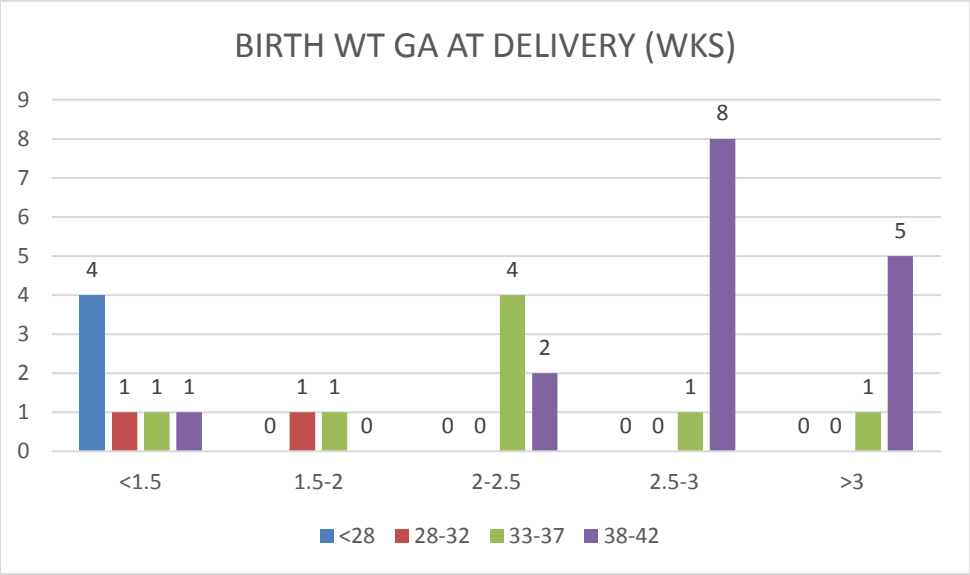


The significant blood transfusions seen among the postnatal mothers p value 0.0003

XXXX COMPARISON OF BIRTH WEIGHT vs GESTATIONAL AGE

BIRTH WT	GA AT DELIVERY (WKS)				**Grand Total**
	<28	**28-32**	**33-37**	**38-42**	
<1.5	4	1	1	1	7
1.5-2	0	1	1	0	2
2-2.5	0	0	4	2	6
2.5-3	0	0	1	8	9
>3	0	0	1	5	6
Grand Total	**4**	**2**	**8**	**16**	**30**
Chi-Square = 32.9018					
Degrees of Freedom = 12					
p = 0.001					

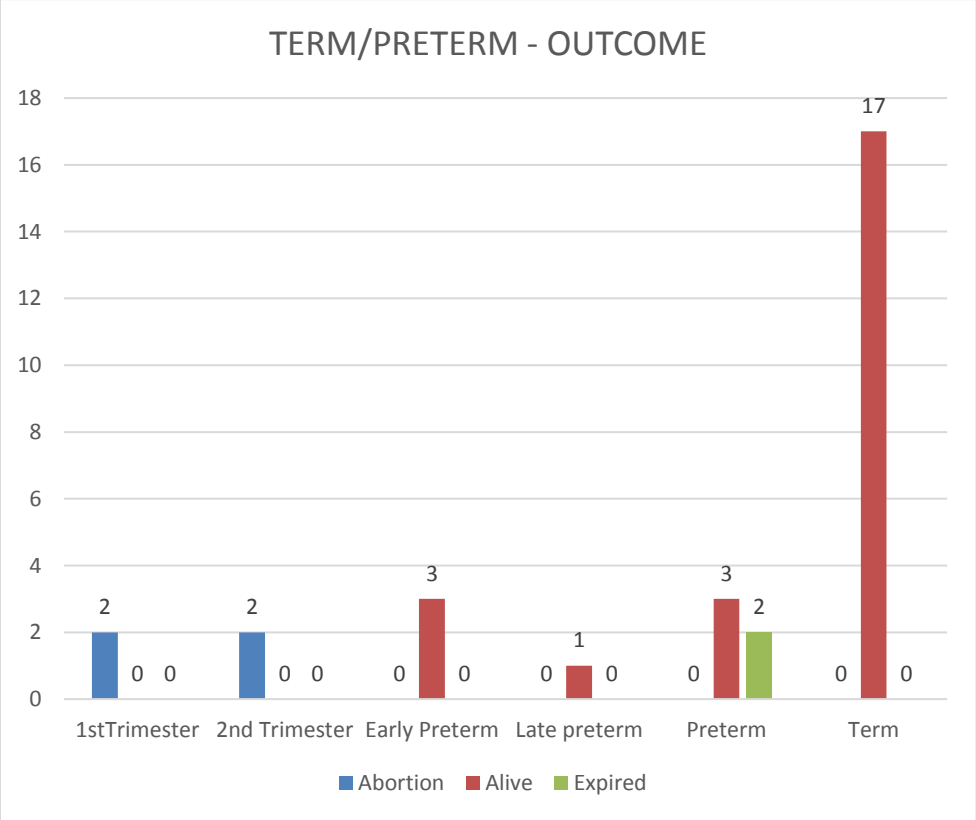
Low birth weight correlates with preterm delivery or less gestational age in our study group with significant p value 0.001.



XXXXI COMPARISON OF TIME OF DELIVERY vs OUTCOME OF DELIVERY

TERM/PRETERM	OUTCOME			Grand Total
	Abortion	**Alive**	**Expired**	
1stTrimester	2	0	0	2
2nd Trimester	2	0	0	2
Early Preterm	0	3	0	3
Late preterm	0	1	0	1
Preterm	0	3	2	5
Term	0	17	0	16
Grand Total	**4**	**24**	**2**	**30**
Chi-Square =40.5				
Degrees of Freedom = 10				
p = 0.0001				

Early weeks of delivery like 1st or 2nd trimesters were associated with worst outcome such as abortion/ death . This association carried statistical significance with a p value of 0.0001.



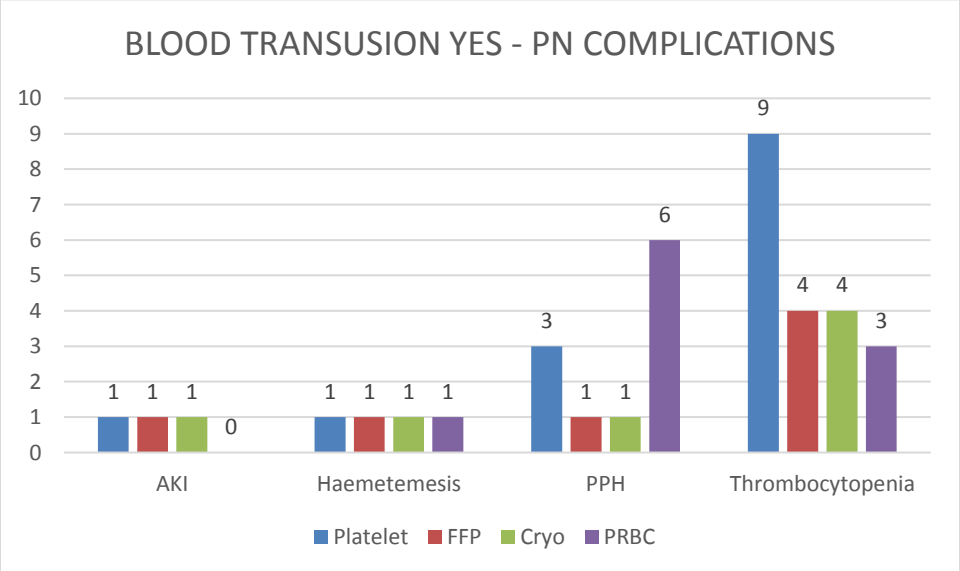
XXXXII COMPARISON OF BLOOD TRANSFUSION vs POSTNATAL COMPLICATIONS

BLOOD TRANSUSION YES	PN COMPLICATIONS				**Grand Total**
	AKI	**Haemetemesis**	**PPH**	**Thrombocytopenia**	
Platelet	**1**	**1**	**3**	**9**	**14**
FFP	**1**	**1**	**1**	**4**	**7**
Cryo	**1**	**1**	**1**	**4**	**7**
PRBC	0	1	**6**	**3**	**10**
Grand Total	3	4	11	20	38
Chi-Square = 20.6954					
Degrees of Freedom = 9					
p = 0.014					

Patients who had postnatal complications like thrombocytopenia received platelet transfusions,

PPH received packed red blood cell transfusion and hematemesis received FFP/Cryo transfusion.

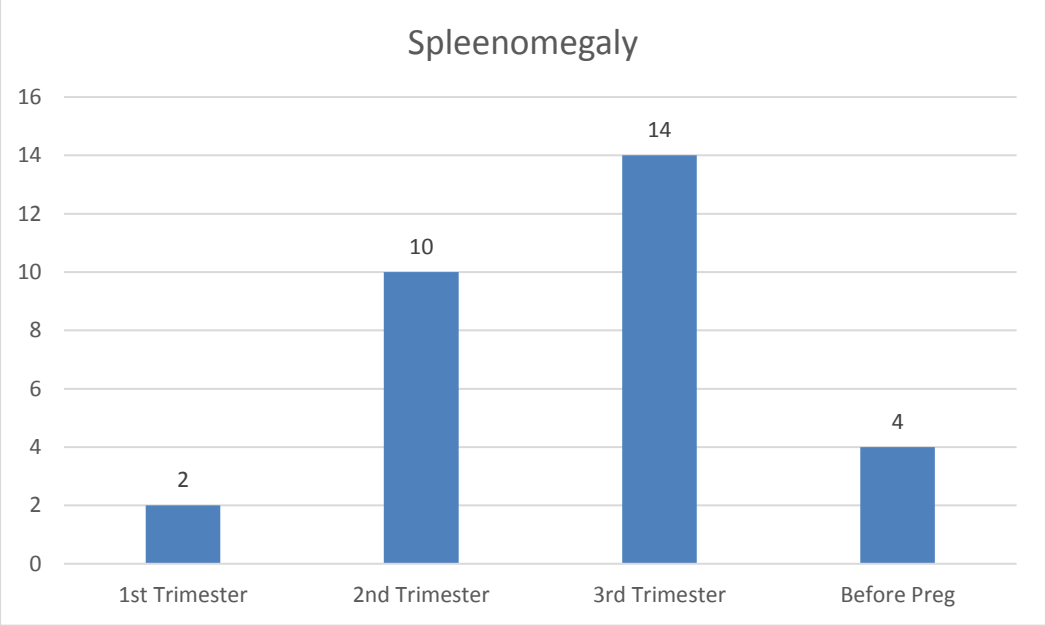
And these associations carried statistical significance (p value 0.014)



XXXXIV COMPARISON OF SPLENOMEGALY vs TIME OF PREGNANCY

TRIMESTER	Spleenomegaly	X^2	df	P
1st Trimester	2	12.13	3	0.0069
2nd Trimester	10			
3rd Trimester	14			
Before Preg	4			
Grand Total	**30**			

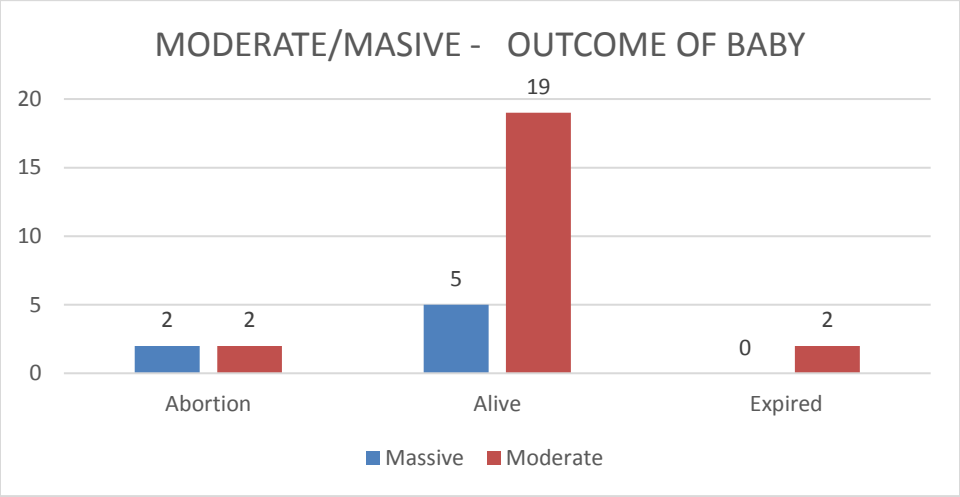
Among our study group, 3rd trimester was more commonly associated with prevalence and diagnosis of splenomegaly and this association had significant statistical value of p value 0.0069



XXXXV COMPARISON OF GRADING OF SPLENOMEGALY vs FOETAL OUTCOME

MODERATE/MASIVE	OUTCOME BABY			**Grand Total**
	Abortion	**Alive**	**Expired**	
Massive	2	5	0	7
Moderate	2	19	2	23
Grand Total	**4**	**24**	**2**	**30**
Chi-Square = 2.28261				
Degrees of Freedom = 2				
p = 0.319402				

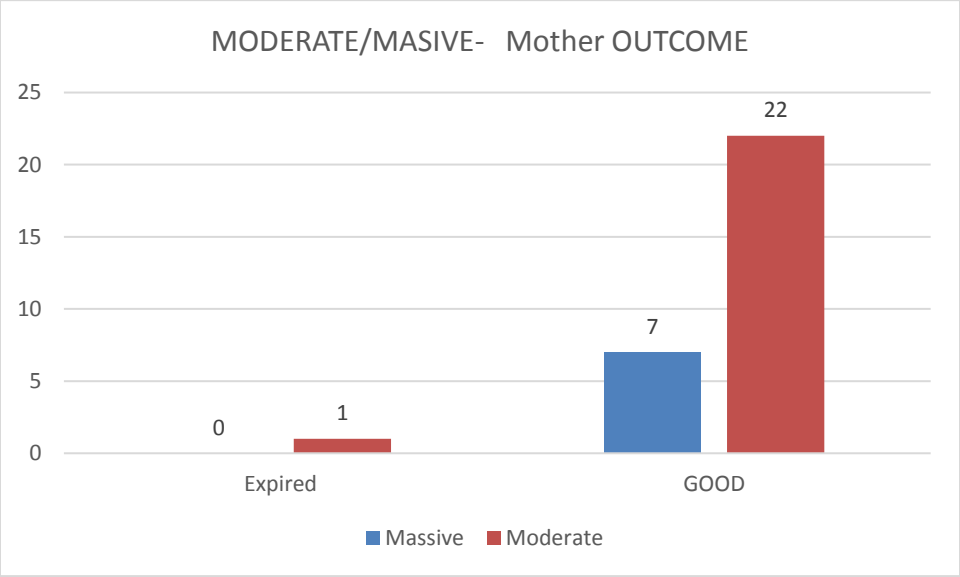
Grading of splenomegaly does not correlate with the outcome of foetus in our study group. P value is 0.319402.



XXXXVI COMPARISON OF GRADING OF SPLENOMEGALY vs MOTHER OUTCOME

MODERATE/MASIVE	Mother Out Come		**Grand Total**
	Expired	**GOOD**	
Massive	0	7	7
Moderate	1	22	23
Grand Total	**1**	**29**	**30**
Chi-Square = 0.314843			
Degrees of Freedom = 1			
p = 0.574724			

Outcome of mother also does not correlate with the grading of splenomegaly in our study group with a p value of 0.574724.



DISCUSSION

Splenomegaly in pregnancy is an important co-morbidity and accounts for lot of medical and pregnancy related antenatal /postnatal complications. It is also thought to intervene in the health of the foetus and outcome of delivery. Diagnosis of definite splenomegaly also becomes difficult in pregnancy as spleen enlarges normally to some extent physiologically in pregnancy. Hence in our study, we analyse the demographic profile, various clinical , etiological, haematological, laboratory , radiological features associated with the pregnant patients with splenomegaly. Also we compared the outcome in mother and foetus and various complications occurred to mother and foetus were observed. In addition, treatment modalities such as transfusion adopted in our hospital were also analysed.

Among 30 pregnant patients with splenomegaly in our study group, most prevalent age group is 25-34 years of age (66.66%). Specific age distribution was not associated with the occurance of splenomegaly in our study group. Most of the patients were primigravida (36.6%)

Thrombocytopenia (26.67%) is commonly associated with splenomegaly in our study group followed by gestational hypertension(16.67%). Heart disease was prevalent in 10% of patients. Acute kidney injury, Congestive Gastropathy , DCLD, Dengue, Diabetes Mellitus, Hypothyroidism, Infertility treatment , Jaundice, Partial HELLP syndrome, Portal hypertension, low lying Placenta, PLHA, Post Covid Pneumonia, PTB, Seizure disorder are the less common c0-morbidities prevalent in our study group of mothers with splenomegaly.

Among our study group, splenomegaly was present even before pregnancy in 13.33% of patients and diagnosed to have splenomegaly during or after pregnancy in 86.67% of patients. Diagnosis and occurance of splenomegaly is more commonly seen during or after pregnancy when

compared to before pregnancy and this association had statistical significance (p value 0. 000001). Also, this is in accordance with the results of study by seema singhal et al. Prior treatment was initiated for 10% of patients.

Particularly maximum diagnosis of splenomegaly was made during 3rd trimester in pregnancy (46.67%). This may be due to the fact that enlarged spleen may cause compressive symptoms over the maximally enlarged uterus in 3rd trimester and makes the patient symptomatic. This association of splenomegaly more commonly prevalent during 3rd trimester of pregnancy carried statistical significance (p value 0.0069) . Massive splenomegaly (> 18 cm) was seen in 23.33 % of patients , whereas moderate splenomegaly (< 18 cm) in 76.67% of pregnant mothers

Thrombocytopenia was prevalent in 40% of individuals with severe thrombocytopenia (< 50000 cells/cu.mm) in 6.67% of patients. Coagulation abnormalities were observed in 16.67% of patiets. Normal liver function test were seen in our study group mostly.

Radiological findings observed in ultrasonagraphy was splenomegaly in which moderate in 83.33% and massive in 16.67% of population. Less prevalent findings were portal hypertension and cavernoma (10% each), portal vein thrombosis (6.67%) and parenchymal liver disease (6.67%). B/L HUN and caudate lobe hypertrophy were each seen in 1 patients. Collaterals were seen in 26.67% of population. However, portal vein Doppler confirmed portal vein thrombosis and cavernoma in only 6.67% of patients. Whereas portal hypertension was confirmed in 20% and extrahepatic portal vein obstruction was seen in 13.33% of patients. 36.67% of patients were confirmed to have collaterals by Doppler study whereas only 26.67% had collaterals in USG. 16.67% had splenorenal collaterals commonly and 13.33% of patients had esophageal varices as the second most common site of collaterals. 6.67% and 3.33% had splenic hilar and

portosystemic collaterals respectively in our population.Endoscopic findings revealed commonly oesophageal varices in 4 patients, with red column (s/o bleeding) in 1 patient and antral gastritis in 2 patients of our study population.

Regarding delivery and maternal outcome, 63% of patients undergone LSCS and 13% had abortions . Only 24% of patients delivered by normal vaginal delivery. LSCS was advocated as the better mode of delivery in study by Aggarwal N et al. Among these patients, 53% had normal term delivery whereas 13.33% of patients had preterm delivery (< 28 weeks). Similar rates of abortion and preterm delivery were observed in study by Aggarwal N et al and Kochhar R et al. Postnatally maternal complications such as PPH, thrombocytopenia, hematemesis and AKI were prevalent in our study group. In contrast, PPH was observed as the most encountered complication in study by Seema singhal et al and Aggarwal N et al probably due to co-existent thrombocytopenia and coagulation abnormalities.

These patients were treated with supportive measures like Blood transfusion. 14 patients received platelet transfusion, 7 patients FFP and cryoprecipitate (each) and 10 patients received Packed red cell transfusion. Postnatal transfusions were done most commonly than antenatal transfusion in splenomegaly patients and occurance of complications also correlates with the blood transfusion (both had significant p value 0.0003, 0.014 respectively). However, severity of thrombocytopenia or anaemia does not correlate with the occurance of transfusion (p value 0.44, 0.88). Fatal outcome of death was observed in 1 patient (3.33%) with splenomegaly.

Regarding foetal outcome, abortion occurred in 13% of patients and dead born / fetal demise was seen in 6.66% of population. Among 80% of normal babies born, 53.33% of babies had SNN admission due to various reasons. very low birth weight (< 1.5 kg) was seen in 23.33% o f

babies and normal weight was seen in 50% (>2.5 kg) of neonates. SNN admission were due to low birth weight (16.67%), IUGR (13.33%), preterm (6.67%), Jaundice(3.33%), RDS (3.33%) and small for gestational age (10%) of neonates. This is similar to study results by Seema singhal et al.(15) Gestational age at birth and birth weight correlates with the outcome of birth in our study group (0.0001, 0.001 p value).

However, grading of splenomegaly does not correlate with the outcome of mother and foetus in our study group (p value 0.31, 0.57 respectively).

CONCLUSION

Splenomegaly in pregnancy is commonly seen in endemic countries like India , however, Studies regarding outcome and management of splenomegaly in pregnancy is still lacking. Our study attempts to document the clinical, radiological and etiological profile of splenomegaly in pregnant patients. It also analyses the outcome, complications of pregnant mother and various treatment modalities followed in our hospital. Also outcomeof fetus is also observed.

- Age distribution does not specific association with occurance of splenomegaly. Primigravida was more common in our study group.
- Thrombocytopenia and gestational hypertension are the common associated /risk factors observed along with splenomegaly in our study group
- Splenomegaly was present and diagnosed during and after pregnancy when compared prior to pregnancy itself in our study group with significant statistical association
- Particularly , splenomegaly was maximally found and diagnosed in 3 rd trimester of pregnancy in 46.67% of population and this association displayed statistical significance.
- Moderate splenomegaly (76.67) was more common than massive enlargement of spleen(23.3%) in our study group.
- Co-existent anaemia , thrombocytopenia and coagulation abnormalities are haematological manifestations seen in our study group.
- Radiological features (USG and portal vein Doppler) contributing to etiology were

USG FINDINGS – ETIOLOGY	PERCENTAGE IN OUR STUDY
• Portal vein thrombosis	• 10%
• Portal vein cavernoma	• 10%

• Portal hypertension	• 20%
• Parenchymal liver disease	• 6.67%
• Extra hepatic portal vein obstruction	• 13.33%
• Non cirrhotic portal fibrosis	• 3.33%

- Etiological is similar to that given by study by seem singhal et al (15) and Hoffbrand AV et al.
- Complications of splenomegaly such as collaterals and oesophageal varices were observed in Doppler studies and endoscopy
- Severe thrombocytopenia, PPH, Hematemesis , AKI, abortions were the postnatal complications observed commonly in splenomegaly mothers
- They were commonly treated with supportive transfusion either platelets, packed red blood cells, FFP, cryo according to the clinical presentation
- Post natal blood transfusion were more common than antenatal transfusion and treatment of transfusion correlates with the presence of postnatal complication with statistical significance.
- Fatal outcome in mother was observed in 1 patient in our study group
- Fetal outcome of preterm delivery, IUGR, very low birth weight , SGA are common in splenomegaly mothers and warrants SNN admission in our study
- Also, abortions (13%) and fetal death (6.67%) were observed in our study group of splenomegaly mothers
- However, outcome in mother or foetus does not correlate with the grading and severity of splenomegaly in our study group

Thus we observed the complete clinical profile of splenomegaly with pregnancy patients and still the obstetric complications encountered and treatment should be studied in detail further.

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நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

S.NO	NAME	AGE	OBS CODE	OBS CODE	BEFORE PREG	AFTER PREG	DIAGNOSIS AT TRIMESTER	PRIOR TREATMENT	TREATMENT DETAILS	GA AT DELIVERY (WKS)	INDUCED/NOT	TERM/PRETERM	MODE OF DELIVERY	BIRTH WT	SNN ADMISSION	REASON FOR ADMISSION(AT DELIVERY)	IP BABY INV DETAILS	OUTCOME BABY	USG FINDINGS	SPLEEN SIZE (CM)	SPLENOMEGALY MODERATE / MASSIVE	PORTAL VEIN DOPPLER (ETIOLOGY)	COLLATERALS	collaterals /varices	OTHER ABNORMALITIES	OTHER AN COMPLICATIONS	RISK FACTORS	PN COMPLICATIONS	BLOOD TRANSUSION	AN TRANSFUSION	PN TRANSFUSION	HIV/VDRL	HB	PCV	PLATELET COUNT	UREA	CREATININE	T.BILIRUBIN	DIRECT	INDIRECT	SGOT/SGPT	ALP	T.PROTEIN	ALB/GLO	NA^K^	PT	INR	APTT	Coagulation Abnormalities	PERIPHERAL SMEAR	OUT COME	FOLLOW UP	ENDOSCOPY	PROGNOSIS
1	Ramalakshmi	27	G2P1L1	G2	No	Yes	3rd Trimester	No	No	37	No	Term	LSCS	2.2	YES	IUGR	HIV prophylaxis	Alive	moderate splenomegaly	14	Moderate	moderate spleenomegaly	No	no	Yes (Hepatomegaly)	PLHA	Nil	PPH	Yes	NO	Yes	Reactive	10	31.2	3	19	0.8	0.8	0.4	0.4	18/18	96	5.1	3.1/2.0	138/4.1	14	1.6	32.4	No	Normal	Discharged	Nil complications	Not Done	GOOD
2	Thangam	32	G3P2L2	G3	Yes	No	Before Preg	Yes(medical)	UDCA	37	No	Term	LSCS	2	yes	LBW	LBW	Alive	massive splenomegaly,b/l hun(phy)	20	Massive	moderate spleen,pv 12mm,EHPVO	No	splenic hilar collaterals	No	Nil	Nil	Thrombocytopenia	Yes	no	yes	Non reactive	10.5	34.4	0.66	20	0.8	1	0.5	0.5	24/22	78	5.2	3.2/2.0	139/4.0	12	1.8	32.6	No	normocytic,normochromic	Discharged	Nil complications	Tiny easophageal varices	GOOD
3	Muthumari	27	Primi	Primi	No	Yes	3rd Trimester	No	No	38	No	Term	LSCS	3	no	Normal	Nil specific	Alive	moderate splenomegaly	15	Moderate	mild splenomegaly	No	no	No	Thrombocytopenia	mild mr (heart disease)	Thrombocytopenia	Yes	no	yes	Non reactive	12.1	38.2	1.2	18	0.8	0.9	0.5	0.4	18/20	96	5.3	3.3/2.0	140/4.2	12	1.6	45	Yes	Normal	Discharged	Nil complications	Not Done	GOOD
4	Thaipriya	27	G3A2	G3	No	Yes	3rd Trimester	No	No	38	No	Term	LSCS	2.7	no	Normal	Nil specific	Alive	portal cavernoma,moderate splenomegaly	14.5	Moderate	?hemanjioma liver,mod splenomegaly	Yes	Easophageal varices,	No	Nil	Nil	Nil	No	no	no	Non reactive	11.5	34.5	3.62	17	0.8	0.4	0.2	0.2	21/20	105	5	3.0/2.0	137/4.0	11	1.04	34.2	No	Normal	Discharged	Nil complications	Not Done	GOOD
5	Jothi	35	G4P3L3	G4	No	Yes	2nd Trimester	No	No	38	No	Term	LSCS	2.8	no	Normal	Nil specific	Alive	moderate spleenomegaly	14	Moderate	mild splenomegaly	No	no	No	Nil	HOB	Nil	no	no	no	Non reactive	11	34	2.65	17	0.7	1	0.5	0.5	22/21	102	5.1	3.1/2.0	136/4.0	11	1	28.6	No	normocytic normochromic	Discharged	Nil complications	Not Done	GOOD
6	Selvi	30	G5P4L3	G5	No	Yes	2nd Trimester	No	No	30	No	Early Preterm	NVD	0.8	yes	IUGR	Jaundice.IUGR, Preterm	Expired	moderate splenomegaly	14	Moderate	mild spleenomegaly	No	no	No	Thrombocytopenia	HOB	PPH	Yes	no	yes	Non reactive	9	33	2.5	16	0.6	0.8	0.4	0.4	19/21	89	5.1	3.1/2.0	142/3.9	12	1.02	29.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
7	Priyanka	21	Primi	Primi	No	Yes	3rd Trimester	No	No	33	yes	Preterm	LSCS	2	yes	IUGR	sepsis,IUGR,Preterm	Alive	moderate splenomegaly	15	Moderate	normal study	No	no	No	Partial hellp, AKI	GHTN	AKI	yes	no	yes	Non reactive	9.9	29	0.93	34	1.1	0.4	0.2	0.2	22/21	101	6	3.0/3.0	141/4.3	13	1.08	28	No	hemolytic	Discharged	Nil complications	Not Done	GOOD
8	rajaselvi	25	Primi	Primi	Yes	No	Before Preg	Yes (Surgery)	endoscopic variceal band ligation,propranol	26	No	2nd Trimester	Abortion	0.85	no	others	Abortion	Abortion	moderate splenomegaly	14.8	Moderate	EHPVO,splenorenal collaterals	Yes	splenorenal collaterals	Yes (Hepatomegaly)	low lying placenta,thrombocytopenia	thrombocytopenia,	haemetemesis	Yes	yes	yes	Non reactive	8.3	26	0.99	18	1.3	1.7	1	0.7	32/28	132	5	3.0/2.0	138/4.1	18	2.5	50	Yes	Hemolytic	Expired	Nil complications	Grade 3 varices with 3 column red signs	Expired
9	valliammal	30	G2P1L1	G2	Yes	No	Before Preg	Yes (medical)	propranolol, UDCA,	8	No	1stTrimester	Abortion	0.1	no	Others	Abortion	Abortion	massive splenomegaly,phtn,cavernous pv	21	Massive	portal gastropathy,EHPVO ,Massive spl	Yes	easophageal varices,	No	Thrombocytopenia	Nil	Thrombocytopenia	Yes	no	yes	Non reactive	9.5	29	0.63	20	0.9	6.3	4	2.3	185/201	25	5	3.0/2.0	141/3.9	16.7	1.3	42	Yes	microcytic,hypo	Discharged	good with medical trt	,antral gastritis	GOOD
10	ulagammal	26	Primi	Primi	No	Yes	1st Trimester	No	No	38	No	Term	LSCS	2.4	yes	SGA	Anemia, asphyxia,SGA	Alive	massive splenomegaly,PHTN	25	Massive	parenchymal liver disease,phtn,splee nomegaly with splenic collaterals	yes	splenorenal collaterals	parenchymal liver disease	Thrombocytopenia	Nil	Thrombocytopenia	Yes	yes	yes	Non reactive	12.3	36.6	0.34	17	0.6	1.5	1	0.5	37/32	123	5.2	3.2/2.0	142/3.8	14.9	1.16	40	Yes	normocytic,normochromic	Discharged	Nil complications	easophageal varices	GOOD
11	lakshmi	31	G3P2L2A1	G4	No	Yes	3rd Trimester	No	No	38	No	Term	LSCS	3.1	no	Normal	Nil specific	Alive	moderate splenomegaly	16	Moderate	portalvein thrombosis,splenomegaly	yes	easophageal varices,	Yes (Hepatomegaly, PHTN)	Mild TR, Old PTB treated,PHTN	Old Ptb, Mild MR(Heart disease)	Nil	No	no	no	Non reactive	10.5	33.6	3.5	18	0.7	0.9	0.5	0.4	21/23	96	5.6	3.3/2.3	139/4.0	11	1.04	23.8	No	Normal	Discharged	Nil complications	Not Done	GOOD
12	sankari	27	G2P1L1	G2	No	Yes	2nd Trimester	No	No	38	No	Term	NVD	2.7	yes	Normal	Nil specific	Alive	moderate splenomegaly,pv thrombosis,portalcavernoma,caudate lobe	17.5	Moderate	chronic pv thrombosis,portal cavernoma,chronic phtn,caudate lobe hypertrophy	Yes	splenorenal collaterals	yes(Hepatomegaly)	Nil	Nil	Nil	No	no	no	Non reactive	11.3	32.6	2.5	16	0.6	1.2	0.6	0.6	18/22	56	5.2	3.2/.0	137/3.9	12	1.09	32.4	No	Normal	Discharged	Nil complications	Not done	GOOD
13	kasthuri	31	G2P1L1	G2	No	Yes	3rd Trimester	No	No	39	No	Term	LSCS	3.2	no	Normal	Nil specific	Alive	moderate splenomegaly	16	Moderate	moderate spleenomegaly	No	Nil	No	Thrombocytopenia	Nil	PPH	yes	NO	YES	Non reactive	9.8	34.2	1.25	16	0.7	0.8	0.4	0.4	16/18	102	5.1	3.1/2.0	139/4.2	11.9	1.08	23.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
14	sankarammal	36	G2P1L1	G2	No	Yes	3rd Trimester	No	No	38	No	Term	LSCS	2.5	yes	SGA,RDS	SGA,Asphyxia,jaundice	Alive	massive splenomegaly,pv thrombosis	23	Massive	chronic pv thrombosis,Lt pv-thrombus+,recanalised umbilical vein with massive splenomegaly	yes	splenic hilar collaterals	chronic liver disease	GHTN	Nil	Thrombocytopenia	yes	no	no	Non reactive	10.5	33.4	1.45	16	0.8	1	0.5	0.4	21/28	96	6	3.5/2.5	139/3.9	12.8	1.96	34.4	No	Normal	Discharged	Nil complications	Easophageal varices	GOOD
15	ANDAL	26	G2P1L1	G2	No	Yes	3rd Trimester	No	No	37	No	Term	LSCS	3.3	yes	Normal	Nil specific	Alive	moderate splenomegaly	16	Moderate	moderate splenomegaly	No	No	No	Nil	Nil	Nil	No	no	no	Non reactive	10.2	30.6	1.96	17	0.8	1	0.5	0.5	19/18	96	5.5	3.2/2.3	140/3.9	12	1.12	25.8	No	Normal	Discharged	Nil complications	Not Done	GOOD
16	kokila	22	G2P1L0	G2	No	Yes	2nd Trimester	No	No	30	No	Preterm	NVD	1.8	yes	LBW,preterm	LBW,Preterm	Expired	moderate splenomegaly	16	Moderate	moderate spleenomegaly	No	No	No	Mild MR, MVP(Heart disease)	GHTN	Thrombocytopenia	yes	Yes	no	Non reactive	11.3	35	1.18	16	0.7	0.5	0.2	0.3	21/20	103	5.8	3.5/2.3	138/3.9	13	1.02	22.3	No	Normal	Discharged	Nil complications	Not Done	GOOD
17	maragatham	33	Primi	Primi	No	Yes	2nd Trimester	No	No	34	Yes	Early Preterm	LSCS	1.85	Yes	LBW,RDS	,Anaemia, ,LBW ,Preterm	Alive	portal cavernomatous changes with splenorenal collateral and massive splenomegaly	22	Massive	portal cavernomatous changes with splenorenal collateral and massive	Yes	splenorenal collaterals	No	Nil	Nil	Thrombocytopenia	Yes	No	yes	Non reactive	10.4	32.3	0.63	17	0.7	0.8	0.4	0.4	24/28	98	5.5	3.5/.5	140/3.8	15.5	1.5	39.5	Yes	thrombocytopenia	Discharged	Nil complications	Normal study	GOOD
18	Arunthil vadivu	35	Primi	Primi	No	Yes	2nd Trimester	No	No	38	Yes	Term	LSCS	2.95	no	Normal	Nil specific	Alive	moderate spleenomegaly	17	Moderate	massive spleenomegaly	No	no	No	Thrombocytopenia,infertility	Nil	Thrombocytopenia	No	No	no	Non reactive	11.7	34.2	1.65	16	0.8	0.9	0.4	0.5	18/21	85	5.1	3.1/2.0	142/4.1	17.9	1.3	25.7	No	Normal	Discharged	Nil complications	tiny easophageal varices	GOOD
19	Savariammal	32	Primi	Primi	No	Yes	1st Trimester	No	No	37	No	Preterm	LSCS	2.4	yes	IUGR	Nil specific	Alive	moderate spleenomegaly	14	Moderate	moderate spleenomegaly, phtn	No	no	No	Thrombocytopenia,infertility	Nil	Nil	No	no	no	Non reactive	12.8	34.6	1	16	0.9	0.6	0.3	0.3	19/25	106	6.2	3.2/3.0	139/40	18.6	1.06	33.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
20	Amudha	29	G4P2L2A1	G4	No	Yes	2nd Trimester	No	No	36	No	Early Preterm	NVD	2.5	yes	Preterm	Anemia	Alive	moderate splenomegaly	14	Moderate	moderate splenomegaly	No	no	Yes (Cholelithiasis)	Type 2 dm	Nil	Nil	No	no	no	Non reactive	9.9	30.6	2.34	16	0.7	0.9	0.5	0.4	23/28	98	5.1	3.1/2.0	137/3.9	14	1.8	27.8	No	Normal	Discharged	Nil complications	Not Done	GOOD
21	petchiammal	43	G4P3L2	G4	No	Yes	3rd Trimester	No	No	38	No	Preterm	NVD	2.3	yes	LBW,SGA	,Jaundice,LBW, SGA	Alive	moderate splenomegaly with caudate lobe hypertrophy	14.5	Moderate	moderate splenomegaly with caudate lobe hypertreophy,hepatomegaly	No	no	Yes (Hepatomegaly)	Hypothyroidism	Hypothyroidism	Nil	No	no	no	Non reactive	11.2	33.5	1.84	14	0.7	1	0.5	0.5	20/21	126	6	3.0/3.0	140/4.1	14.8	1.5	26.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
22	malliga	26	Primi	Primi	No	Yes	3rd Trimester	No	No	39	Yes	Term	LSCS	3.3	no	normal	Nil specific	Alive	moderate splenomegaly	15	Moderate	mild splenomegaly	No	no	No	Post covid pneumonia	Covid recoveed	PPH	Yes	no	Yes	Non reactive	10.8	31.5	2.34	17	0.8	0.7	0.4	0.3	23/22	134	5.3	3.3/2.0	143/3.9	15.3	1.05	24.4	No	Normal	Discharged	Nil complications	Not Done	GOOD

23	jeevammal	30	G3P2L2	G3	No	Yes	3rd Trimester	No	No	38	No	Term	LSCS	2.8	Yes	Observation	Nil specific	Alive	moderate splenomegaly	15.5	Moderate	hepatospleenomegaly	No	no	perfusion defect liver	Seizure disorder	seizure disorder	PPH	Yes	no	yes	Non reactive	9.8	31	1.75	18	0.7	0.9	0.5	0.4	25/26	101	5.4	3.2/2.2	139/4.1	17.8	1.15	23.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
24	Vardhini	20	Primi	Primi	No	Yes	3rd Trimester	No	No	38	Yes	Term	LSCS	3.5	no	Normal	Nil specific	Alive	moderate splenomegaly	16.5	Moderate	mild splenomegaly	No	no	No	GHTN	GHTN	Nil	No	no	no	Non reactive	10.4	29.5	2.56	17	0.7	0.8	0.4	0.4	19/18	99	5.1	3.0/.1	135/4	15.3	1.06	27.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
25	poomari	33	G2P1L1	G2	No	Yes	3rd Trimester	No	No	38	Yes	Late preterm	bour Natu	1.35	yes	BW,RDS,IUG	jaundice,LBW,IUGR,RDS	Alive	massive splenomegaly	26	Massive	massive spleenomegaly	Yes	easophageal varices,	No	GHTN	GHTN	PPH	Yes	no	yes	Non reactive	9	28.6	1.14	17	0.6	3.26	2.16	1.1	32/25	122	5.6	3.3/2.3	137/4.1	18.05	1.92	31.6	No	Normal	Discharged	Nil complications	easophageal varices	GOOD
26	CHITRA	33	Primi	Primi	No	Yes	2nd Trimester	No	No	34	No	Early Preterm	bour Natu	1.4	yes	LBW,preterm	,LBW,Preterm	Alive	moderate splenomegaly,parenchymal liver disease, severe PHTN with collaterals	17	Moderate	,NCPF,moderate splenomegaly,severe PHTN with portosystemic collaterals	Yes	porosystemic collaterals	yes(parenchymal liver disease)	dengue +ve,GHTN	mild MR(Heart disease)Dengue +ve	Thrombocytopenia	Yes	no	yes	Non reactive	11.5	34.5	0.35	17	0.7	0.5	0.3	0.2	24/29	96	5.2	3.2/2	138/4.3	21.5	1.35	32.8	No	thrombocytopenia	Discharged	good with medical trt	easophageal varices	GOOD
27	maari	20	Primi	Primi	No	Yes	2nd Trimester	No	No	12	No	1stTrimester	Abortion	0.5	no	Others	Abortion	Abortion	massive splenomegaly	20	Massive	,EHPVO,PHTN	Yes	no	No	Jaundice	Nil	PPH	Yes	no	yes	Non reactive	10	29.5	1.8	17	0.6	0.8	0.4	0.4	20/21	105	5.3	3.3/2	135/4.2	19.5	1.04	33	No	Normal	Discharged	Nil complications	Antral gastritis	GOOD
28	mariammal	36	G3P1L1A1	G3	No	Yes	2nd Trimester	No	No	14	Yes	2nd Trimester	Abortion	0.85	no	Others	Abortion	Abortion	moderate splenomegaly,	16	Moderate	moderate splenomegaly	No	no	No	Nil	Nil	Nil	No	no	no	Non reactive	9.8	28.6	1.7	17	0.7	0.9	0.4	0.5	22/22	127	5	3/2	137/3.9	18.5	1.98	34.5	No	Normal	Discharged	Nil complications	Not Done	GOOD
29	suganya	29	G4P2L2A1	G4	No	Yes	3rd Trimester	No	No	38	No	Term	LSCS	2.7	no	Normal	Nil specific	Alive	moderate spleenomegaly	16	Moderate	mild splenomegaly	No	no	No	GHTN, MVP AML(Heart disease)	GHTN, MPV AML(Heart disease)	Nil	No	no	no	Non reactive	12.5	36.4	2.56	16	0.7	0.5	0.3	0.2	19/18	112	5.5	3.0/2.5	139/3.7	13.4	1.07	31.2	No	Normal	Discharged	Nil complications	Not Done	GOOD
30	mariammal	24	G2P1L1	G2	Yes	No	Before Preg	Yes (Surgery)	propranolol,band ligation	38	No	Term	LSCS	2.9	no	Normal	Nil specific	Alive	moderate splenomegaly,parenchymal liver disease,	16	Moderate	paenchymal liver disease,moderate splenomegaly	yes	splenorenal collaterals	No	DCLD, Congestive gastropathy on treatment	Nil	Thrombocytopenia	Yes	no	yes	Non reactive	1.1	33	3.1	17	0.7	1	0.5	0.5	21/22	113	5.3	3.3/2	138/4	17.5	1.5	28	No	Normal	Discharged	Nil complications	Normal study	GOOD