A STUDY OF NON INFECTIVE JAUNDICE IN PREGNANCY

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CERTIFICATE

This is to certify that the dissertation entitled "**A STUDY OF NON INFECTIVE JAUNDICE IN PREGNANCY"** submitted by **Dr.P.NANDINI,** to the Tamil Nadu Dr. M.G.R. Medical University in partial fulfilment of the requirement for the award of M.S.(Obstetrics & Gynaecology) Branch II is a Bonafide research work carried out by her under our direct supervision and guidance.

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This is to certify that the dissertation entitled **"A STUDY OF NON INFECTIVE JAUNDICE IN PREGNANCY "** is a bonafide and genuine research work done by **Dr.P.NANDINI,** in partial fulfilment of the requirement for the degree in M.S.(Obstetrics & Gynaecology), under guidance of **Prof.Dr.JOTHI SUNDARAM.,MD(OG)** Additional Professor, Department of Obstetrics & Gynaecology.

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DECLARATION

I, **Dr.P.NANDINI,** hereby declare that, I carried out this work on "**A STUDY OF NON INFECTIVE JAUNDICE IN PREGNANCY "** in GOVT RAJAJI HOSPITAL at the Department of Obstetrics & Gynaecology, Madurai under the guidance of **Prof. Dr.JOTHI SUNDARAM, MD.(OG).,** Professor of Obstetrics & Gynaecology during the period of January 2016 to July 2016. I also declare that this bonafide work has not been submitted in part or full by me or and others for any award, degree or diploma to any other University or Board either in India or abroad.

This is submitted to The Tamilnadu Dr. M. G. R. Medical University, Chennai in partial fulfilment of the rules and regulation for the award of M.S degree Branch – II (Obstetrics & Gynecology) to be held in April 2018.

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INTRODUCTION

Pregnancy with jaundice is considered as high risk pregnancy. Jaundice affects a small percentage of pregnant women, yet it takes a major toll on health of both mother and fetus especially in developing countries like India.

It complicates 3-5% of pregnancies and is one of the important causes of maternal and neonatal morbidity and mortality worldwide. Throughout the pregnancy there exists alteration in normal physiological and hormonal profiles.

Incidence of jaundice in pregnancy is 0.4-0.9/1000 in India.

It could be peculiar to the pregnancy such as acute fatty liver of pregnancy, recurrent cholestatic jaundice in pregnancy and jaundice complicating preeclampsia of pregnancy.

It can be concurrent with pregnancy such as due to infective pathology like viral hepatitis and also due to preeclampsia ,HELLP,Acute fatty liver in pregnancy,intrahepatic cholestasis in pregnancy, hyperemesis gravidorum,Wilson disease,cirrhosis with portal hypertension or it could be due to drugs administered during pregnancy

The present study analyses the cause of the disease, altered liver function, maternal and fetal morbidity and mortality and preventive measures in jaundice complicating pregnancy. This study will be helpful in better understanding and improving the maternal and perinatal outcome in jaundice complicating pregnancy

AIM OF THE STUDY

1. Studying the incidence of non infective jaundice in pregnant women in this tertiary care centre
2. To enumerate the various causes of non infective jaundice in pregnancy
3. To follow the course of the disease and the numerous complications it ensues
4. To form a preplanned algorithm of investigations to diagnose non infective jaundice in pregnancy
5. To study the best protocol for the management of non infective jaundice in pregnancy
6. To follow maternal and fetal outcomes in non infective jaundice in pregnancy

REVIEW OF LITERATURE

During the state of pregnancy, the human body undergoes a multitude of changes in the process of its acclimatisation to the growing fetus. Inspite of most of these changes being physiological, the potential for morbidity and mortality to both mother and fetus is a real threat.

Liver is prominent as the bed of many important metabolic and synthetic functions of the body. In pregnancy, the liver is not palpable normally. Serum albumin concentrations decreases due to rise in plasma volume. ALP rises in third trimester as a result of rise in placental ALP and bone iso-enzyme. Serum transaminases increase only during labour as a consequence of leakage from uterine muscles. 3%-5% of pregnancies show abnormal liver function tests. The potential causes are coexisting liver disease (commonest being viral hepatitis or gallstones) and underlying chronic liver disease. A kaleidoscopic range of liver diseases are encountered in pregnancy.

The liver could be targeted by diseases specific to the pregnancy like the intrahepatic cholestasis of pregnancy and the acute fatty liver of pregnancy, there being a paucity of available means which may be used to predict how and when such illnesses may occur with reasonable certainity. India, being a tropical country, shows a high morbidity and mortality statistics due to liver diseases in pregnancy. Also, morbidity is more likely to occur in the presence of a preexisting liver disease in a pregnant woman as in autoimmune hepatitis or when a new onset liver disease occurs during the period of gestation as in herpes simplex hepatitis.

Many physiologic changes which are the norm during pregnancy could pose problems in evaluating hepatobiliary function because they may misleadingly appear as pathological. For instance, the plasma volume expands during pregnancy following retention of salt and water. This leads to a state of hemodilution. .

These changes which peak during the second trimester later plateau until delivery. As a result, serum levels of uric acid, total protein, albumin, and

hematocrit are lowered. On the other hand, serum alkaline phosphatase levels may rise in third trimester as a result of placental production, while serum values of liver enzymes namely aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl transpeptidase (GGT), and also the bilirubin and prothrombin time remain well within the normal range.

Oestrogens favour biliary cholesterol saturation and also inhibit the hepatic synthesis of chenodeoxycholic acid, while progesterone plays a role in decreasing the gall bladder contractility and favours lithogenicity leading to the accumulation of sludge and gallstone formation. But when appropriately and timely diagnosed and managed, the outcome may be positive and the liver disease in pregnancy could resolve on its own without any chronic consequences in the future.

Although there exists various associations between hepatic dysfunction and pregnancy, it is to be re emphasized that hepatic disease is a rarity in pregnancy. The leading cause of jaundice in pregnancy is acute viral hepatitis. The course of acute hepatitis is not affected in pregnancy in all viral hepatitis except hepatitis E virus which is associated with many complications which peak in the third trimester of gestation.

The liver is the hot house of a myriad functions- the biotransformation of those compounds which are insoluble (i.e bilirubin, drugs, toxins), the metabolism and excretion of cholesterol and bilirubin, the synthesis of plasma proteins (i.e albumin, globulin, coagulation factors, transferrin, haptoglobin), and amino acid metabolism, carbohydrates and lipid metabolism. Pregnant women with chronic liver disease show a higher risk of fetal loss during gestation. Pre-eclampsia especially when associated with HELLP syndrome results in an increase in maternal and fetal morbidity and mortality.

There is no single liver function test which successfully quantifies liver disease. A rise in level of AST and ALT predict liver cell necrosis, while synthetic

function is quantified by ranging albumin level and determining prothrombin time. Cholestasis and biliary obstruction are determined by measuring the levels of bilirubin, alkaline phosphatase, gamma glutamyl transpeptidase and 5-nucleotidase. In normal pregnancies, there may be an increase in ALP which is a result of placental ALP

Though liver is not palpable in normal pregnancy, palmar erythema and vascular nevi may appear in pregnancy. Serum biochemical tests show increase in alkaline phosphatase and cholesterol and serum bile acid in the last trimester . This elevation rarely exceeds two or four folds the non pregnant value and mainly is of placental origin. Also serum albumin concentration may fall to values 0-60% than those in the non gravid state secondary to the increase in plasma volume. Changes in liver function is also confirmed by reduced bromsulphthalein uptake.

The point to be noted is that blood flow constitutes 35% of the cardiac output in nongravid patient, in contrast during pregnancy it falls to 28% as the remaining blood is shunted through the placenta. Therefore liver is one of the primary organs affected during pregnancy due to metabolic and hormonal changes associated with gestation.

Jaundice in pregnancy is considered as high risk pregnancy. Though Jaundice affects just a small percentage of pregnant women, yet it plays a major detrimental role on health of both the mother and fetus especially in our country.

It complicates 3-5% of all pregnancies and is one of the leading causes of maternal , fetal and neonatal morbidity and mortality all over the world . Throughout the pregnancy there appears changes in both normal physiological and hormonal profiles.

Incidence of jaundice in pregnancy in India is 0.4-0.9/1000. It could be specific to the gestation such as acute fatty liver of pregnancy or recurrent

cholestatic jaundice of pregnancy or jaundice complicating toxemia of pregnancy. It may occur concurrently with pregnancy such jaundice caused by infective pathology like viral hepatitis or jaundice due to gallstones or it may be a result of drugs administered during the gestational period.

My current study analyses the cause of the disease, altered liver function ,maternal and fetal morbidity and mortality and preventive measures in jaundice complicating pregnancy as well as the outcome of the disease and the course of the pregnany. This study will play a role in understanding better and improving the maternal and perinatal outcome in jaundice complicating pregnancy.

While taking into consideration liver disease complicating pregnancy, it is useful to use **Sherlock's** classification and group

1) liver disease specific to pregnancy;
2) intercurrent liver disease co existing in pregnancy;
3) pregnancy complicating liver disease

-Jaundice specific to pregnancy such as cholestasis of pregnancy or pre eclampsia

-Jaundice aggravated by pregnancy which include viral hepatitis like Hepatitis B, Hepatitis E, Herpes Simplex virus infection during pregnancy leading to fulminant hepatic failure

-Jaundice due to liver disorders which are pre existing like Wilson’s diseases and chronic active hepatitis.

Viral hepatitis leads as the commonest cause of jaundice associated with pregnancy. The incidence of hepatitis varies markedly in different parts of the world. The incidence is around 0.1% in developed countries but it can range from 3-20% or more in developing countries. In developed countries there is not much of a difference in the course of the jaundice in both pregnant and non pregnant

women in however the incidence of maternal morbidity and mortality presented with fulminant hepatitis is higher in developing countries.

Liver Disease in Pregnancy Jaundice in Pregnancy May Be

A) Intercurrent In Pregnancy
B) Specific To Pregnancy
C) Acute On Underlying Chronic Disease

A) **Intercurrent In Pregnancy**
   1. Viral hepatitis
   2. Drug induced
   3. Gall stones

B) **Specific To Pregnancy**
   1. Cholestatic jaundice
   2. Acute fatty liver of pregnancy
   3. HELLP syndrome

C) **Underlying Chronic Liver Disease**
   1. Cirrhosis of liver
   2. Chronic hepatitis

Physiological Changes during Pregnancy

Increase

1. Plasma volume, cardiac rate and rise in cardiac output by 35%-50% which peaks at 32 weeks. Furthermore it increases by 20% occur in twin pregnancies

2. Alkaline phosphatase levels increase to around 150U/L from pre pregnant values of 30- 115U/L
3. Rise in clotting factors
4. Rise in Ceruloplasmin
5. Increase in Transferrin
6. Increase CRP, C3 and C4

Decrease

1. Gallbladder contractility decreases.
2. Uric Acid level decreases
3. Fall in total protein (pre pregnancy 6.7- 8.6g/dl to 5-7 g/dl in third trimester) and Albumin pre pregnancy 4.1- 5.3g/dl to 2.3 -4g/dl in third trimester)
4. Decrease in Antithrombin III and protein S
5. Modest or no fall in platelet levels

No Change

1. Liver transaminase levels (AST, ALT) remain unaltered
2. GGT remains the same
3. Bilirubin level is unaltered by pregnancy
4. Prothrombin time remains the same
5. Blood flow to the liver follows the same pre pregnant pattern.

HYPEREMESIS GRAVIDARUM (0.3- 2%)

Hyperemesis gravidarum is intractable nausea and vomiting during pregnancy which results in fluid and electrolyte imbalance, 5% weight loss or greater, and nutritional deficiency which requires hospital admission. The incidence of hyperemesis gravidarum ranges from 0.3%-2% of all live births It occurs usually between the 4^{th} and 10th week of gestation and resolves by the 20th week.

Risk factors for Hyperemesis include multiple pregnancies , molar pregnancy, trophoblastic disease, hyperemesis in previous pregnancy and fetal abnormalities (triploidy, trisomies and hydrops fetalis). Liver function test abnormalities are also common in hyperemesis and resolve when the vomiting is controlled.

Liver is involved in around 50%-60% of patients with Hyperemesis . Most commonly mild serum aminotransferases elevations are seen, but cases of severe transaminase elevations (alanine aminotransferase (ALT) levels 400 to over 1000 U/L) have been reported. Mild hyperbilirubinemia presenting as mild jaundice can be seen as well. Other complications are disturbances in electrolytes and in water and acid-base balance which usually resolve when treated adequately with hydration.

Available data suggest minimal or no differences between fetuses born to mothers with Hyperemetic and non-Hyperemetic mothers . A large cohort study showed that infants of hyperemetic mothers were found to be of lower birth weights and showed higher rates of being small for gestational age . Treatment of Hyperemesis is mainly supportive. Patients must avoid triggers that aggravate their nausea, and eat small, frequent, meals low in fat. Intravenous fluids, folate and thiamine supplementation and antiemetic therapy may be given. The first line agent is Promethazine, but other medications such as ondansetron, and steroids have been tried.

PREECLAMPSIA /ECLAMPSIA

Preeclampsia is diagnosed by the triad of hypertension, edema, and proteinuria. It is encountered in about 5%-10% of all pregnancies and occurs well into the second trimester or in the third trimester of pregnancy. In pregnancy, hypertension is defined as a systolic pressure more than 140 mmHg and a diastolic

pressure more than 90 mmHg on at least two occasions which are atleast 4 to 6 hours apart in a patient who was previously normotensive.

Proteinuria is quantified as equal to or more than 300 mg of protein in a 24 h urine collection which corresponds to 1+ protein or more on urine dipstick test of two random urine samples collected 4 to 6 h apart. Eclampsia includes all the features of preeclampsia along with neurologic symptoms including headache, visual disturbance and seizures or coma. The risk factors for preeclampsia and eclampsia are nulliparity, extremes of maternal age, insulin resistance, obesity, and infection. The pathology of preeclampsia and eclampsia is involves procoagulant and proinflammatory states that lead to rise in vascular permeability and a systemic inflammatory response that contributes to end organ damage due to hypoperfusion.

Organ dysfunction in severe preeclampsia includes hepatomegaly and hepatocellular injury. 7.5% of all pregnancies are affected, but only 25% of preeclampsia cases turn severe. Liver involvement is not an usual feature and when it occurs symptoms are non-specific.

Eclampsia occurs when grand mal seizures occur. Liver involvement can present with epigastric or right hypochondrial pain, from hepatomegaly causing a stretch of Glisson's capsule. Liver injury is a consequence of vasoconstriction and fibrin precipitation in the liver cells. Complications can include hematoma under Glisson's capsule and hepatic rupture .

The depth of the liver biochemistry abnormalities parallels the maternal risks but not fetal outcomes. Liver function tests cannot exclusively be used to make clinical decisions, as the normal liver function tests does not rule out the disease. An expectant approach can be used till after 34 weeks gestation to limit fetal morbidity.

Laboratory values are altered which include 10- to 20 times elevation in aminotransferases, alkaline phosphatase levels that exceed the normal values observed in pregnancy and bilirubin rises but less than 5 mg/dL. The histology of

the liver generally shows periportal hemorrhage, hepatic sinusoidal deposition of fibrin, hepatocyte necrosis, and in severe conditions liver infarction. It is thought that these changes are mostly because of vasoconstriction of hepatic vasculature.

Microvesicular fatty infiltration has been reported in some cases of preeclampsia, showing a possible overlap with the mechanism of acute fatty liver of pregnancy. Maternal mortality from preeclampsia and eclampsia is much reduced in developed countries but in developing countries it may reach 15%-20%. The fetal mortality rate is low, occurring only in 1%-2% of births. The only effective treatment for preeclampsia is delivering the fetus and placenta. Pharmacological agents administered in preeclampsia are antihypertensives such as labetalol. Magnesium sulphate may be administered if eclampsia develops.

HEMOLYSIS, ELEVATED LIVER TESTS AND LOW PLATELETS (HELLP) (0.5 – 0.9%)

HELLP syndrome is a multi systemic disorder exclusive to pregnancy presenting with hemolysis, elevated liver enzymes, and low platelets. Almost 70% of cases occur antenatally, more during the third trimester of pregnancy.

The pathophysiology of HELLP is alterations in platelet activation, leading to an increase in proinflammatory cytokines, and segmental vasospasm along with vascular endothelial damage. A distant association with a defect in long-chain 3-hydroxyacylcoenzyme A dehydrogenase (LCHAD) enzyme has been suspected, suggesting a possible overlap of HELLP syndrome and acute fatty liver of pregnancy.

Patients usually present with right hypochondrial abdominal pain, vomiting, malaise, and pedal edema. Other associated conditions include antiphospholipid syndrome and diabetes insipidus.

Other late features of HELLP include disseminated intravascular coagulopathy (DIC), placental abruption, pulmonary edema and retinal detachment. Laboratory findings feature hemolysis along with increased bilirubin levels (usually less than 5 mg/dL) and lactate dehydrogenase (LDH) levels greater than 600 IU/L, moderate elevation of aspartate aminotransferase (AST) and ALT levels (200 IU/L to 700 IU/L), and thrombocytopenia (less than 100000/mL). In early presentations, prothrombin time and activated partial thromboplastin time are within normal range, but in later phases, DIC may occur with increasing levels of fibrin degradation products, D-dimer and thrombin- antithrombin complex.

Although HELLP typically presents in late second trimester or third trimester between 28 and 36 weeks of gestation, 30% show symptoms in the first seven days postpartum. The hypertension-related liver diseases often show similar presentations so differentiation is difficult, as usually there is overlap in their features. The diagnosis of HELLP is mostly made by typical laboratory results. Signs of hemolytic anemia and thrombocytopenia with platelets less than 10000/l or abdominal pain radiating to the right shoulder, cross-sectional imaging can exclude hepatic complications more accurately than ultrasound.

Hepatic infarction can be suspected with right upper quadrant pain and fever whereas abdominal distension and shock can occur with hepatic rupture. Surgery is the dictum only for those with enlarging hematomas or showing evidence of rupture along with features of hemodynamic instability.On the other hand, successful percutaneous embolization of the hepatic arteries in stable women has been reported.

Pathophysiology includes intravascular fibrin deposition and sinusoidal obstruction that can result in hepatic infarction. Histologically focal hepatocyte ,peri portalnecrosis is seen, Hemorrhage and fibrin deposits. The maternal mortality from HELLP is reported to 1%. The perinatal mortality rate is around

7%-22% and may be due to premature detachment of placenta, intrauterine asphyxia of the fetus and prematurity.

Other complications of HELLP syndrome are acute renal failure, pulmonary edema, stroke, adult respiratory distress syndrome liver failure, and hepatic infarction. The only curative treatment for HELLP syndrome is delivery.

If the pregnant woman is more than 34 week gestation, immediate termination is the treatment. If the gestational age falls between 24 week and 34 week, corticosteroids are given to accelerate fetal lung maturity in order to prepare for delivery 48 hours later. After delivery, the mother should be closely monitered, as data shows worsening thrombocytopenia and increasing LDH up to 48 hours postpartum. But laboratory values (transaminases, bilirubin and LDH) normalize in 48 hours. For patients with worsening postpartum symptoms of HELLP, antithrombotic agents, plasmapheresis and dialysis may be tried.

ACUTE FATTY LIVER OF PREGNANCY (1 in 10,000 to 1 in 20,000)

Acute fatty liver of pregnancy (AFLP) is a rare and severe maternal illness that occurs in late pregnancy. The incidence is 1 in 10 000 to 1 in 15 000 pregnancies, but it has a maternal mortality rate of 18% and shows a fetal mortality rate of 23%. AFLP is more in nulliparous women and those
with multiple gestation.

The pathogenesis of AFLP is the defect in mitochondrial fatty acid beta-oxidation. In normal circumstances, an women who is heterozygous for enzymatic mutations in fatty acid oxidation enzyme will not show abnormal fatty oxidation. But when the same heterozygous woman carries a fetus which is homozygous for such mutations, fetal fatty acids begin to accumulate and is transferred to the mother's circulation. This extra load of long chain fatty acids and triglyceride accumulation becomes too much for the mother's enzymes to handle and result in hepatic fat deposition and therefore impaired hepatic function in the mother.

A deficiency in long chain 3-hydroxyacyl-CoA dehydrogenase (LCHAD) is associated with the development of AFLP. LCHAD is a constituent of the enzyme complex , the mitochondrial trifunctional protein (MTP), and it is known that the G1528C and E474Q mutations of the MTP play a role in causing LCHAD deficiency that leads to AFLP. Patients with AFLP usually present with a 2 week history of nausea, vomiting, abdominal pain. Jaundice is a common occurrence, and some women have moderate to severe hypoglycemia, hepatic encephalopathy, and coagulopathy.

Around 50% of these patients will show signs of preeclampsia, although hypertension is usually not in the severe range. Laboratory findings include rise in aminotransferase levels, from being mildly elevated to approaching 1000IU/L.

Since AFLP causes significant maternal and fetal morbidity and mortality, early diagnosis must be made. The gold standard test is liver biopsy. Histopathology reveals pale, swollen hepatocytes in the central zones surrounded with microvesicular fatty infiltration which can be identified on frozen section with oil red O staining. Electron microscopy reveals mega-mitochondria and para crystalline mitochondrial inclusions.

Although liver biopsy is the standard investigation to confirm the diagnosis, it is usually not done because of coexisting coagulopathy. Hence the diagnosis of AFLP is usually made on basis of clinical and laboratory findings.

As with most pregnancy-related liver diseases, the definitive treatment of AFLP is delivering of the fetus. Rarely, patients will end up in fulminant hepatic failure necessitating liver transplantation . Careful monitoring of the infant is warranted as there is increased risk of cardiomyopathy, neuropathy, hypoglycemia, hepatic failure, and death associated with fatty acid oxidation defects in newbown

The median gestation at the time of identification is 36 weeks. Risk factors are twin pregnancies and low body mass index. Therefore early recognition, immediate delivery and care are essential to improve maternal and fetal

prognosis, as the postpartum course is dependent on the interval between development of symptoms and termination of the gestation. If hepatic function is not rapidly corrected, liver transplantation offers the patient the best chance for survival. Concomitant preeclampsia is seen in one half of the affected women. Aminotransferase elevations and hyperbilirubinemia are typically seen. Hepatic failure can show signs of hepatic dysfunction such as encephalopathy, coagulopathy, and hypoglycemia. Renal dysfunction and pancreatitis are also common. The Swansea Criteria is a combination symptoms and laboratory derangements . These criteria has been supported in a large cohort in England, where the incidence of AFLP is 5 cases per 100,000 maternities. The Swansea Criteria has agreement with the clinical diagnosis of AFLP. Although there was only 1 death in this study group 65% were admitted to an intensive care unit . When the Swansea Criteria were applied in a large group of women with pregnancy related liver disease liver biopsy was taken, this offered an 85% positive predictive value and a total 100% negative predictive value for hepatic microvesicular fatty infiltration.

INTRAHEPATIC CHOLESTASIS OF PREGNANCY (1.5- 2%)

Intrahepatic cholestasis of pregnancy (ICP), also known as obstetric cholestasis, is a rare pregnancy specific liver condition that occurs in the late second or third trimester and has a prevalence of about 1/1000 to 1/10 000. IHCP is the most common liver disease in pregnancy with prevalence ranging between 0.3 and 5.6%. It is significantly more common in South Asia, South America and Scandinavian countries.

ICP is more common in women of advanced maternal age, multiparous women, and in women with a personal history of cholestasis with oral contraceptive use . ICP has good prognosis, but it is associated with increased fetal

morbidity and mortality, particularly from chronic placental insufficiency, preterm labor, fetal distress, and intrauterine death.

The cause of ICP is likely multifactorial. Mutations in the phospholipid translocator known as the ATPcassette transporter B4 (ABCB4) or multidrug resistant protein-3 (MDR3) are associated with the development of ICP. Changes caused by these genetic mutations lead to increased sensitivity to estrogen, which impairs the sulfation and transportation of bile acids. Estrogens are thought to act on hepatocytes by decreasing membrane permeability and bile acid uptake by the liver. The maternal-to fetal transfer of bile acids across the placenta becomes impaired, leading to potentially toxic bile acid levels in the fetus. The elevation in bile acid levels is also thought possibly to affect myometrial contractility and to cause vasoconstriction of chorionic veins in the placenta, which may contribute to preterm deliveries and fetal distress seen in ICP

Pruritis is the the classic symptom that usually begins in the second or third trimester. It usually occurs in the palms and soles and may progress to the rest of the body, and worse at night. Pruritus may be severe in some cases.

Jaundice occurs in approximately 10%-25% of patients and may appear within the first four weeks of the onset of pruritus . ICP women has more Cholelithiasis and cholecystitis . Abnormal laboratory findings include elevated total bile acid levels up to 10- to 25-fold, with an increase in cholic acid and a decrease in chenodeoxycholic acid leading to a marked elevation in the cholic/ chenodeoxycholic acid ratio.

Bilirubin levels may be elevated, but are usually less than 6 mg/dL. Serum alkaline phosphatase levels may also be elevated, but this is usually less helpful to follow given typical alkaline phosphatase elevations seen in pregnancy. Liver biopsy is usually not required to make the diagnosis of ICP.

The treatment of choice for ICP is ursodeoxycholic acid (UDCA), which helps to relieve pruritus and improve liver function test abnormalities. Other medications, such as cholestyramine and S-adenosyl-L-methionine, have been

associated with improving pruritus and normalizing biochemical profiles, but studies have found UCDA to be superior over cholestyramine and S-adenosyl- L-methionine . Dexamethasone has also been used, but has shown to be much less effective in reducing bile acids and bilirubin and ineffective in relieving pruritus . Antihistamines are frequently used to alleviate pruritus, and vitamin K and other fat-soluble vitamin supplementation should also be administered if fat malabsorption is suspected.

CHRONIC LIVER DISEASE AND PORTAL HYPERTENSION

Women with significant hepatic dysfunction has decreased fertility due to hypothalamic-pituitary dysfunction. However, cirrhosis is not a contraindication, as pregnancy may be tolerated if cirrhosis is well-compensated and without features of portal hypertension . Portal hypertension leads to increased maternal complications, including variceal hemorrhage , hepatic failure, encephalopathy, jaundice, malnutrition , and splenic artery
aneurysm .

Bleeding from esophageal varices has been reported in 20%-25% of pregnant women with cirrhosis . All pregnant women with cirrhosis should be screened for varices starting in the second trimester and started on beta-blockers if indicated.

The treatment of variceal bleeding consists of both endoscopic and pharmacologic treatment but in this time and age the management for varices is prophylactic with beta blockers or endoscopic variceal ligation. However, vasopressin is avoided as it has been shown to cause placental ischemia, necrosis, and amputation of fetal digits and is contraindicated in pregnancy.

There are no published systematic reviews on the management of cirrhosis or portal hypertension during pregnancy, likely because of the low prevalence of cirrhosis in women of reproductive age and reduced fertility of women with

cirrhosis. Pregnant women with cirrhosis should ideally be managed in a multidisciplinary setting with maternal–fetal medicine along with gastroenterology. Pregnancy in women with underlying cirrhosis has been associated with an increase in prematurity, spontaneous abortions, and maternal–fetal mortality.

Non-cirrhotic portal hypertension has better outcomes than cirrhotics with portal hypertension . Variceal bleeding is the most common complication of portal hypertension during pregnancy, with an increased risk at delivery and the second trimester, aggravated by an increase in intravascular volume, compression from the gravid uterus. Up to 30% of cirrhotic pregnant women bleed from esophageal varices during pregnancy, and the risk of variceal bleeding increases up to 50–78% if there are pre-existing varices . Each episode of variceal bleeding leads to maternal mortality rates as high as 20–50%, with an even higher risk of fetal loss .

Variceal bleeding during pregnancy is managed very similarly to variceal bleeding in general given the acute and life-threatening nature of the event, with a focus on endoscopic hemostasis and supportive care for the mother and fetus. Octreotide is a pregnancy category B drug and appears to be safe as an adjunct treatment in acute variceal bleeding along with antibiotics.

Endoscopy during pregnancy appears safe, but must be considered carefully in terms of the indication for endoscopy, the risks vs benefit evaluation and whether it will lead to management changes . Given the risks of variceal bleeding in cirrhotic women during pregnancy, the significantly increased mortality associated with such bleeding, and the opportunity to intervene if varices are identified preemptively, the indications for screening for esophageal varices appears to have at least a moderate indication. Considered against the small, but not insignificant risk of sedation and endoscopy, the benefits appear to outweigh the risks.

Timing of screening for esophageal varices appears most prudent in the second trimester, after organogenesis is complete in the first trimester and before

the greatest risk of bleeding at delivery. Despite acceptance of band ligation and beta-blockers as firstline management of esophageal varices for non-pregnant patients, there are limited data on their efficacy and safety in pregnancy . Propranolol is a pregnancy category C drug, but has been used to treat fetal arrhythmias as well as maternal conditions such as thyrotoxicosis, arrhythmias, or hypertension.

There are risks of intra uterine growth retardation, neonatal bradycardia, and hypoglycemia, but propranolol appears overall to be safe in pregnant patients. Traditionally, vaginal delivery with a short second stage of labour with forceps. Cesarean sections may be required only for obstetric indications, but carries an increased risk of bleeding complications from the surgical site in the setting of portal hypertension.

WILSON DISEASE (1 in 30,000 to 1 in 1,00,000)

Wilson disease lead to reduced fertility as copper deposition in the uterus may interfere with embryo implantation leading to an increase in miscarriages and spontaneous abortions . Pregnancy in general does not appear to change the course of Wilson disease progression

Pregnancy in general does not appear to change the course of WD progression . However, treatment discontinuation or a lack of treatment has been reported to lead to disease flares with attendant risk of hepatic decompensation or liver failure. There is one recent systematic review on the treatment of WD in general, but no reports specific to treatment for WD during pregnancy Practice guidelines for WD recommends continuing treatment during pregnancy, but reducing penicillamine or trientine doses by 25–50% to promote wound healing in the event that a cesarean section is needed . There is inadequate data to make recommendations on a preferred treatment for WD during pregnancy, between

penicillamine, trientine, or zinc. Some data on conversion to zinc therapy during pregnancy has also been reported .

There are also multiple case reports of fetal myelosuppression or embryopathy associated with penicillamine treatment during pregnancy for Wilson disease. On the other hand, treatment discontinuation or lack of treatment for WD can not only lead to maternal hepatic decompensation but can also lead to copper deposition in the placenta and fetal liver, damaging the fetus along with recognized risks of maternal hepatic decompensation. Th e risks of treatment discontinuation or lack of treatment for WD during pregnancy appears to outweigh the potential risks of treatment. The data to recommended dose reduction of penicillamine in anticipation of possible caesarean section appears to be very limited.

AUTOIMMUNE LIVER DISEASE (1 in 1000 to 1 in 10,000)

Autoimmune diseases of all types including autoimmune hepatitis are more common in women then in men. In women, classic (type 1) autoimmune hepatitis typically presents around the expected time of menarche but is associated with amenorrhea.

Immunosuppressive therapy is highly effective in controlling the disease in most patients; treated women who subsequently conceive a child should continue taking immunosuppressive medications during pregnancy. The doses of azathioprine prescribed as part of standard treatment regimens are belived not to be teratogenic.

Occasionally autoimmune liver disease will worsen during the postpartum period when the physiologic immunosuppression of pregnancy resolves. For this reason affected patients should have frequent measurements of serum aminotransferase levels for approximately 6 months after delivery.

Primary biliary cirrhosis

Older literature suggested poor outcomes of pregnancy in patients with PBC . As patients with PBC tend to present at an older age aft er the usual child-bearing age, and as women with PBC were discouraged in the past from pursuing pregnancy, there is an extremely limited number of studies on PBC and pregnancy. However, more recent studies have reported good maternal and fetal outcomes . PBC has been associated with disease fl are aft er delivery. UDCA is a pregnancy category B drug that is generally recommended for PBC .

Studies on the use of UDCA treatment for PBC during pregnancy have been limited, especially in the fi rst trimester. Similar to the situation with AIH, the potential risks of UDCA during pregnancy appear small compared with the potential positive effect of treatment on maternal and fetal outcomes. With growing evidence that UDCA is safe during pregnancy in other diseases such as intrahepatic cholestasis of pregnancy, and the unlikely scenario that large scale studies will be performed on the efficacy and safety of UDCA during pregnancy for PBC, it appears prudent to recommend continuation of UDCA for PBC during pregnancy at this time

MATERIALS AND METHODS

Study design

The study was a single institution prospective randomized study conducted in Madurai Medical College over a period of six months.

A total of 40 patients were included in the study. All the patients were thoroughly examined and case sheets were written in the same fashion to facilitate comparison.

All of them underwent clinical examination and an algorithm of investigations required to approach the diagnosis of non infective jaundice.

The patients were subjected to various forms of treatment available in the tertiary care centre.

The protocol was approved by the Ethical Review Board of the institute and an informed consent was obtained from all patients.

PARTICIPANTS

The patients were selected from those attending the obstetric department at the hospital with no specific limitation imposed on age and gestational age.

All the patients were diagnosed to have jaundice clinically with the exclusion criteria being infective hepatitis in pregnancy.

METHODS

All the patients satisfying the inclusion criteria were carefully evaluated in terms of clinical examination, recording of BP, obstetric examination, laboratory investigations including Hb, platelet, urine albumin, clotting time, renal function test, liver function test, serum uric acid, LDH, viral markers, ultrasonogram.

The patients were followed through the course of the disease taking into account the complications of the disease, the management protocols including the obstetric management and blood product transfusion, the maternal and fetal outcomes and the recovery time.

The maternal outcome was derived from the rate of complications, ICU stay, recovery interval and maternal mortality.

The fetal outcome was studied by the number of live births, NICU admission and perinatal mortality.

The patients were followed up for the duration of hospital stay and the recovery time was recorded.

STATISTICS

Tables

		Gravida				Total	
		Multi		Primi		Count	Col %
		Count	Col %	Count	Col %		
Diagnosis	AFLP	0	.0%	3	12.0%	3	7.5%
	Cirrhosis	2	13.3%	1	4.0%	3	7.5%
	CRIGGLER NAJAR SYN	0	.0%	1	4.0%	1	2.5%
	Hellp	7	46.7%	7	28.0%	14	35.0%
	Partial hellp	6	40.0%	13	52.0%	19	47.5%
Diagnosis	Hellp	13	86.7%	20	80.0%	33	82.5%
	Non-Hellp	2	13.3%	5	20.0%	7	17.5%
Total		15	100.0%	25	100.0%	40	100.0%

Diagnosis * Gravida

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.706(a)	4	.319
Likelihood Ratio	5.999	4	.199
Linear-by-Linear Association	.054	1	.816
N of Valid Cases	40		

a 6 cells (60.0%) have expected count less than 5. The minimum expected count is .38.

Diagnosis * Gravida

Chi-Square Tests					
	Value	df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	.289(b)	1	.591		
Continuity Correction(a)	.012	1	.914		
Likelihood Ratio	.298	1	.585		
Fisher's Exact Test				.691	.467
Linear-by-Linear Association	.281	1	.596		
N of Valid Cases	40				
a Computed only for a 2x2 table					
b 2 cells (50.0%) have expected count less than 5. The minimum expected count is 2.63.					

	Gravida						**Statistical inference**
	Multi		**Primi**		**Total**		
Diagnosis							
AFLP	0	.0%	3	12.0%	3	7.5%	
Cirrhosis	2	13.3%	1	4.0%	3	7.5%	X^2=4.706 Df=4
CRIGGLER NAJAR SYN	0	.0%	1	4.0%	1	2.5%	.319>0.05 Not Significant
Hellp	7	46.7%	7	28.0%	14	35.0%	
Partial hellp	6	40.0%	13	52.0%	19	47.5%	
Diagnosis							
Hellp	13	86.7%	20	80.0%	33	82.5%	X^2=.289 Df=1
Non-Hellp	2	13.3%	5	20.0%	7	17.5%	.591>0.05 Not Significant
Total	**15**	**100.0%**	**25**	**100.0%**	**40**	**100.0%**	

		Complication				Total	
		Absent		Present		Count	Col %
		Count	Col %	Count	Col %		
BP	Below 130/90	13	50.0%	2	14.3%	15	37.5%
	Above 130/90	13	50.0%	12	85.7%	25	62.5%
Total		26	100.0%	14	100.0%	40	100.0%

Crosstabs

Chi-Square Tests					
	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	4.952(b)	1	.026		
Continuity Correction(a)	3.546	1	.060		
Likelihood Ratio	5.398	1	.020		
Fisher's Exact Test				.040	.027
Linear-by-Linear Association	4.829	1	.028		
N of Valid Cases	40				

a Computed only for a 2x2 table

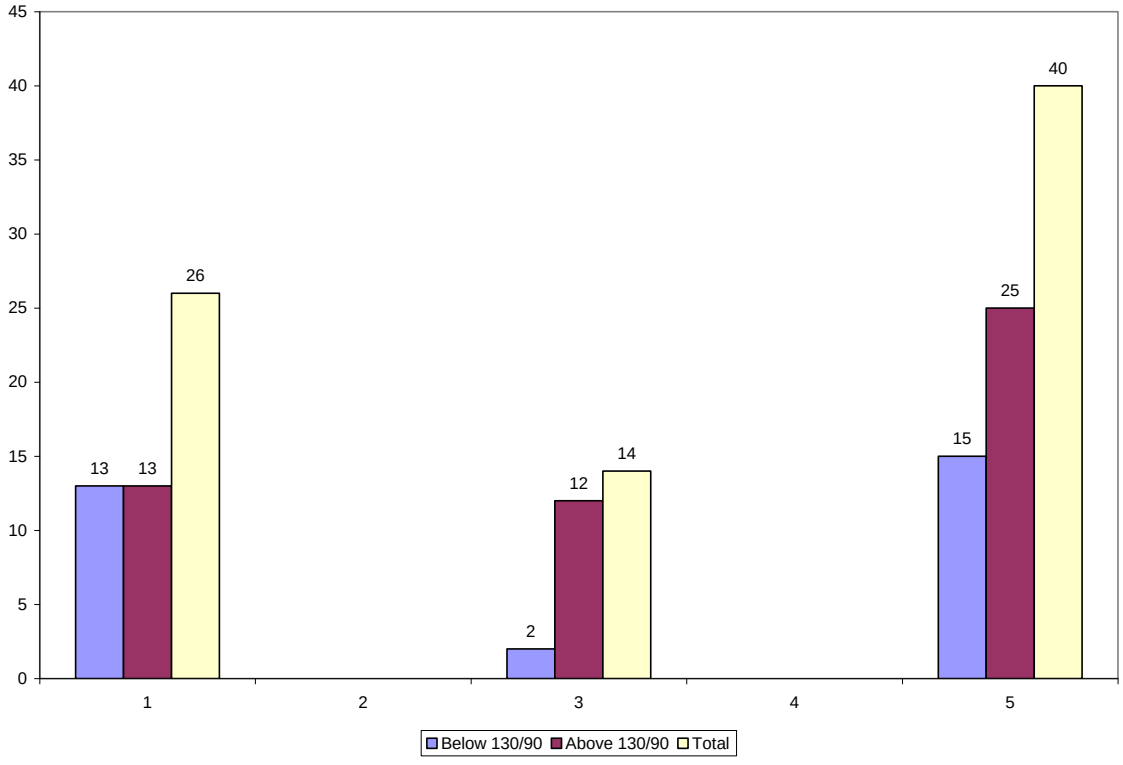
b 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.25.

Table No – 1

Tables

	Complication						Statistical inference
	Absent		Present		Total		
BP							
Below 130/90	13	50.0%	2	14.3%	15	37.5%	X^2=4.952 Df=1
Above 130/90	13	50.0%	12	85.7%	25	62.5%	.026<0.05 Significant
Total	**26**	**100.0%**	**14**	**100.0%**	**40**	**100.0%**	

Diagrams No – 1



T-Test

Group Statistics					
	Complication	N	Mean	Std. Deviation	Std. Error Mean
Bilirubin	Absent	26	2.5462	1.35506	.26575
	Present	14	4.1643	2.51323	.67169

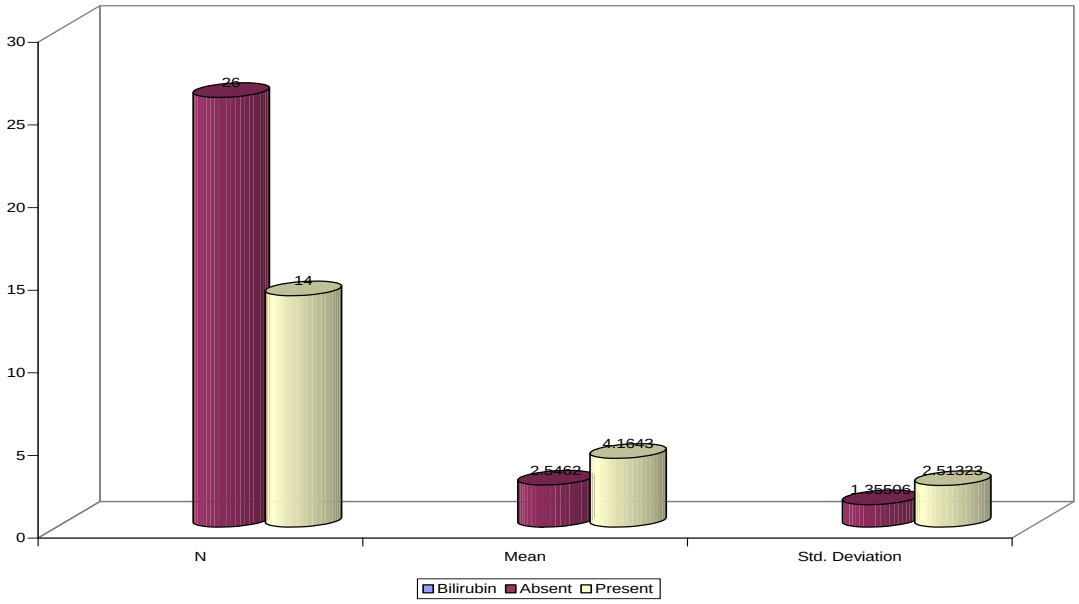
Independent Samples Test										
		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	T	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
Bilirubin	Equal variances assumed	3.016	.091	-2.659	38	.011	-1.6181	.60844	-2.84986	-.38640
	Equal variances not assumed			-2.240	17.170	.039	-1.6181	.72235	-3.14101	-.09525

Table No – 2

T-Test

Complication	**N**	**Mean**	**Std. Deviation**	**Statistical inference**
Bilirubin				
Absent	26	2.5462	1.35506	T=-2.659 Df=38 .011<0.05 Significant
Present	14	4.1643	2.51323	

Diagrams No – 2



T-Test

Group Statistics

	Complication	N	Mean	Std. Deviation	Std. Error Mean
HB	Absent	26	8.0077	1.06393	.20865
	Present	14	7.3571	.93126	.24889

Independent Samples Test

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
HB	Equal variances assumed	.196	.660	1.923	38	.062	.6505	.33829	-.03428	1.33538
	Equal variances not assumed			2.003	29.991	.054	.6505	.32478	-.01275	1.31385

Complication	N	Mean	Std. Deviation	Statistical inference
HB				
Absent	26	8.0077	1.06393	T=1.923 Df=38 .062>0.05
Present	14	7.3571	.93126	Not Significant

Tables

		Complication				Total	
		Absent		Present		Count	Col %
		Count	Col %	Count	Col %		
Pre Eclamptic features	Absent	0	.0%	2	14.3%	2	5.0%
	Present	26	100.0%	12	85.7%	38	95.0%
Total		26	100.0%	14	100.0%	40	100.0%

Crosstabs

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	3.910(b)	1	.048		
Continuity Correction(a)	1.481	1	.224		
Likelihood Ratio	4.398	1	.036		
Fisher's Exact Test				.117	.117
Linear-by-Linear Association	3.812	1	.051		
N of Valid Cases	40				

a Computed only for a 2x2 table

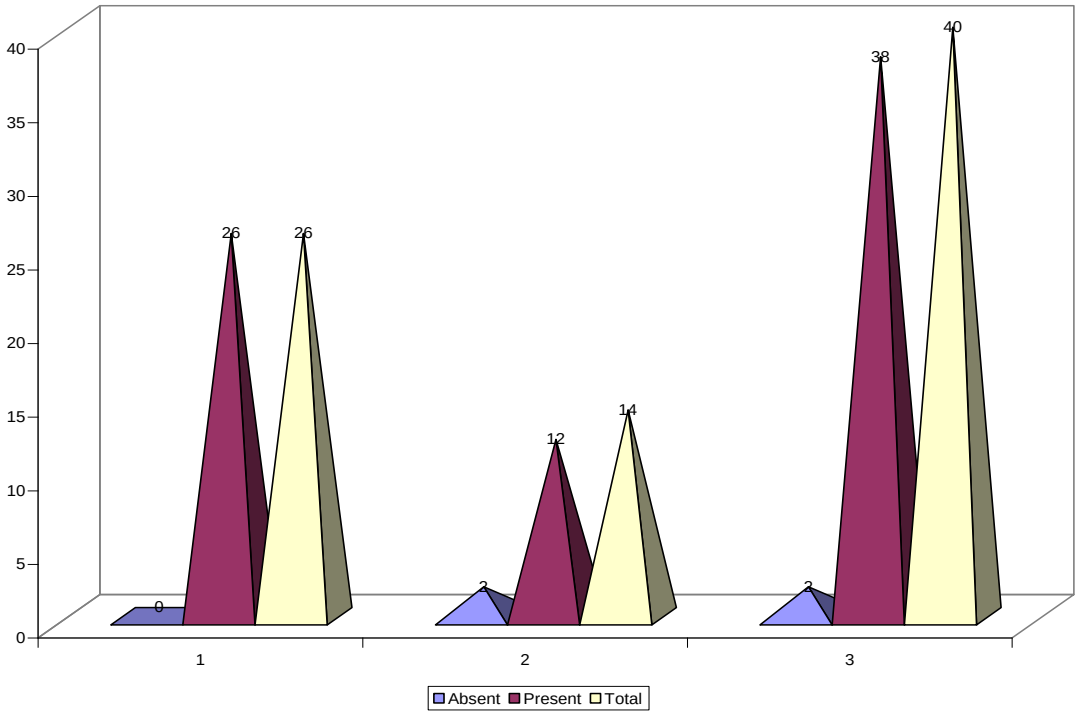
b 2 cells (50.0%) have expected count less than 5. The minimum expected count is .70.

Table No – 3

Tables

	Complication						Statistical inference
	Absent		Present		Total		
Pre Eclamptic features							
Absent	0	.0%	2	14.3%	2	5.0%	X^2=3.910 Df=1
Present	26	100.0%	12	85.7%	38	95.0%	.048<0.05 Significant
Total	**26**	**100.0%**	**14**	**100.0%**	**40**	**100.0%**	

Diagrams No – 3



Tables

	Complication		Total	
	Absent	Present	Count	Col %

		Count	Col %	Count	Col %		
Diagnosis Delivery intervel	<24	21	80.8%	4	28.6%	25	62.5%
	>24	5	19.2%	10	71.4%	15	37.5%
Total		26	100.0%	14	100.0%	40	100.0%

Chi-Square Tests

	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	10.579(b)	1	.001		
Continuity Correction(a)	8.469	1	.004		
Likelihood Ratio	10.717	1	.001		
Fisher's Exact Test				.002	.002
Linear-by-Linear Association	10.314	1	.001		
N of Valid Cases	40				

a Computed only for a 2x2 table

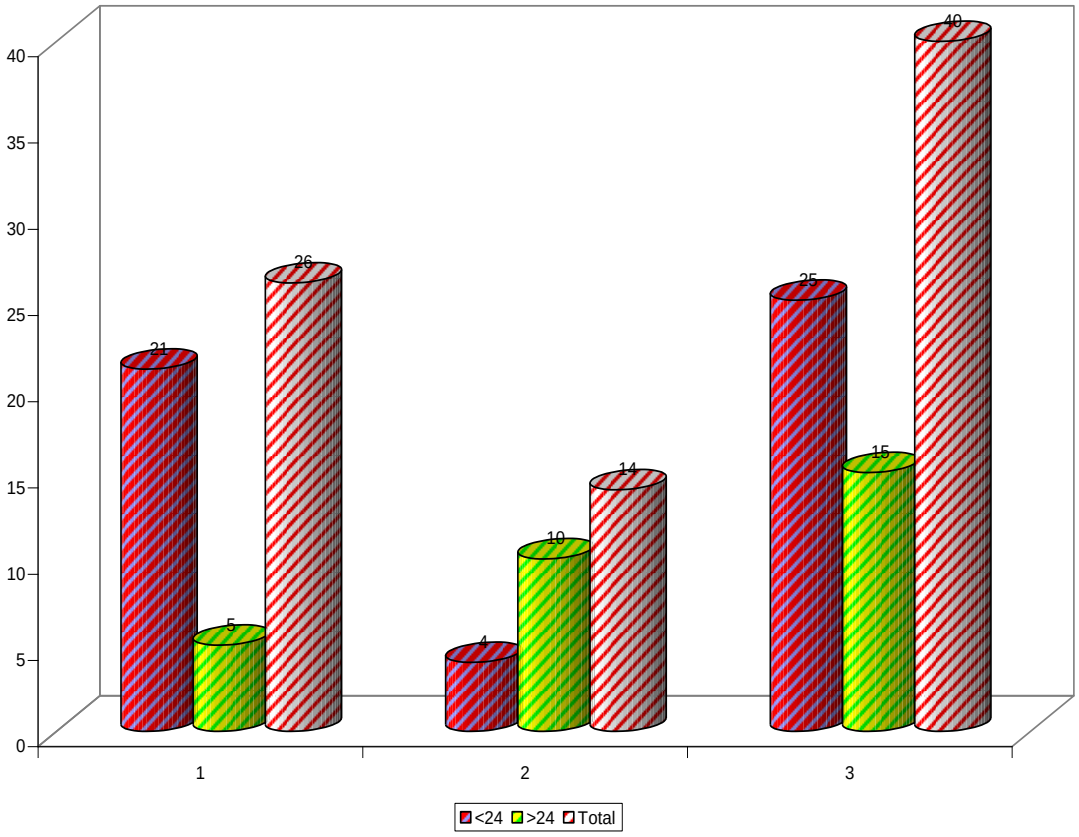
b 0 cells (.0%) have expected count less than 5. The minimum expected count is 5.25.

Table No – 4

Tables

	Complication						**Statistical inference**
	Absent		**Present**		**Total**		
Diagnosis Delivery intervel							
<24	21	80.8%	4	28.6%	25	62.5%	X^2=10.579 Df=1
>24	5	19.2%	10	71.4%	15	37.5%	.001<0.05 Significant
Total	**26**	**100.0%**	**14**	**100.0%**	**40**	**100.0%**	

Diagrams No – 4



Tables

		Gravida				Total	
		Multi		Primi		Count	Col %
		Count	Col %	Count	Col %		
Maternal Mortality	Absent	13	86.7%	24	96.0%	37	92.5%
	Present	2	13.3%	1	4.0%	3	7.5%
Total		15	100.0%	25	100.0%	40	100.0%

Maternal Mortality * Gravida

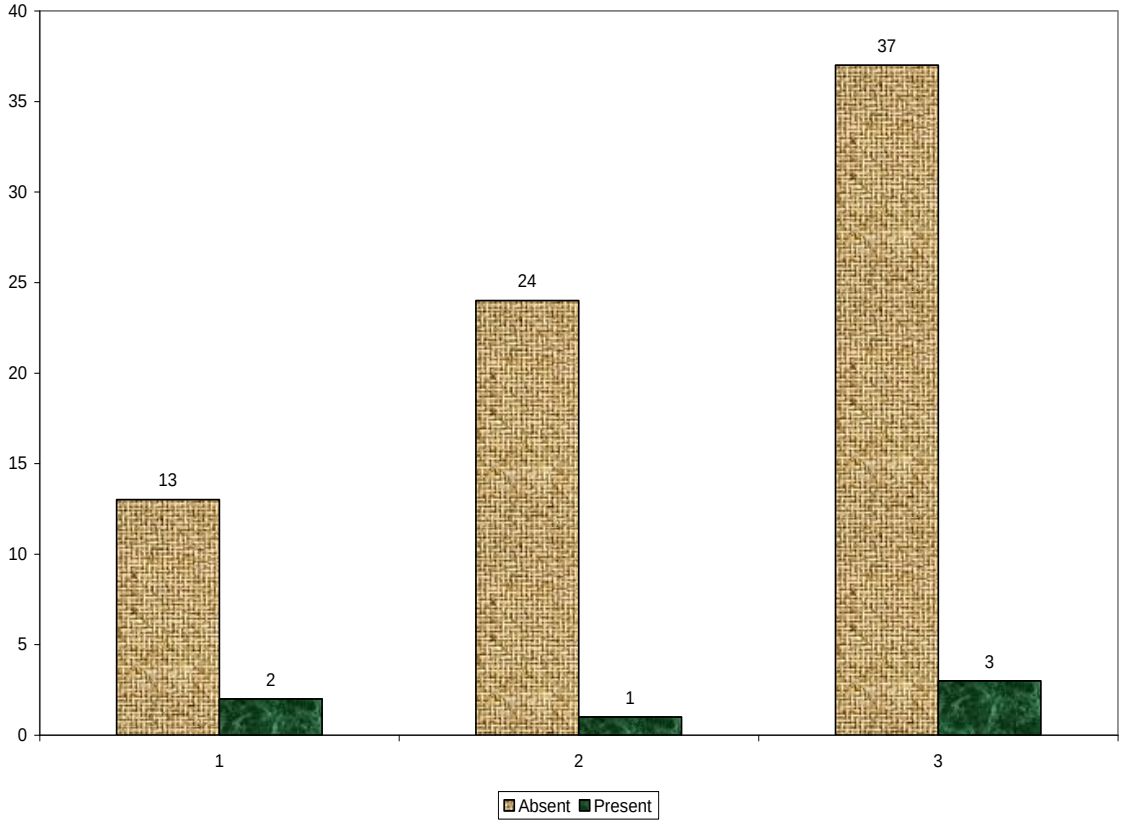
Chi-Square Tests					
	Value	Df	Asymp. Sig. (2-sided)	Exact Sig. (2-sided)	Exact Sig. (1-sided)
Pearson Chi-Square	1.177(b)	1	.278		
Continuity Correction(a)	.216	1	.642		
Likelihood Ratio	1.133	1	.287		
Fisher's Exact Test				.545	.312
Linear-by-Linear Association	1.148	1	.284		
N of Valid Cases	40				
a Computed only for a 2x2 table					
b 2 cells (50.0%) have expected count less than 5. The minimum expected count is 1.13.					

Table No – 5

Tables

	Gravida						**Statistical inference**
	Multi		**Primi**		**Total**		
Maternal Mortality							
Absent	13	86.7%	24	96.0%	37	92.5%	X^2=5.177 Df=1
Present	2	13.3%	1	4.0%	3	7.5%	.027<0.05 Significant
Total	**15**	**100.0%**	**25**	**100.0%**	**40**	**100.0%**	

Diagrams No – 5



Tables

		Delivery recovery time						Total	
		NA		<7 days		Above 7days		Count	Col %
		Count	Col %	Count	Col %	Count	Col %		
BP	Below 130/90	1	16.7%	10	55.6%	4	25.0%	15	37.5%
	Above 130/90	5	83.3%	8	44.4%	12	75.0%	25	62.5%
Total		6	100.0%	18	100.0%	16	100.0%	40	100.0%

Crosstabs

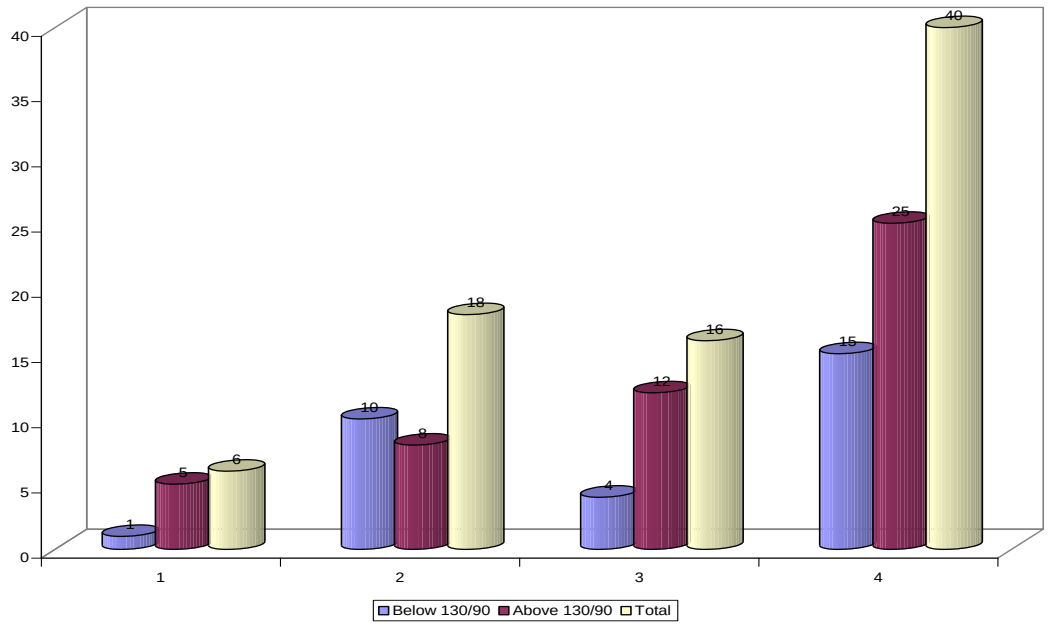
Chi-Square Tests			
	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	4.681(a)	2	.096
Likelihood Ratio	4.793	2	.091
Linear-by-Linear Association	.120	1	.729
N of Valid Cases	40		
a 2 cells (33.3%) have expected count less than 5. The minimum expected count is 2.25.			

Table No – 6

Tables

	Delivery recovery time								**Statistical inference**
	NA		**<7 days**		**Above 7days**		**Total**		
BP									
Below 130/90	1	16.7%	10	55.6%	4	25.0%	15	37.5%	X^2=6.681 Df=2 .017<0.05 Significant
Above 130/90	5	83.3%	8	44.4%	12	75.0%	25	62.5%	
Total	**6**	**100.0%**	**18**	**100.0%**	**16**	**100.0%**	**40**	**100.0%**	

\Diagrams No – 6



Tables

		NICU						Total	
		NA		NICU		No NICU		Count	Col %
		Count	Col %	Count	Col %	Count	Col %		
Gestational age	<37	5	55.6%	19	70.4%	4	100.0%	28	70.0%
	>37	4	44.4%	8	29.6%	0	.0%	12	30.0%
Total		9	100.0%	27	100.0%	4	100.0%	40	100.0%

Crosstabs

Chi-Square Tests

	Value	df	Asymp. Sig. (2-sided)
Pearson Chi-Square	2.610(a)	2	.271
Likelihood Ratio	3.688	2	.158
Linear-by-Linear Association	2.345	1	.126
N of Valid Cases	40		

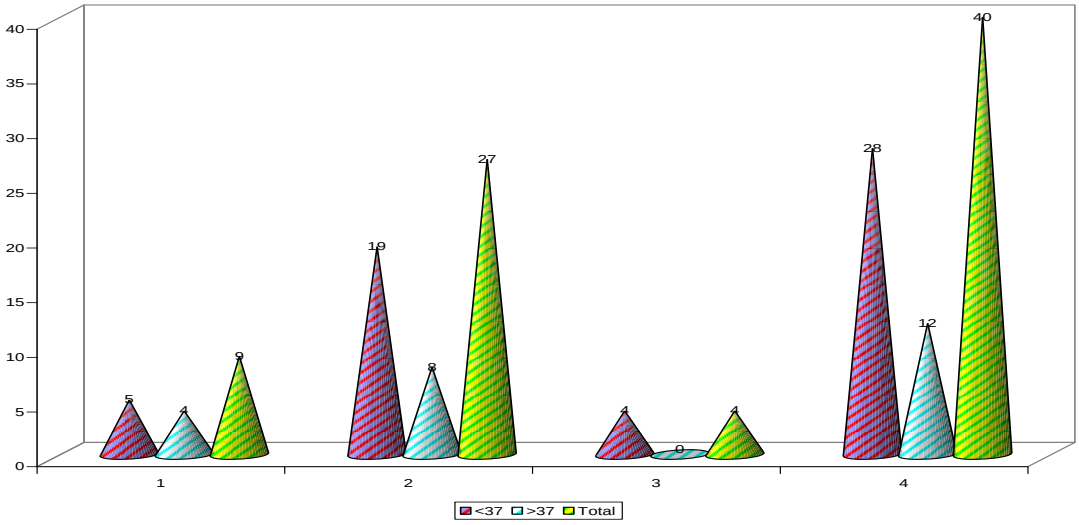
a 3 cells (50.0%) have expected count less than 5. The minimum expected count is 1.20.

Table No – 7

Tables

	NICU								Statistical inference
	NA		NICU		No NICU		Total		
Gestational age									
<37	5	55.6%	19	70.4%	4	100.0%	28	70.0%	X^2=8.932 Df=2 .027<0.05 Significant
>37	4	44.4%	8	29.6%	0	.0%	12	30.0%	
Total	**9**	**100.0%**	**27**	**100.0%**	**4**	**100.0%**	**40**	**100.0%**	

Diagrams No – 7



Frequency Table

Age					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	22	5	12.5	12.5	12.5
	23	2	5.0	5.0	17.5
	24	4	10.0	10.0	27.5
	25	3	7.5	7.5	35.0
	26	6	15.0	15.0	50.0
	27	7	17.5	17.5	67.5
	28	2	5.0	5.0	72.5

	29	1	2.5	2.5	75.0
	30	4	10.0	10.0	85.0
	31	1	2.5	2.5	87.5
	32	2	5.0	5.0	92.5
	33	2	5.0	5.0	97.5
	34	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Frequency Table

Age

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
22	5	12.5
23	2	5.0
24	4	10.0
25	3	7.5
26	6	15.0
27	7	17.5
28	2	5.0
29	1	2.5
30	4	10.0
31	1	2.5
32	2	5.0
33	2	5.0
34	1	2.5

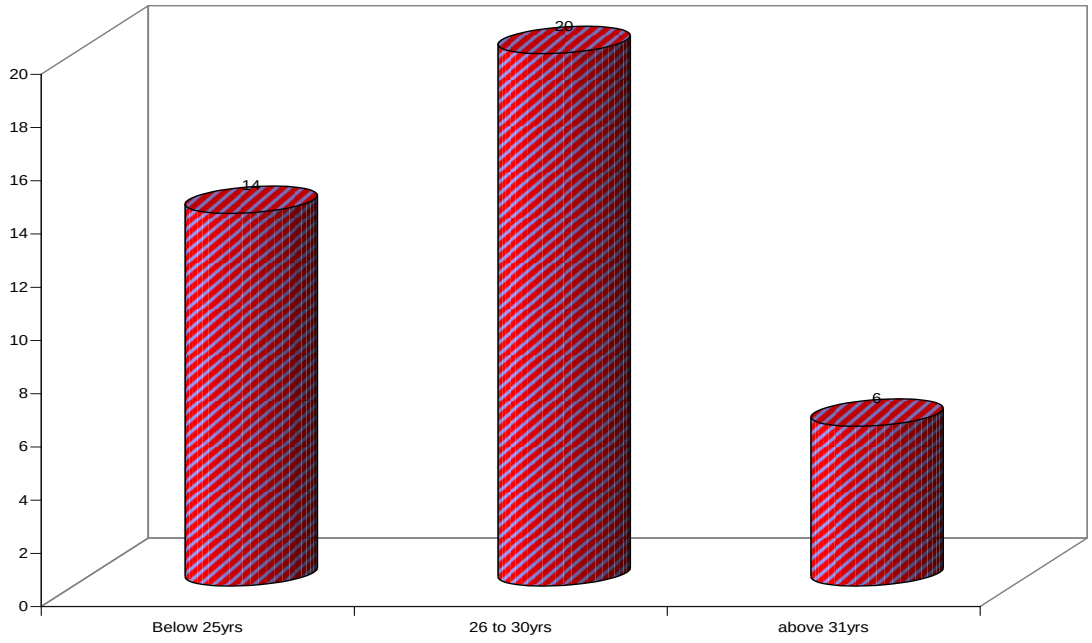
Frequency Table

Table No – 8

Age

Particulars	No.of respondents (n=40)	Percentage (100%)
Below 25yrs	14	35.0
26 to 30yrs	20	50.0
above 31yrs	6	15.0

Diagrams No – 8



Age					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Below 25yrs	14	35.0	35.0	35.0
	26 to 30yrs	20	50.0	50.0	85.0
	above 31yrs	6	15.0	15.0	100.0
	Total	40	100.0	100.0	

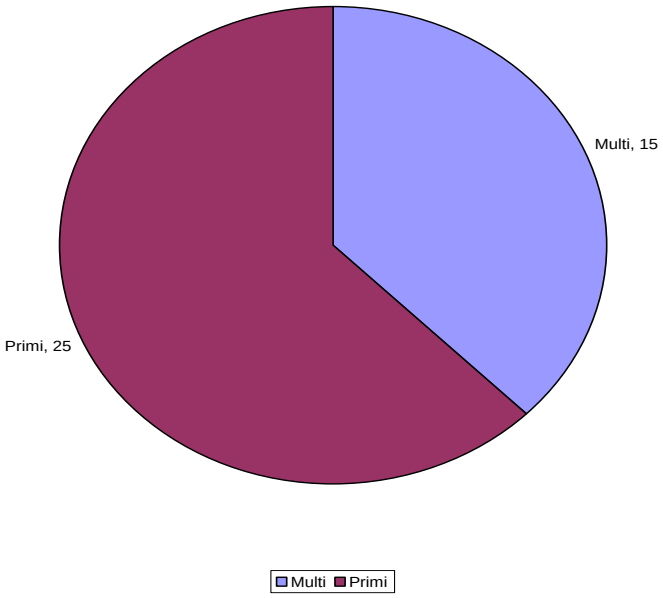
Gravida					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Multi	15	37.5	37.5	37.5
	Primi	25	62.5	62.5	100.0
	Total	40	100.0	100.0	

Table No – 9

Gravida

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Multi	15	37.5
Primi	25	62.5

Diagrams No – 9



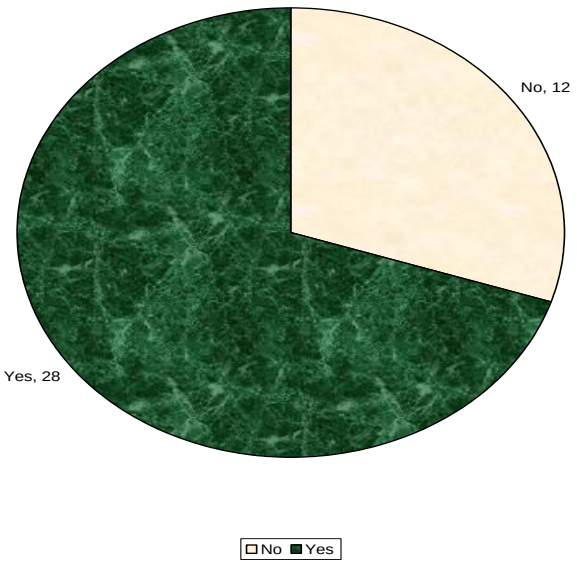
Gestational age <37					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	12	30.0	30.0	30.0
	Yes	28	70.0	70.0	100.0
	Total	40	100.0	100.0	

Table No – 10

Gestational age <37

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
No	12	30.0
Yes	28	70.0

Diagrams No – 10



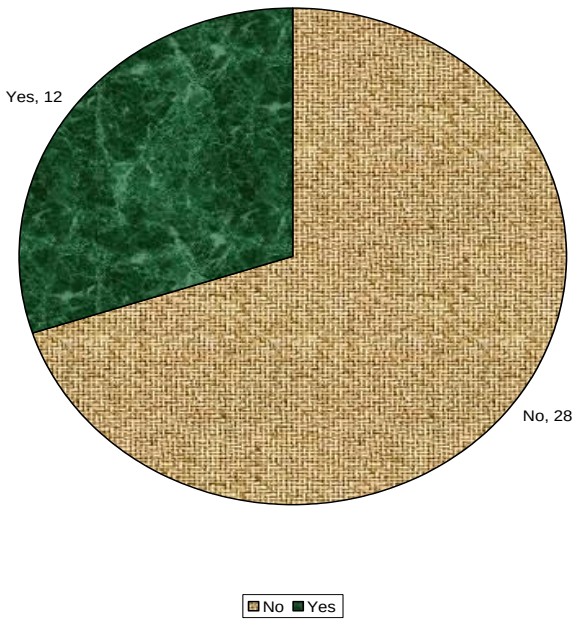
Gestational age >37					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	28	70.0	70.0	70.0
	Yes	12	30.0	30.0	100.0
	Total	40	100.0	100.0	

Table No – 11

Gestational age >37

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
No	28	70.0
Yes	12	30.0

Diagrams No – 11



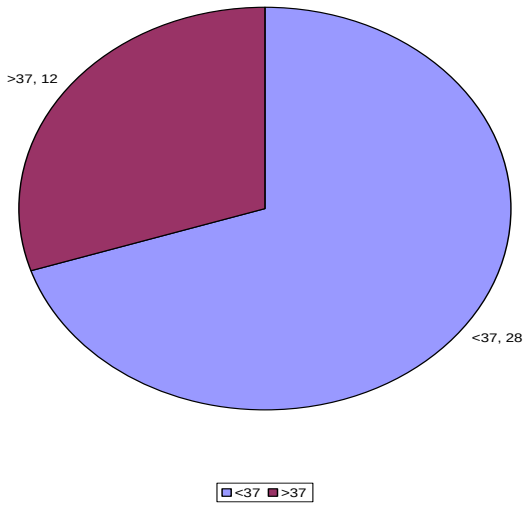
Gestational age					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<37	28	70.0	70.0	70.0
	>37	12	30.0	30.0	100.0
	Total	40	100.0	100.0	

Table No – 12

Gestational age

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
<37	28	70.0
>37	12	30.0

Diagrams No – 12



BP1					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	110	4	10.0	10.0	10.0
	120	6	15.0	15.0	25.0

	130	6	15.0	15.0	40.0
	140	9	22.5	22.5	62.5
	150	5	12.5	12.5	75.0
	160	10	25.0	25.0	100.0
	Total	40	100.0	100.0	

BP1

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
110	4	10.0
120	6	15.0
130	6	15.0
140	9	22.5
150	5	12.5
160	10	25.0

BP2

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	70	3	7.5	7.5	7.5
	80	2	5.0	5.0	12.5
	90	27	67.5	67.5	80.0
	100	8	20.0	20.0	100.0
	Total	40	100.0	100.0	

BP2

Particulars	No.of respondents (n=40)	Percentage (100%)
70	3	7.5
80	2	5.0
90	27	67.5
100	8	20.0

BP

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	110/70	3	7.5	7.5	7.5
	110/80	1	2.5	2.5	10.0
	120/90	6	15.0	15.0	25.0
	130/100	1	2.5	2.5	27.5
	130/80	1	2.5	2.5	30.0
	130/90	4	10.0	10.0	40.0
	140/90	9	22.5	22.5	62.5
	150/100	1	2.5	2.5	65.0
	150/90	4	10.0	10.0	75.0
	160/100	8	20.0	20.0	95.0
	160/90	2	5.0	5.0	100.0
	Total	40	100.0	100.0	

BP

Particulars	No.of respondents (n=40)	Percentage (100%)
110/70	3	7.5
110/80	1	2.5
120/90	6	15.0
130/100	1	2.5
130/80	1	2.5
130/90	4	10.0
140/90	9	22.5
150/100	1	2.5
150/90	4	10.0
160/100	8	20.0
160/90	2	5.0

BP

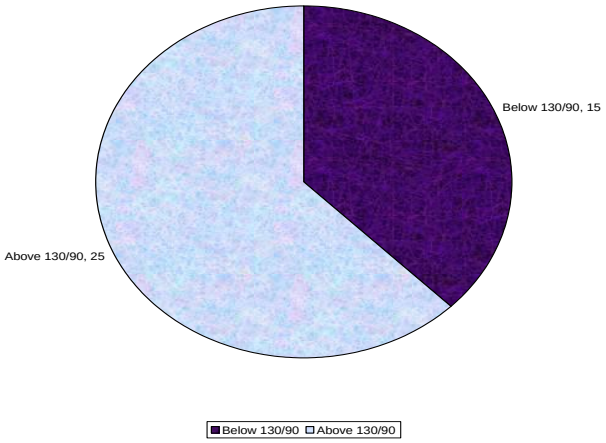
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Below 130/90	15	37.5	37.5	37.5
	Above 130/90	25	62.5	62.5	100.0
	Total	40	100.0	100.0	

Table No – 13

BP

Particulars	No.of respondents (n=40)	Percentage (100%)
Below 130/90	15	37.5
Above 130/90	25	62.5

Diagrams No – 13



Pedal edema					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	5	12.5	12.5	12.5
	Present	35	87.5	87.5	100.0
	Total	40	100.0	100.0	

Pedal edema

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	5	12.5
Present	35	87.5

Icteric					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Present	40	100.0	100.0	100.0

Icteric

Particulars	No.of respondents (n=40)	Percentage (100%)
Present	40	100.0

Fever

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	34	85.0	85.0	85.0
	Present	6	15.0	15.0	100.0
	Total	40	100.0	100.0	

Fever

Particulars	No.of respondents (n=40)	Percentage (100%)
Absent	34	85.0
Present	6	15.0

Pruritis

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	33	82.5	82.5	82.5
	Present	7	17.5	17.5	100.0
	Total	40	100.0	100.0	

Pruritis

Particulars	No.of respondents (n=40)	Percentage (100%)
Absent	33	82.5
Present	7	17.5

ganomegaly					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	29	72.5	72.5	72.5
	Present	11	27.5	27.5	100.0
	Total	40	100.0	100.0	

Organomegaly

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	29	72.5
Present	11	27.5

Pre Eclamptic features					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	2	5.0	5.0	5.0
	Present	38	95.0	95.0	100.0
	Total	40	100.0	100.0	

Pre Eclamptic features

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	2	5.0
Present	38	95.0

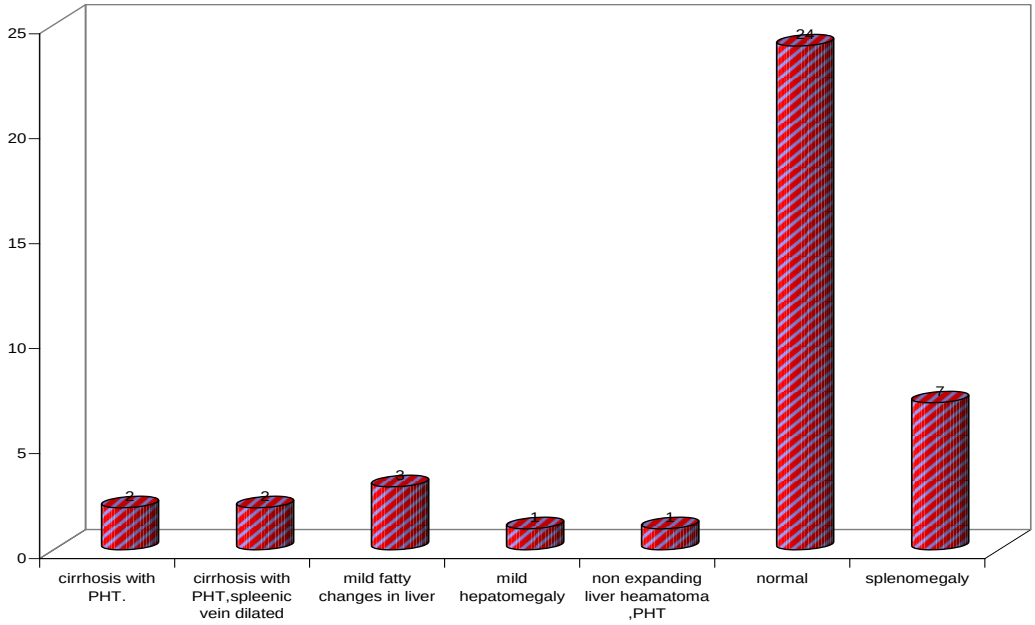
USG					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	cirrhosis with PHT.	2	5.0	5.0	5.0
	cirrhosis with PHT,spleenic vein dilated	2	5.0	5.0	10.0
	mild fatty changes in liver	3	7.5	7.5	17.5
	mild hepatomegaly	1	2.5	2.5	20.0
	non expanding liver heamatoma ,PHT	1	2.5	2.5	22.5
	Normal	24	60.0	60.0	82.5
	Splenomegaly	7	17.5	17.5	100.0
	Total	40	100.0	100.0	

Table No – 14

USG

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
cirrhosis with PHT.	2	5.0
cirrhosis with PHT,spleenic vein dilated	2	5.0
mild fatty changes in liver	3	7.5
mild hepatomegaly	1	2.5
non expanding liver heamatoma ,PHT	1	2.5
Normal	24	60.0
Splenomegaly	7	17.5

Diagrams No – 14



HB

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	6.00	2	5.0	5.0	5.0
	6.20	3	7.5	7.5	12.5
	6.40	1	2.5	2.5	15.0
	6.60	1	2.5	2.5	17.5
	6.70	2	5.0	5.0	22.5
	6.80	2	5.0	5.0	27.5
	7.00	1	2.5	2.5	30.0
	7.20	3	7.5	7.5	37.5
	7.50	1	2.5	2.5	40.0
	7.80	4	10.0	10.0	50.0
	8.00	5	12.5	12.5	62.5
	8.20	1	2.5	2.5	65.0
	8.50	1	2.5	2.5	67.5

	8.60	1	2.5	2.5	70.0
	8.80	5	12.5	12.5	82.5
	9.00	5	12.5	12.5	95.0
	9.20	1	2.5	2.5	97.5
	9.80	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

HB

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<7	12	30.0	30.0	30.0
	>7	28	70.0	70.0	100.0
	Total	40	100.0	100.0	

HB

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
6.00	2	5.0
6.20	3	7.5
6.40	1	2.5
6.60	1	2.5
6.70	2	5.0
6.80	2	5.0
7.00	1	2.5
7.20	3	7.5
7.50	1	2.5
7.80	4	10.0
8.00	5	12.5

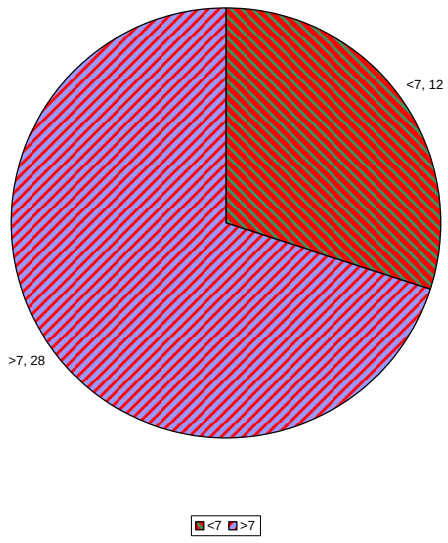
8.20	1	2.5
8.50	1	2.5
8.60	1	2.5
8.80	5	12.5
9.00	5	12.5
9.20	1	2.5
9.80	1	2.5

Table No – 15

HB

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
<7	12	30.0
>7	28	70.0

Diagrams No – 15



Platelet					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	36000.00	3	7.5	7.5	7.5
	40000.00	3	7.5	7.5	15.0
	43000.00	1	2.5	2.5	17.5
	45000.00	1	2.5	2.5	20.0
	54000.00	1	2.5	2.5	22.5
	56000.00	3	7.5	7.5	30.0
	60000.00	2	5.0	5.0	35.0
	65000.00	2	5.0	5.0	40.0
	67000.00	2	5.0	5.0	45.0
	70000.00	1	2.5	2.5	47.5
	72000.00	2	5.0	5.0	52.5
	74000.00	1	2.5	2.5	55.0
	76000.00	1	2.5	2.5	57.5
	78000.00	1	2.5	2.5	60.0
	80000.00	4	10.0	10.0	70.0
	87000.00	1	2.5	2.5	72.5
	88000.00	1	2.5	2.5	75.0
	90000.00	2	5.0	5.0	80.0
	94000.00	1	2.5	2.5	82.5
	100000.00	1	2.5	2.5	85.0
	110000.00	1	2.5	2.5	87.5
	120000.00	1	2.5	2.5	90.0

	125000.00	1	2.5	2.5	92.5
	150000.00	2	5.0	5.0	97.5
	200000.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Platelet

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
36000.00	3	7.5
40000.00	3	7.5
43000.00	1	2.5
45000.00	1	2.5
54000.00	1	2.5
56000.00	3	7.5
60000.00	2	5.0
65000.00	2	5.0
67000.00	2	5.0
70000.00	1	2.5
72000.00	2	5.0
74000.00	1	2.5
76000.00	1	2.5
78000.00	1	2.5
80000.00	4	10.0
87000.00	1	2.5
88000.00	1	2.5
90000.00	2	5.0
94000.00	1	2.5
100000.00	1	2.5
110000.00	1	2.5
120000.00	1	2.5
125000.00	1	2.5

150000.00	2	5.0
200000.00	1	2.5

Urine albumin					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	3	7.5	7.5	7.5
	Mild	12	30.0	30.0	37.5
	Moderate	16	40.0	40.0	77.5
	Severe	9	22.5	22.5	100.0
	Total	40	100.0	100.0	

Urine albumin

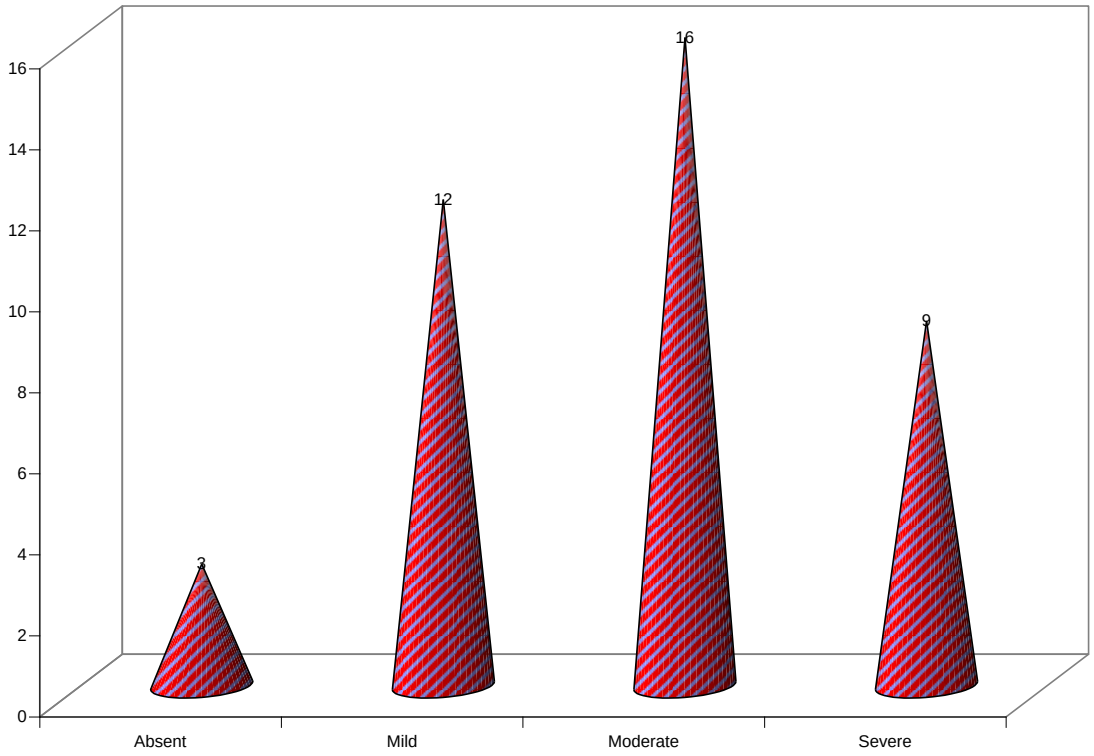
Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	3	7.5
Mild	12	30.0
Moderate	16	40.0
Severe	9	22.5

Table No – 16

Urine albumin

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	3	7.5
Mild	12	30.0
Moderate	16	40.0
Severe	9	22.5

Diagrams No – 16



Urea

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	24.00	1	2.5	2.5	2.5
	26.00	1	2.5	2.5	5.0
	28.00	2	5.0	5.0	10.0
	30.00	1	2.5	2.5	12.5
	32.00	2	5.0	5.0	17.5
	34.00	5	12.5	12.5	30.0
	36.00	2	5.0	5.0	35.0
	38.00	7	17.5	17.5	52.5

	40.00	2	5.0	5.0	57.5
	41.00	2	5.0	5.0	62.5
	42.00	5	12.5	12.5	75.0
	43.00	1	2.5	2.5	77.5
	44.00	2	5.0	5.0	82.5
	46.00	2	5.0	5.0	87.5
	48.00	2	5.0	5.0	92.5
	53.00	1	2.5	2.5	95.0
	56.00	1	2.5	2.5	97.5
	73.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Urea

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
24.00	1	2.5
26.00	1	2.5
28.00	2	5.0
30.00	1	2.5
32.00	2	5.0
34.00	5	12.5
36.00	2	5.0
38.00	7	17.5
40.00	2	5.0
41.00	2	5.0
42.00	5	12.5
43.00	1	2.5

44.00	2	5.0
46.00	2	5.0
48.00	2	5.0
53.00	1	2.5
56.00	1	2.5
73.00	1	2.5

Creatinine

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.80	6	15.0	15.0	15.0
	.90	7	17.5	17.5	32.5
	1.00	1	2.5	2.5	35.0
	1.10	2	5.0	5.0	40.0
	1.20	5	12.5	12.5	52.5
	1.30	3	7.5	7.5	60.0
	1.40	3	7.5	7.5	67.5
	1.70	1	2.5	2.5	70.0
	1.80	8	20.0	20.0	90.0
	2.00	1	2.5	2.5	92.5
	2.10	1	2.5	2.5	95.0
	2.20	2	5.0	5.0	100.0
	Total	40	100.0	100.0	

\Creatinine

Particulars	No.of respondents (n=40)	Percentage (100%)
.80	6	15.0
.90	7	17.5
1.00	1	2.5
1.10	2	5.0
1.20	5	12.5
1.30	3	7.5
1.40	3	7.5
1.70	1	2.5
1.80	8	20.0
2.00	1	2.5
2.10	1	2.5
2.20	2	5.0

Bilirubin

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	1.40	1	2.5	2.5	2.5
	1.50	1	2.5	2.5	5.0
	1.80	5	12.5	12.5	17.5
	1.90	1	2.5	2.5	20.0
	2.00	1	2.5	2.5	22.5
	2.10	4	10.0	10.0	32.5
	2.20	3	7.5	7.5	40.0
	2.30	3	7.5	7.5	47.5
	2.40	1	2.5	2.5	50.0

	2.60	2	5.0	5.0	55.0
	2.80	1	2.5	2.5	57.5
	3.00	4	10.0	10.0	67.5
	3.10	3	7.5	7.5	75.0
	3.60	4	10.0	10.0	85.0
	4.40	2	5.0	5.0	90.0
	4.50	1	2.5	2.5	92.5
	7.40	1	2.5	2.5	95.0
	8.30	1	2.5	2.5	97.5
	11.70	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Bilirubin					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<1.5	2	5.0	5.0	5.0
	>1.5	38	95.0	95.0	100.0
	Total	40	100.0	100.0	

Bilirubin

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
1.40	1	2.5
1.50	1	2.5
1.80	5	12.5
1.90	1	2.5
2.00	1	2.5
2.10	4	10.0

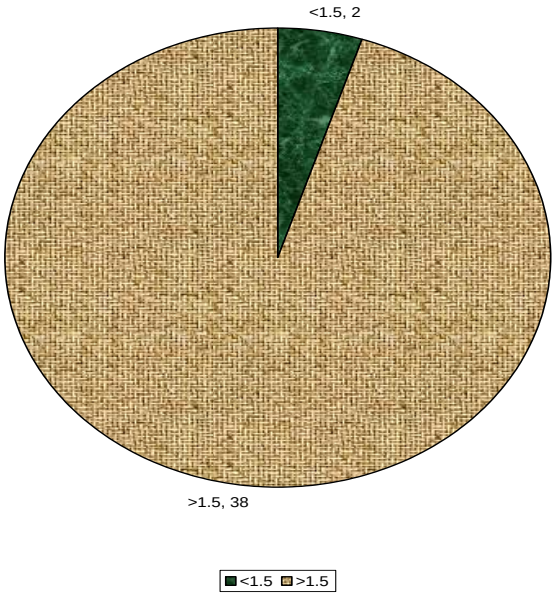
2.20	3	7.5
2.30	3	7.5
2.40	1	2.5
2.60	2	5.0
2.80	1	2.5
3.00	4	10.0
3.10	3	7.5
3.60	4	10.0
4.40	2	5.0
4.50	1	2.5
7.40	1	2.5
8.30	1	2.5
11.70	1	2.5

Table No – 17

Bilirubin

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
<1.5	2	5.0
>1.5	38	95.0

Diagrams No – 17



SGPT					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	56.00	1	2.5	2.5	2.5
	65.00	1	2.5	2.5	5.0
	68.00	1	2.5	2.5	7.5
	72.00	1	2.5	2.5	10.0
	74.00	1	2.5	2.5	12.5
	75.00	1	2.5	2.5	15.0
	78.00	2	5.0	5.0	20.0
	80.00	2	5.0	5.0	25.0
	81.00	1	2.5	2.5	27.5
	85.00	1	2.5	2.5	30.0
	87.00	1	2.5	2.5	32.5
	88.00	1	2.5	2.5	35.0
	89.00	1	2.5	2.5	37.5
	90.00	4	10.0	10.0	47.5
	98.00	1	2.5	2.5	50.0
	102.00	3	7.5	7.5	57.5
	106.00	2	5.0	5.0	62.5
	110.00	2	5.0	5.0	67.5
	121.00	1	2.5	2.5	70.0
	122.00	1	2.5	2.5	72.5
	124.00	1	2.5	2.5	75.0
	130.00	1	2.5	2.5	77.5
	132.00	1	2.5	2.5	80.0

	140.00	2	5.0	5.0	85.0
	143.00	1	2.5	2.5	87.5
	170.00	1	2.5	2.5	90.0
	206.00	1	2.5	2.5	92.5
	207.00	1	2.5	2.5	95.0
	226.00	1	2.5	2.5	97.5
	253.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

SGPT

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
56.00	1	2.5
65.00	1	2.5
68.00	1	2.5
72.00	1	2.5
74.00	1	2.5
75.00	1	2.5
78.00	2	5.0
80.00	2	5.0
81.00	1	2.5
85.00	1	2.5
87.00	1	2.5
88.00	1	2.5
89.00	1	2.5
90.00	4	10.0
98.00	1	2.5
102.00	3	7.5
106.00	2	5.0
110.00	2	5.0

121.00	1	2.5
122.00	1	2.5
124.00	1	2.5
130.00	1	2.5
132.00	1	2.5
140.00	2	5.0
143.00	1	2.5
170.00	1	2.5
206.00	1	2.5
207.00	1	2.5
226.00	1	2.5
253.00	1	2.5

SGOT

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	60.00	1	2.5	2.5	2.5
	65.00	1	2.5	2.5	5.0
	68.00	1	2.5	2.5	7.5
	78.00	1	2.5	2.5	10.0
	80.00	1	2.5	2.5	12.5
	84.00	1	2.5	2.5	15.0
	85.00	1	2.5	2.5	17.5
	86.00	1	2.5	2.5	20.0
	88.00	2	5.0	5.0	25.0
	89.00	1	2.5	2.5	27.5
	94.00	2	5.0	5.0	32.5
	98.00	4	10.0	10.0	42.5
	101.00	1	2.5	2.5	45.0

	102.00	2	5.0	5.0	50.0
	108.00	1	2.5	2.5	52.5
	124.00	1	2.5	2.5	55.0
	126.00	1	2.5	2.5	57.5
	130.00	1	2.5	2.5	60.0
	134.00	1	2.5	2.5	62.5
	140.00	6	15.0	15.0	77.5
	164.00	1	2.5	2.5	80.0
	168.00	1	2.5	2.5	82.5
	178.00	2	5.0	5.0	87.5
	188.00	1	2.5	2.5	90.0
	228.00	1	2.5	2.5	92.5
	240.00	1	2.5	2.5	95.0
	246.00	1	2.5	2.5	97.5
	278.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

SGOT

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
60.00	1	2.5
65.00	1	2.5
68.00	1	2.5
78.00	1	2.5

80.00	1	2.5
84.00	1	2.5
85.00	1	2.5
86.00	1	2.5
88.00	2	5.0
89.00	1	2.5
94.00	2	5.0
98.00	4	10.0
101.00	1	2.5
102.00	2	5.0
108.00	1	2.5
124.00	1	2.5
126.00	1	2.5
130.00	1	2.5
134.00	1	2.5
140.00	6	15.0
164.00	1	2.5
168.00	1	2.5
178.00	2	5.0
188.00	1	2.5
228.00	1	2.5
240.00	1	2.5
246.00	1	2.5
278.00	1	2.5

RBS

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	60.00	1	2.5	2.5	2.5
	62.00	1	2.5	2.5	5.0
	64.00	2	5.0	5.0	10.0

	80.00	1	2.5	2.5	12.5
	85.00	1	2.5	2.5	15.0
	90.00	6	15.0	15.0	30.0
	95.00	1	2.5	2.5	32.5
	100.00	2	5.0	5.0	37.5
	101.00	1	2.5	2.5	40.0
	107.00	1	2.5	2.5	42.5
	110.00	3	7.5	7.5	50.0
	122.00	1	2.5	2.5	52.5
	124.00	2	5.0	5.0	57.5
	126.00	1	2.5	2.5	60.0
	127.00	1	2.5	2.5	62.5
	130.00	5	12.5	12.5	75.0
	135.00	1	2.5	2.5	77.5
	136.00	1	2.5	2.5	80.0
	137.00	1	2.5	2.5	82.5
	140.00	2	5.0	5.0	87.5
	150.00	1	2.5	2.5	90.0
	160.00	2	5.0	5.0	95.0
	250.00	2	5.0	5.0	100.0
	Total	40	100.0	100.0	

RBS

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
60.00	1	2.5
62.00	1	2.5

64.00	2	5.0
80.00	1	2.5
85.00	1	2.5
90.00	6	15.0
95.00	1	2.5
100.00	2	5.0
101.00	1	2.5
107.00	1	2.5
110.00	3	7.5
122.00	1	2.5
124.00	2	5.0
126.00	1	2.5
127.00	1	2.5
130.00	5	12.5
135.00	1	2.5
136.00	1	2.5
137.00	1	2.5
140.00	2	5.0
150.00	1	2.5
160.00	2	5.0
250.00	2	5.0

LDH

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	300.00	1	2.5	2.5	2.5
	410.00	6	15.0	15.0	17.5
	412.00	1	2.5	2.5	20.0
	420.00	3	7.5	7.5	27.5
	422.00	1	2.5	2.5	30.0
	424.00	1	2.5	2.5	32.5

	440.00	1	2.5	2.5	35.0
	445.00	1	2.5	2.5	37.5
	447.00	1	2.5	2.5	40.0
	450.00	2	5.0	5.0	45.0
	460.00	2	5.0	5.0	50.0
	464.00	1	2.5	2.5	52.5
	470.00	2	5.0	5.0	57.5
	490.00	1	2.5	2.5	60.0
	510.00	2	5.0	5.0	65.0
	520.00	2	5.0	5.0	70.0
	521.00	1	2.5	2.5	72.5
	526.00	1	2.5	2.5	75.0
	540.00	1	2.5	2.5	77.5
	550.00	2	5.0	5.0	82.5
	570.00	1	2.5	2.5	85.0
	600.00	1	2.5	2.5	87.5
	640.00	1	2.5	2.5	90.0
	650.00	1	2.5	2.5	92.5
	653.00	1	2.5	2.5	95.0
	700.00	1	2.5	2.5	97.5
	830.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

LDH

Particulars	No.of respondents (n=40)	Percentage (100%)
300.00	1	2.5
410.00	6	15.0
412.00	1	2.5
420.00	3	7.5
422.00	1	2.5
424.00	1	2.5
440.00	1	2.5
445.00	1	2.5
447.00	1	2.5
450.00	2	5.0
460.00	2	5.0
464.00	1	2.5
470.00	2	5.0
490.00	1	2.5
510.00	2	5.0
520.00	2	5.0
521.00	1	2.5
526.00	1	2.5
540.00	1	2.5
550.00	2	5.0
570.00	1	2.5
600.00	1	2.5
640.00	1	2.5
650.00	1	2.5
653.00	1	2.5
700.00	1	2.5
830.00	1	2.5

Clotting time					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	3	2	5.0	5.0	5.0
	4	12	30.0	30.0	35.0
	5	9	22.5	22.5	57.5
	6	5	12.5	12.5	70.0
	7	6	15.0	15.0	85.0
	8	2	5.0	5.0	90.0
	10	2	5.0	5.0	95.0
	12	1	2.5	2.5	97.5
	14	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Clotting time					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<7	34	85.0	85.0	85.0
	>7	6	15.0	15.0	100.0
	Total	40	100.0	100.0	

Clotting time

Particulars	No.of respondents (n=40)	Percentage (100%)
3	2	5.0
4	12	30.0
5	9	22.5
6	5	12.5
7	6	15.0
8	2	5.0
10	2	5.0
12	1	2.5
14	1	2.5

Table No – 18

Clotting time

Particulars	No.of respondents (n=40)	Percentage (100%)
<7	34	85.0
>7	6	15.0

Diagrams No – 18

Blood transfusion (WBBlood transfusion)					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.00	5	12.5	12.5	12.5
	1.00	4	10.0	10.0	22.5
	2.00	14	35.0	35.0	57.5
	3.00	5	12.5	12.5	70.0
	4.00	11	27.5	27.5	97.5
	6.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Blood transfusion (WBBlood transfusion)

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
.00	5	12.5
1.00	4	10.0
2.00	14	35.0
3.00	5	12.5
4.00	11	27.5
6.00	1	2.5

Blood transfusion (FFPBlood transfusion)					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.00	3	7.5	7.5	7.5

	1.00	8	20.0	20.0	27.5
	2.00	13	32.5	32.5	60.0
	3.00	1	2.5	2.5	62.5
	4.00	7	17.5	17.5	80.0
	5.00	1	2.5	2.5	82.5
	6.00	5	12.5	12.5	95.0
	8.00	1	2.5	2.5	97.5
	14.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Blood transfusion (FFPBlood transfusion)

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
.00	3	7.5
1.00	8	20.0
2.00	13	32.5
3.00	1	2.5
4.00	7	17.5
5.00	1	2.5
6.00	5	12.5
8.00	1	2.5
14.00	1	2.5

Blood transfusion (PlateletBlood transfusion)

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	.00	23	57.5	57.5	57.5
	2.00	9	22.5	22.5	80.0
	4.00	7	17.5	17.5	97.5

	8.00	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Blood transfusion (PlateletBlood transfusion)

Particulars	No.of respondents (n=40)	Percentage (100%)
.00	23	57.5
2.00	9	22.5
4.00	7	17.5
8.00	1	2.5

Viral markers

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	0.00	40	100.0	000.0	100.0

Viral markers

Particulars	No.of respondents (n=40)	Percentage (100%)
0.00	40	000.0

Serum Uric Acid

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	4.50	3	7.5	7.5	7.5
	5.20	2	5.0	5.0	12.5
	5.60	5	12.5	12.5	25.0
	5.80	3	7.5	7.5	32.5
	6.00	1	2.5	2.5	35.0

	6.20	2	5.0	5.0	40.0
	6.40	1	2.5	2.5	42.5
	6.50	4	10.0	10.0	52.5
	6.60	2	5.0	5.0	57.5
	6.80	7	17.5	17.5	75.0
	7.00	5	12.5	12.5	87.5
	7.30	1	2.5	2.5	90.0
	7.50	3	7.5	7.5	97.5
	7.80	1	2.5	2.5	100.0
	Total	40	100.0	100.0	

Serum Uric Acid

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
4.50	3	7.5
5.20	2	5.0
5.60	5	12.5
5.80	3	7.5
6.00	1	2.5
6.20	2	5.0
6.40	1	2.5
6.50	4	10.0
6.60	2	5.0
6.80	7	17.5
7.00	5	12.5
7.30	1	2.5
7.50	3	7.5
7.80	1	2.5

Seizure					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	32	80.0	80.0	80.0
	Present	8	20.0	20.0	100.0
	Total	40	100.0	100.0	

Seizure

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	32	80.0
Present	8	20.0

Hepetic Encephalopathy					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	38	95.0	95.0	95.0
	Present	2	5.0	5.0	100.0
	Total	40	100.0	100.0	

Hepetic Encephalopathy

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	38	95.0
Present	2	5.0

IUD					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	32	80.0	80.0	80.0
	Present	8	20.0	20.0	100.0
	Total	40	100.0	100.0	

IUD

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	32	80.0
Present	8	20.0

Bleeding manifestation					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	35	87.5	87.5	87.5
	Present	5	12.5	12.5	100.0
	Total	40	100.0	100.0	

Bleeding manifestation

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	35	87.5
Present	5	12.5

Complication					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	26	65.0	65.0	65.0
	Present	14	35.0	35.0	100.0
	Total	40	100.0	100.0	

Complication

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Absent	26	65.0
Present	14	35.0

Mode of Delivery					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Normal	16	40.0	40.0	40.0
	Caeserian	24	60.0	60.0	100.0
	Total	40	100.0	100.0	

Mode of Delivery

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Normal	16	40.0
Caeserian	24	60.0

Diagnosis Delivery intervel <12					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Negative	32	80.0	80.0	80.0
	Positive	8	20.0	20.0	100.0
	Total	40	100.0	100.0	

Diagnosis Delivery intervel <12

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Negative	32	80.0
Positive	8	20.0

Diagnosis Delivery intervel 12-24					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Negative	23	57.5	57.5	57.5
	Positive	17	42.5	42.5	100.0
	Total	40	100.0	100.0	

Diagnosis Delivery intervel 12-24

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Negative	23	57.5
Positive	17	42.5

Diagnosis Delivery intervel >24					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Negative	25	62.5	62.5	62.5
	Positive	15	37.5	37.5	100.0
	Total	40	100.0	100.0	

Diagnosis Delivery intervel >24

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
Negative	25	62.5
Positive	15	37.5

Diagnosis Delivery intervel					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<12	8	20.0	20.0	20.0
	12 to 24	17	42.5	42.5	62.5
	>24	15	37.5	37.5	100.0
	Total	40	100.0	100.0	

Diagnosis Delivery intervel

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
<12	8	20.0
12 to 24	17	42.5
>24	15	37.5

Diagnosis Delivery intervel					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	<24	25	62.5	62.5	62.5
	>24	15	37.5	37.5	100.0
	Total	40	100.0	100.0	

Diagnosis Delivery interval

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
<24	25	62.5
>24	15	37.5

NICU					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	13	32.5	32.5	32.5
	Yes	27	67.5	67.5	100.0
	Total	40	100.0	100.0	

NICU

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
No	13	32.5
Yes	27	67.5

No NICU					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	35	87.5	87.5	87.5
	Yes	5	12.5	12.5	100.0
	Total	40	100.0	100.0	

NICU					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	NA	9	22.5	22.5	22.5
	NICU	27	67.5	67.5	90.0
	No NICU	4	10.0	10.0	100.0
	Total	40	100.0	100.0	

No NICU

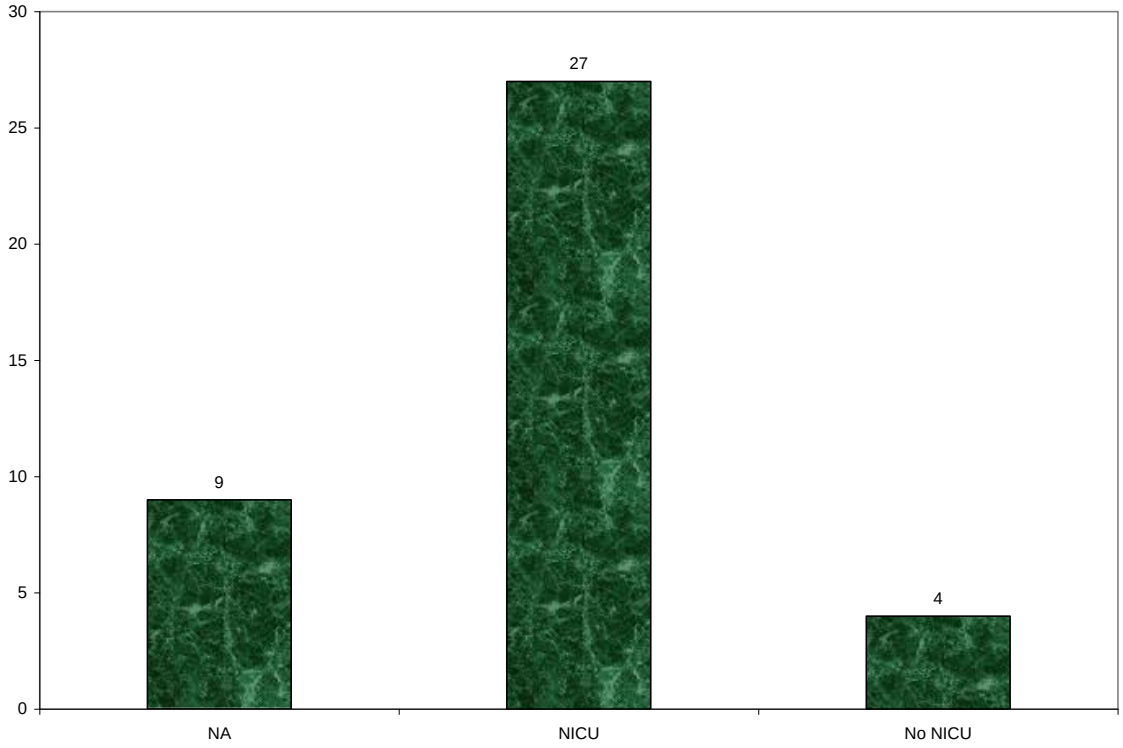
Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
No	35	87.5
Yes	5	12.5

Table No – 19

NICU

Particulars	No.of respondents (n=40)	Percentage (100%)
NA	9	22.5
NICU	27	67.5
No NICU	4	10.0

Diagrams No – 19



Perinatal Mortality

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	19	47.5	47.5	47.5
	Present	21	52.5	52.5	100.0
	Total	40	100.0	100.0	

Perinatal Mortality

Particulars	No.of respondents (n=40)	Percentage (100%)
Absent	19	47.5
Present	21	52.5

Maternal Mortality

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Absent	37	92.5	92.5	92.5
	Present	3	7.5	7.5	100.0
	Total	40	100.0	100.0	

Maternal Mortality

Particulars	No.of respondents (n=40)	Percentage (100%)
Absent	37	92.5
Present	3	7.5

Delivery recovery time <7 days

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Negative	22	55.0	55.0	55.0
	Positive	18	45.0	45.0	100.0
	Total	40	100.0	100.0	

Delivery recovery time <7 days

Particulars	No.of respondents (n=40)	Percentage (100%)
Negative	22	55.0
Positive	18	45.0

Delivery recovery time 7 to 14days

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Negative	30	75.0	75.0	75.0
	Positive	10	25.0	25.0	100.0
	Total	40	100.0	100.0	

Delivery recovery time 7 to 14days

Particulars	No.of respondents (n=40)	Percentage (100%)
Negative	30	75.0
Positive	10	25.0

Delivery recovery time 14days

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Negative	34	85.0	85.0	85.0
	Positive	6	15.0	15.0	100.0
	Total	40	100.0	100.0	

Delivery recovery time 14days

Particulars	No.of respondents (n=40)	Percentage (100%)
Negative	34	85.0
Positive	6	15.0

Delivery recovery time

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	NA	6	15.0	15.0	15.0
	<7days	18	45.0	45.0	60.0
	7 to 14days	10	25.0	25.0	85.0
	>14 days	6	15.0	15.0	100.0
	Total	40	100.0	100.0	

Delivery recovery time

Particulars	No.of respondents (n=40)	Percentage (100%)
NA	6	15.0
<7days	18	45.0
7 to 14days	10	25.0
>14 days	6	15.0

Delivery recovery time

		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	NA	6	15.0	15.0	15.0
	<7 days	18	45.0	45.0	60.0

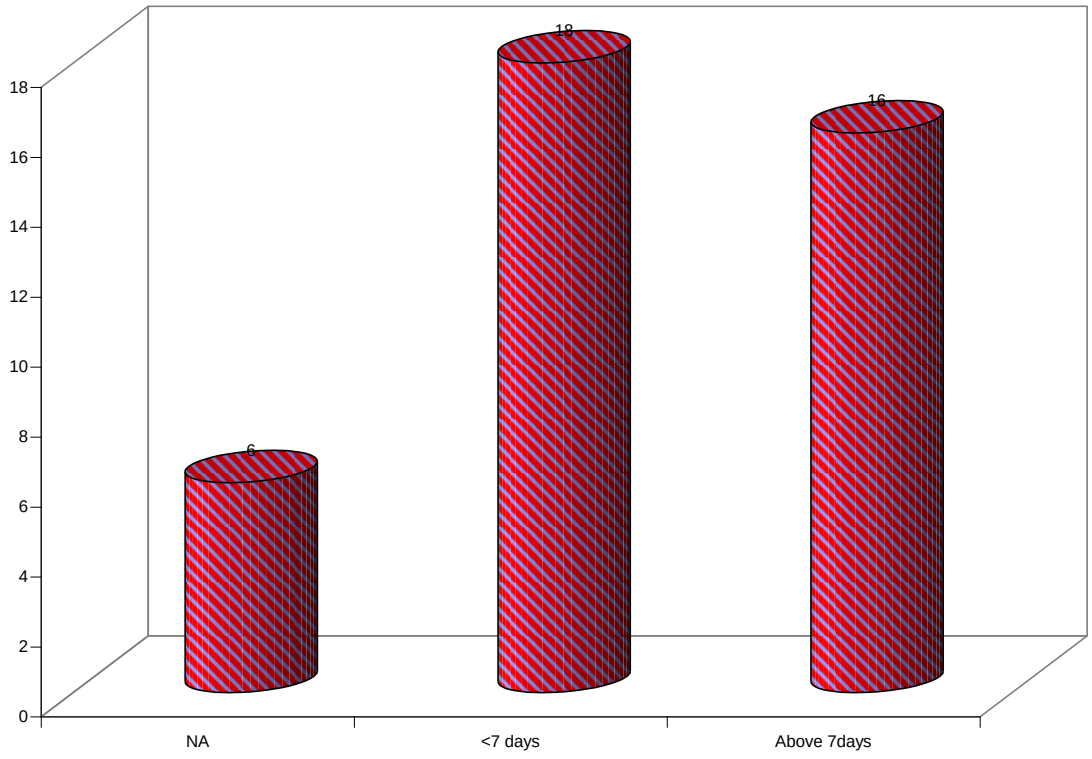
	Above 7days	16	40.0	40.0	100.0
	Total	40	100.0	100.0	

Table No – 20

Delivery recovery time

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
NA	6	15.0
<7 days	18	45.0
Above 7days	16	40.0

Diagrams No – 20



Diagnosis					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	AFLP	3	7.5	7.5	7.5
	Cirrhosis	3	7.5	7.5	15.0
	CRIGGLER NAJAR SYN	1	2.5	2.5	17.5
	Help	14	35.0	35.0	52.5
	Partial help	19	47.5	47.5	100.0
	Total	40	100.0	100.0	

Diagnosis

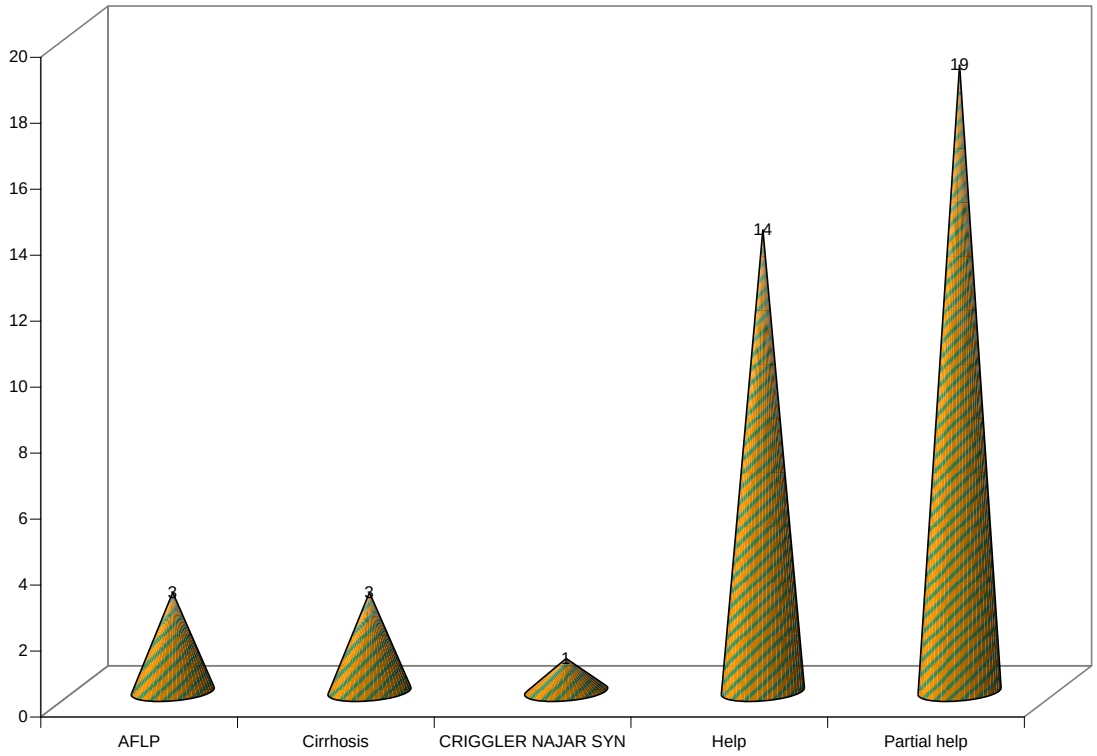
Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
AFLP	3	7.5
Cirrhosis	3	7.5
CRIGGLER NAJAR SYN	1	2.5
Help	14	35.0
Partial help	19	47.5

Table No – 21

Diagnosis

Particulars	**No.of respondents (n=40)**	**Percentage (100%)**
AFLP	3	7.5
Cirrhosis	3	7.5
CRIGGLER NAJAR SYN	1	2.5
Help	14	35.0
Partial help	19	47.5

Diagrams No – 21



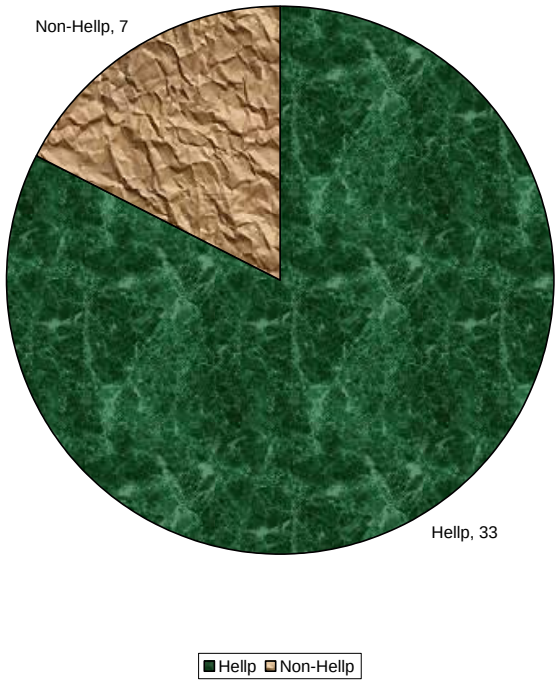
Diagnosis					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Hellp	33	82.5	82.5	82.5
	Non-Hellp	7	17.5	17.5	100.0
	Total	40	100.0	100.0	

Table No – 22

Diagnosis

Particulars	No.of respondents (n=40)	Percentage (100%)
Hellp	33	82.5
Non-Hellp	7	17.5

Diagrams No – 22



Descriptives

Descriptive Statistics					
	N	Minimum	Maximum	Mean	Std. Deviation
Age	40	22	34	26.80	3.353
BP1	40	110	160	138.75	16.823
BP2	40	70	100	90.00	7.511
HB	40	6.00	9.80	7.7800	1.05519

Urea	40	24.00	73.00	39.5250	8.73539
Creatinine	40	.80	2.20	1.3250	.44592
Bilirubin	40	1.40	11.70	3.1125	1.97318
SGPT	40	56.00	253.00	111.6500	45.19505
SGOT	40	60.00	278.00	127.1500	52.38396
RBS	40	60.00	250.00	117.9750	40.39643
LDH	40	300.00	830.00	492.8500	99.25558
Clotting time	40	3	14	5.83	2.374
Blood transfusion (WBBlood transfusion)	40	.00	6.00	2.4250	1.44803
Blood transfusion (FFPBlood transfusion)	40	.00	14.00	3.0500	2.65011
Blood transfusion (PlateletBlood transfusion)	40	.00	8.00	1.3500	1.88856
Viral markers	40	1.00	1.00	1.0000	.00000
Serum Uric Acid	40	4.50	7.80	6.3375	.83993
Valid N (listwise)	40				

Descriptives

Items	**N**	**Min.**	**Max.**	**S.D**	**Mean**
Age	40	22	34	3.353	26.80
BP1	40	110	160	16.823	138.75
BP2	40	70	100	7.511	90.00
HB	40	6.00	9.80	1.05519	7.7800
Urea	40	24.00	73.00	8.73539	39.5250
Creatinine	40	.80	2.20	.44592	1.3250
Bilirubin	40	1.40	11.70	1.97318	3.1125
SGPT	40	56.00	253.00	45.19505	111.6500

SGOT	40	60.00	278.00	52.38396	127.1500
RBS	40	60.00	250.00	40.39643	117.9750
LDH	40	300.00	830.00	99.25558	492.8500
Clotting time	40	3	14	2.374	5.83
Blood transfusion (WBBlood transfusion)	40	.00	6.00	1.44803	2.4250
Blood transfusion (FFPBlood transfusion)	40	.00	14.00	2.65011	3.0500
Blood transfusion (PlateletBlood transfusion)	40	.00	8.00	1.88856	1.3500
Viral markers	40	1.00	1.00	.00000	1.0000
Serum Uric Acid	40	4.50	7.80	.83993	6.3375

RESULTS

RESULTS

OBSTETRIC CODE

In our study of non infective jaundice in pregnancy, more number of patients belonged to the primi gravida category (62.5%) than multi gravid (37.5%)

INCIDENCE

Among the incidence of all the non infective jaundice cases HELLP and PARTIAL HELLP were the most common and together comprised a staggering (82.5%) of the patients. The rest 17.5% included cases of AFLP, cirrhosis and criggler najar syndrome.

GESTATIONAL AGE

Most of the pregnancy complicated by the non infective jaundice in this study presented at a gestational age earlier than 37 weeks (70%) when compared to the (30%) who presented at term.

BLOOD PRESSURE

In our study patients with higher BP reading >130/90 (62.5%) at the time of admission manifested a more severe course of the disease as evidenced by higher complications, higher maternal and fetal morbidity and mortality.

PRE ECLAMPTIC FEATURES

95% patients who constituted the study presented with preeclamptic features of pedal edema ,abdominal wall edema and urine albumin showed higher incidence of partial HELLP and HELLP syndrome.

BILIRUBIN

It was observed that patients with bilirubin values higher than the standard deviations of 1.5mg/dl (95%) in our study were more prone for higher risk of complications.

DIAGNOSIS DELIVERY INTERVAL

In our study it was observed that patient who delivered within 24hrs (28.6%) of diagnosis showed better prognosis as evidenced by milder course of the disease, lesser incidence of complications and no maternal mortality

MATERNAL MORTALITY

In my study, multi gravida presented with severe form of the disease had higher incidence of maternal mortality the features with increased maternal mortality were cirrhosis with portal hypertension

DELIVERY RECOVERY TIME

Many patients presented with Bp<130/90 (37.5%) in our study showed recovery within 7 days when compared to 40 % of those with Bp > 130/90 who recovered in a week.

GESTATIONAL AGE- NICU ADMISSION

In this study, the babies born to mother who presented with the disease at the gestational age <37 weeks showed higher morbidity as observed by admission in NICU (70.4%) whereas only (29.6%) of the babies delivered in term patients with non infective jaundice were admitted in NICU.

CONCLUSION

CONCLUSION

HELLP constitutes most of the causes of non infective jaundice ,it leads to increased maternal morbidity than mortality. Hence mortality is preventable with early diagnosis.

Higher blood pressure reading showed higher risk of complications

Higher bilirubin values and altered LFT were more prone for severe manifestations of the disease. Advanced maternal age at gestation are higher risk of complication. Cirrhosis with portal hypertension was found to be the cause of maternal mortality among all causes of non infective jaundice in a tertiary care centre.

Early intervention and delivery of the fetus within 24hrs reduces the incidence of complications and improved prognosis and reduce delivery recovery time.

Patients presented with lower BP recovered earlier Preterm babies of affected mothers ran a higher risk of complications

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ANNEXURE

MADURAI MEDICAL COLLEGE
MADURAI, TAMILNADU, INDIA -625 020
(Affiliated to The Tamilnadu Dr.MGR Medical University, Chennai, Tamil Nadu)

ETHICS COMMITTEE CERTIFICATE

Name of the Candidate	:	**Dr.P.Nandini**
Course	:	PG in MS., Obstetrics & Gynaecology
Period of Study	:	2015 - 2018
College	:	MADURAI MEDICAL COLLEGE
Research Topic	:	Study of non infective Jaundice in Pregnancy.
Ethical Committee as on	:	11.09.2017

The Ethics Committee, Madurai Medical College has decided to inform that your Research proposal is accepted.

Member Secretary Chairman Dean / Convenor

Prof Dr V Nagaraajan
M.D., MNAMS, D.M., Dsc.,(Neuro), Dsc (Hon)
CHAIRMAN
IEC - Madurai Medical College
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ABBREVIATION

AST	ASPARTATE AMINO TRANSFERASE
ALT	ALANINE AMINO TRANSFERASE
ALP	ALKALINE PHOSPHATASE
LDH	LACTATE DEHYDROGENASE
HELLP	HEMOLYSIS ELEVATED LIVER ENZYMES LOW PLATELET
AFLP	ACUTE FATTY LIVER OF PREGNANCY
LCHAD	LONG CHAIN THREE HYDROXY ACYL CO A DEHYDROGENASE
ICP	INTRA HEPATIC CHOLESTASIS OF PREGNANCY
UDCA	URSODEOXY CHOLIC ACID
WD	WILSON DISEASE
PBC	PRIMARY BILIARY CIRRHOSIS

PROFORMA

NON INFECTIVE / INFECTIVE JAUNDICE PROFORMA

PATIENT DETAILS :

NAME	:		ADDRESS		
AGE	:				
IP.No	:				
			D.O.A.	:	
			D.O.P	:	
			D.O.D	:	

HISTORY

HISTORY OF	PRESENT	ABSENT
Blood Transfusion		
Iv. Drug abuse		
Occupational Exposure		
Multiple Sexual Partners		
Use of Drugs		
Alcohol Intake		
Any jaundice in past		

CONSTITUTIONAL FEATURES :

HISTORY OF	PRESENT	ABSENT
Fever		
Jaundice		
Vomitting		
Abdominal Pain		
Loose Stools		
Hematemesis		
Clay coloured Stools		
Malena		
Pruritis		
Arthralgia		
Weightloss		

EXAMINATION :

LEVEL OF SENSORUM
TEMPERATURE
PALLOR
ICTERUS
PEDAL EDEMA
PURPURA
PR
BP
RR
SPO2
CVS
RS
CNS
P/A
ORGANOMEGALY
ASCITIS
P/v

OBSTETRICS :

OBSTETRIC CODE
LMP
EDD
GA
ANTENTAL COURSE
GA DETECTION
DIAGNOSIS

INVESTIGATIONS :

ROUTINE	DATE	DATE	DATE
Hb			
TC			
DC			
ESR			
PLATELETS			
CLOTTING TIME			
RBS			
UREA			
CREATININE			
PT /INR			
Na+			
K+			
Cl-			

LIVER FUNCTION TESTS	DATE	DATE	DATE
BILIRUBIN			
DIRECT			
INDIRECT			
SGOT			
SGPT			
ALP			
LDH			
ALBUMIN			
GLOBULIN			
PERIPHERALSMEAR			

VIRAL MARKERS

HBsAg	
HBcIgM	
HBcIgG	
HBeAg	
Anti HCV	
Anti HAV	
Anti HEV	
MOLECULAR DETECTION	

OTHER INVESTIGATIONS

USG ABDOMEN	
ENDOSCOPY	
LIVER BIOPSY	

MANAGEMENT :

TREATMENT OF PRIMARY PATHOLOGY
TREATMENT OF JAUNDICE
OBSTETRIC MANAGEMENT
POSTNATAL MANAGEMENT
TRANSFUSIONS
COMPLICATIONS
OUTCOME
ADMISSION – RECOVERY INTERVAL
FOLLOW UP

MASTER CHART