MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY

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DECLARATION

I Dr.S.GAYATHRI solemnly declare that the dissertation titled **" MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY "** has been prepared by me. I also declare that this Bonafide work or a part of this work was not submitted by me or any other for any award , degree , diploma to any other university board either in India or abroad.

This dissertation is submitted to the **Tamil Nadu Dr.M.G.R Medical University** , Chennai in partial fulfillment of the rules and regulation for the award of **M.D Degree , Branch – II (Obstetrics and Gynaecology)** to be held in April 2013.

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BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled "**MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY** " is a bonafide work done by **Dr.S.GAYATHRI** , under direct supervision and guidance , submitted to the **TAMIL NADU Dr.M.G.R MEDICAL UNIVERSITY** , in partial fulfillment of university regulation for M.D degree , Branch – II, (Obstetrics and Gynaecology).

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MASTER CHART

INTRODUCTION

INTRODUCTION

The incidence of jaundice in India varies from 0.4 to 0.9/1000 deliveries. Jaundice in pregnancy carries a grave prognosis for both the mother and the fetus , and is responsible for 10% of maternal deaths. Liver disease in pregnancy is an important medical disorder seen more often in developing countries than in developed ones. The present study analyzes the causes and the fetomaternal outcome in pregnancies affected with jaundice.

Abnormal liver test results are obtained in 3% to 5% of pregnancies because of many potential causes and the clinical outcomes ranges from self-limiting to rapidly fatal.

The main causes for abnormal liver tests in pregnant patientsare:

(1) Pregnancy-related liver disease. These are the common reasons for abnormal liver function tests in pregnancy. Five liver diseases unique to pregnancy includes the following -

(i) Hyperemesis gravidarum (HG)

(ii) Intrahepatic cholestasis of pregnancy (ICP)

(iii) Preeclampsia

(iv) Hemolysis, elevated liver enzymes, and low platelets (HELLP)

(v) Acute fatty liver of pregnancy (AFLP).

(2) Newly acquired liver diseases like acute viral hepatitis, drug induced liver injury, or gallstones

(3) Preexisting chronic liver disease such as cholestatic liver disease, autoimmune hepatitis, Wilson disease, and chronic viral hepatitis.

(4) physiologic changes in pregnancy - Abnormal liver function test due to physiological changes in pregnancy without liver dysfunction have a unique pattern.

The common maternal complications encountered are Encephalopathy, Disseminated intravascular coagulation , Renal failure , Shock, Postpartum hemorrhage , Pyrexia and also Death.

Elevated level of serum bilirubin causes vasoconstrictive effect on the placental vessels and cardiotoxic effect resulting in fetal asphyxia and intrauterine death . Also elevated bilirubin produce cellular effect which stimulates uterine contractility and sensitizes myometrium to oxytocin resulting in preterm labour.

High maternal mortality and morbidity in our country are due to many factors like Poor hygiene , inadequate sanitation , malnutrition, prevalence of anemia, delay in seeking medical advice, lack of awareness ,and delay in referral to the higher centers. Many patients are brought in moribund condition to the hospital at admission itself and hence they do not respond to treatment.

The prevalence of viral hepatitis in pregnancy can be reduced by creating public awareness, proper sanitation facilities, safe drinking water, immunization against viral hepatitis , improved antenatal care for early detection and well equipped hospitals for intensive care . Thereby, mortality and morbidity of jaundice complicating pregnancy can be decreased.

The aim of this study is to identify the various etiologies and distribution of jaundice with reference to age , parity and trimesters and also to determine the fetomaternal outcome among the pregnant women affected by jaundice treated at Government Rajaji Hospital , Madurai.

HISTORY

Jaundice was once called the "morbus regius" with a belief that only the touch of a king could cure it. Jaune in French means yellow from which the word jaundice was derived.

Before 400 B.C Hippocrates has written about the yellow discoloration in association with fever. Hippocratic physicians have treated liver disease and night blindness with raw ox liver soaked in honey.

In 1724 Cotton Mather has stated that `` Morbus Regius, or The Royal Disease; because it brings with it the Colour of Gold unto them that have it.``

In 1864 , Stadeler coined the term bilirubin.

From 1862 Friedrich Theodor Frerichs is called as the father of modern liver pathology following his publication " Klinik der Leberkrankheiten " in the year 1858.

(Friedrich Theodor Frerichs)

ANATOMY OF LIVER

Liver is the largest exocrine gland in the body weighing about 1.5 kg in an average adult weighing about 70 kg. This is located in the right hypochondrium and a part of epigastric region.

Liver is attached to the anterior abdominal wall and the diaphragm by four distinctive ligaments:

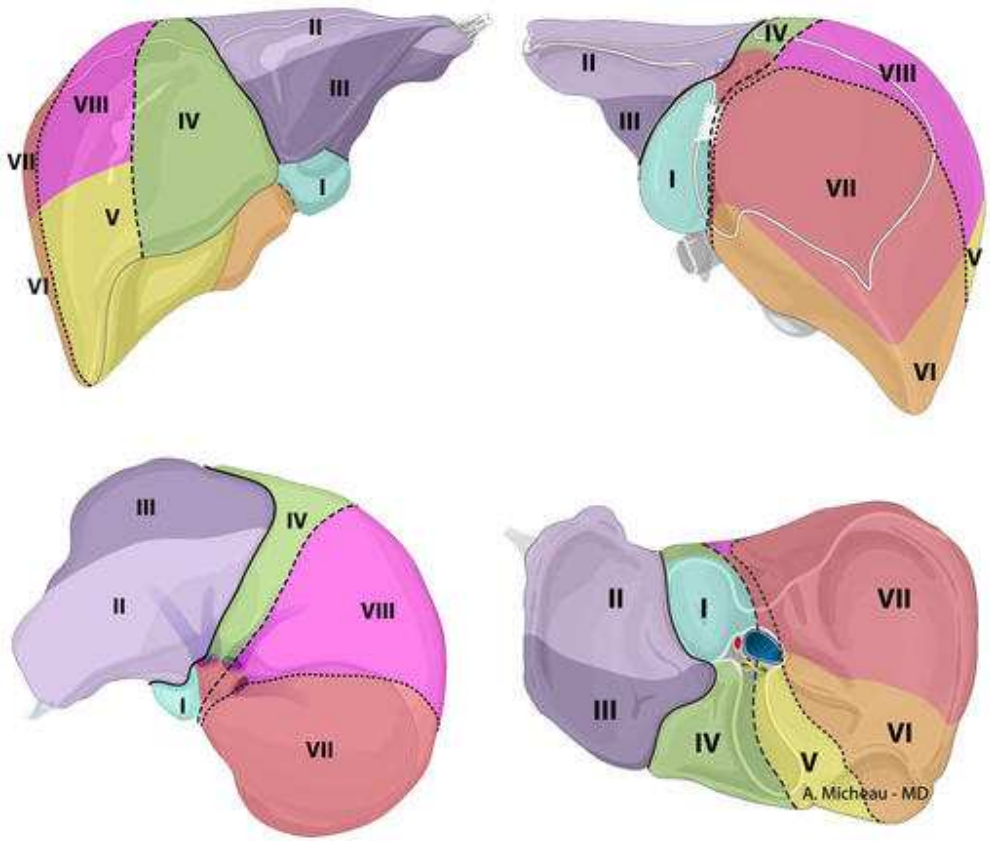
(1)Coronary ligament which connects the posterior surface of the right hepatic lobe to the diaphragm with a superior and an inferior layer between that lies in the bare area of the liver;

(2)Right triangular ligament which is formed by fusion of the superior and inferior layers of the coronary ligament;

(3)Left triangular ligament which connects the posterior surface of the left lobe of the liver to the diaphragm

(4)Falciform ligament which extends from the diaphragm and anterior wall above the level of the umbilicus to the surface of the liver, where it divides the left hepatic lobe into the left lateral and left medial segments

Liver segment is divided into eight segments . COUINAUD coined a system for liver segmental nomenclature (8 segments). Liver is divided into segments by a longitudinal planes drawn through each hepatic vein to the vena cava and a transverse plane at the level of the main portal bifurcation .Cantlie’s line marks the course of the middle hepatic vein



BLOOD SUPPLY

The two sources that supply blood to the liver are Hepatic artery and portal vein. Hepatic artery is a branch from celiac trunk of aorta . Portal vein is formed by the confluence of the superior mesenteric vein and the splenic vein at the level of the second lumbar vertebra behind the head of pancreas . It supplies about 75% of the total liver blood supply by volume.

Blood exits the liver via the central vein accounting for 25% of cardiac output. Blood flow into the liver is controlled by number of factors like Muscular sphincters , autonomic nervous system, circulating hormones, bile salts, and metabolites .

VENOUS DRAINAGE

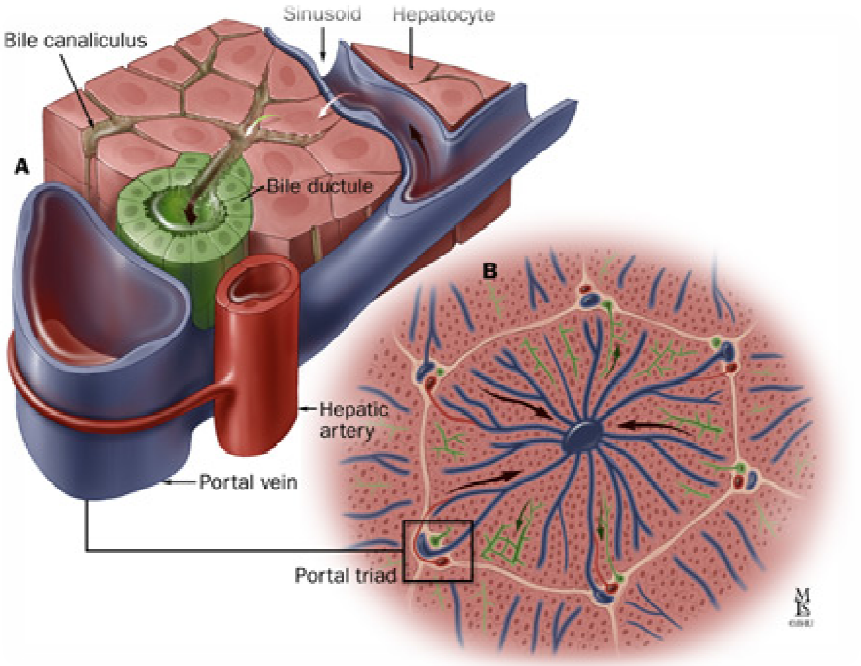
Majority of the venous drainage of the liver occurs through three hepatic veins . Right hepatic vein drains the segments 6,7,8 and enters directly into the vena cava.

Middle hepatic vein drains segments 5 and inferior part of segment 4 . Left hepatic vein drains the segments 2,3 and superior part of 4.

MICROANATOMY

Liver is composed of hexagonal shaped units called hepatic lobules .Each lobule is a tiny hexagonal or pentagonal cylinder of about 2x1 mm. These form the anatomical units of the liver. A tributary of hepatic vein extends through the centre of each lobule called interlobular vein (central vein).

Around this central vein hepatic cells are arranged as plates or irregular walls radiating outward. On the outer corners of each lobule , portal triad consisting of a branch of portal vein , a branch of hepatic artery and an interlobular bile ductules is arranged. Three adjoining parts of hepatic lobules constitute portal lobule with common drainage of bile into bile ductules of portal triad.



PORTAL TRIAD

Branches of portal vein, hepatic artery and the biliary ducts bound together in the perivascular fibrous capsule to form portal triad. Hepatocytes are arranged in plates joined with tight junctions and the apical membrane forms the biliary caniculi

Hepatocytes are segregated from the blood-filled sinusoids by fenestrated endothelial cells without a basement membrane, and by a loose connective tissue layer known as the space of Disse.

HEPATIC STELLATE CELLS

These are Star shaped cells that reside in the space of Disse. It helps to Store lipids particularly vitamin A.

Inflammatory cytokines cause activation, which involves the loss of stores of vitamin A and a dramatic upregulation in the production of extracellular matrix materials, such as collagen. When collagen is deposited in the space of Disse it impairs hepatic function.

KUPFFER CELLS

Kupffer cells belongs to macrophage lineage. The sinusoids are lined by kupffer cells which clear the cellular debris and other particulate material and to some extent bacteria that enters the portal triad.

They express cell-surface receptors for altered proteins. Fc immunoglobulin receptors used to internalize foreign proteins or microorganisms that have been coated with host antibodies.

BILIARY SYSTEM

Gallbladder is a biliary reservoir that lies against the inferior surface of segments IV and V of the liver, usually making an impression against it. A peritoneal layer covers most of the gallbladder except for the portion adherent to the liver. The size is variable, but usually about 10 cm long and 3 to 5 cm wide. Gallbladder is composed of a fundus, body, infundibulum, and neck . Ultimately empties into the cystic duct

BIOCHEMICAL FUNCTIONS OF LIVER

1. storage of substances like protein , glycogen , vitamins and folic acid..
2. Synthesis of plasma proteins , glycogen , phospholipids , bile acids and heparin .
3. Secretion of bile acids and bile pigments into the bile.
4. Metabolism of carbhohydrate , protein and fat.
5. Excretion of heavy metals , hormones, cholesterol and bile pigments.
6. Detoxification of ingested drugs.
7. In fetal life the liver produces RBC and WBC.
8. Kupfer cells acts as body defence.

9. Thyroxine is converted into triiodothyronin.and also participates in the activation of vitamin D.
10. Converts toxic substances into nontoxic substances e.g., benzoic acid is converted into hippuric acid by conjugation with glycine. Ammonia is converted to urea.

PRODUCTION AND METABOLISM OF BILIRUBIN:

SOURCE OF BILIRUBIN:

Mainly 80% is from senescent RBC and about 15 -20 % from ineffective erythropoiesis

Metabolism of haem containing protein can be divide into three phases:

(i) Hepatic uptake

(ii) Conjugation

(iii) Excretion into bile (rate limiting step)

UPTAKE:

Unconjugated bilirubin bound to albumin enters liver and the complex dissociates. Non-polar bilirubin enters the hepatocyte by diffusion or transport across plasma membrane. It binds to cytoplasmic anion binding protein ligandin glutathione-s-transferase and prevents efflux of bilirubin back into plasma.

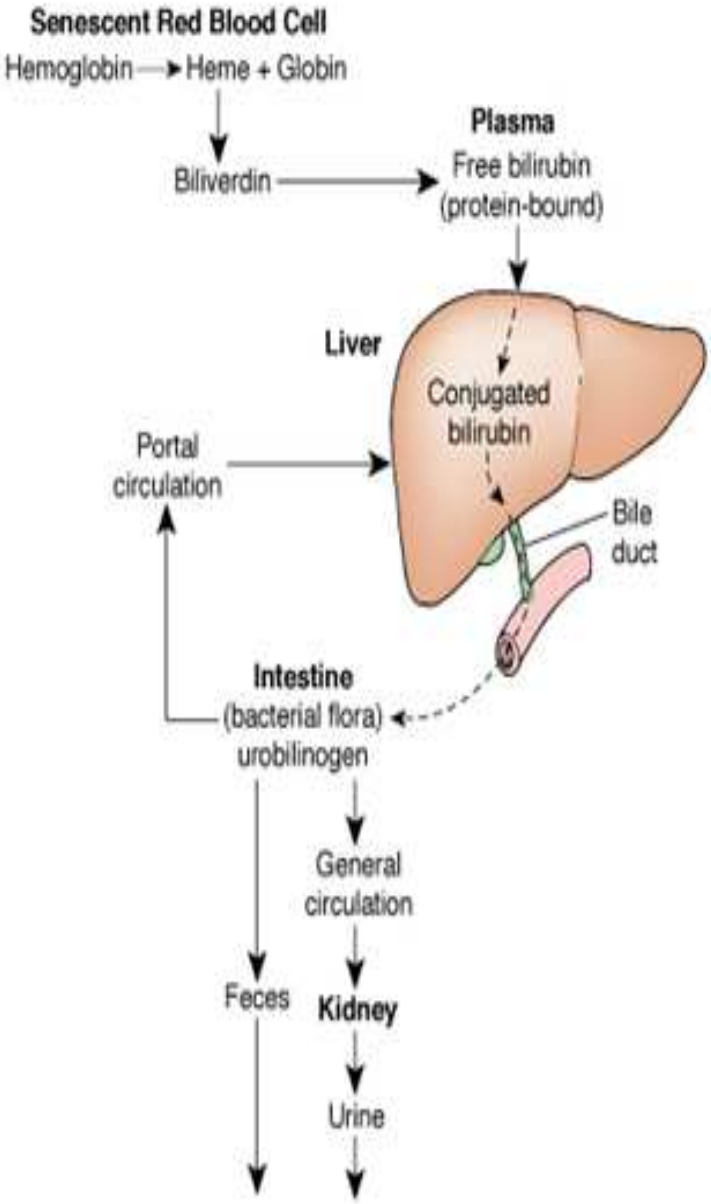
CONJUGATION:

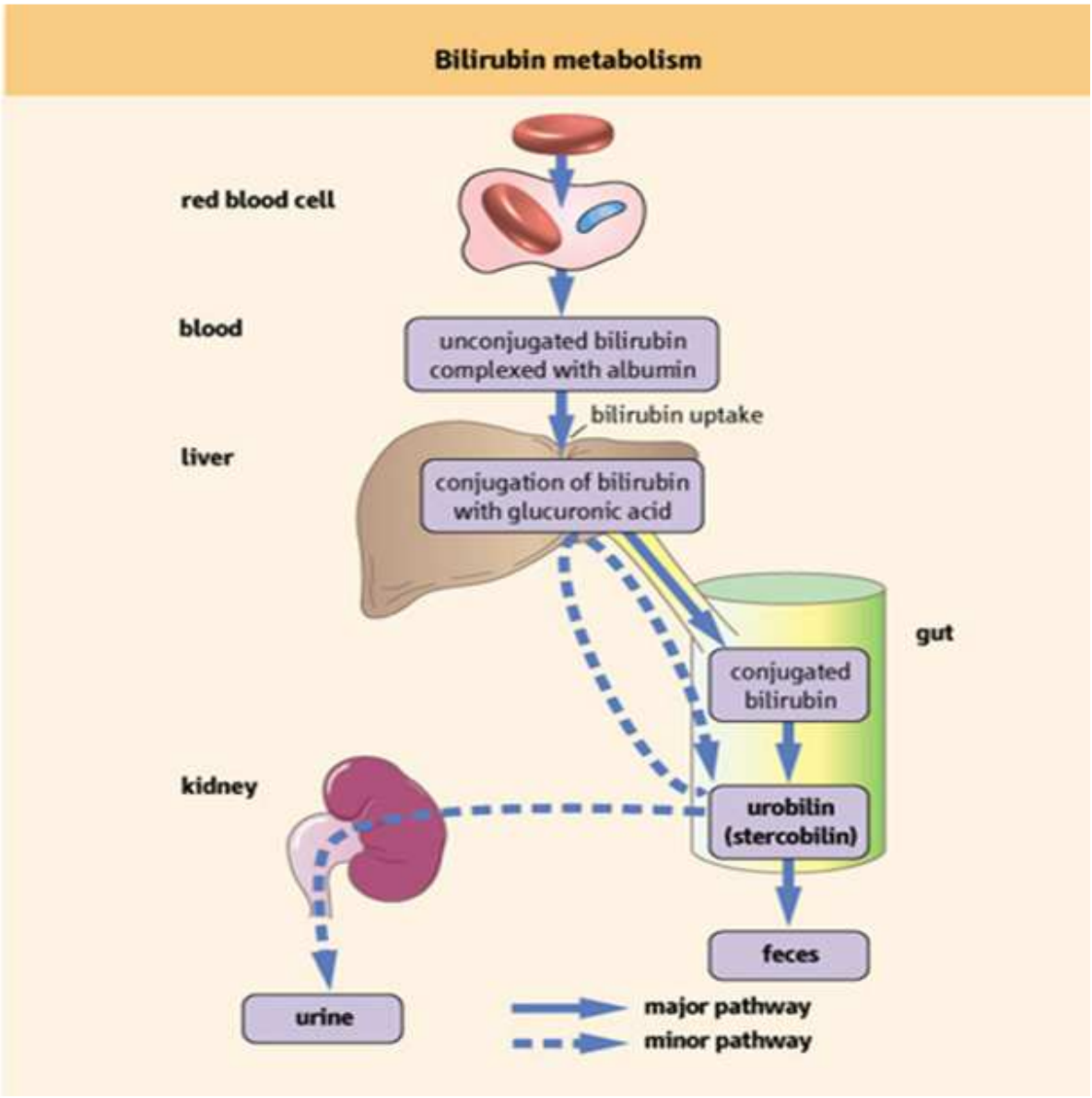
Unconjugated bilirubin is water insoluble. Hence gets conjugated to glucuronic acid forming bilirubin glucoranide , which is water-soluble.

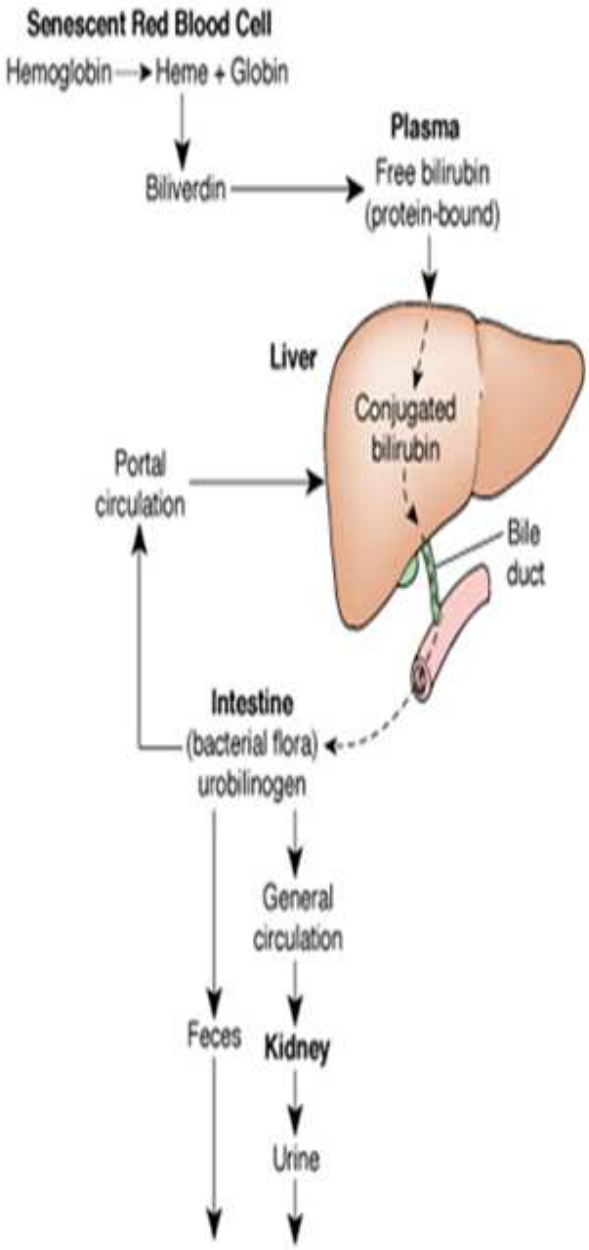
Conjugation occurs in endoplasmic reticulam. Catalysed by glucuronyl transferase in two step reaction.

EXCRETION:

- Conjugated bilirubin is water soluble and is secreted by the hepatocytes into the biliary canaliculi . It is converted to stercobilinogen by bacteria in the gut am Oxidized to stercobilin which is colored. Excreted in feces. Some stercobilin may be re-adsorbed by the gut and re-excreted by either the liver or kidney


Senescent Red Blood Cell
Hemoglobin
Heme + Globin
Biliverdin
Plasma
Free bilirubin
(protein-bound)
Liver
Conjugated
bilirubin
Portal
circulation
Bile
duct
Intestine
(bacterial flora)
urobilinogen
General
circulation
Feces
Kidney
Urine


Bilirubin metabolism
red blood cell
blood
unconjugated bilirubin complexed with albumin
bilirubin uptake
liver
conjugation of bilirubin with glucuronic acid
gut
conjugated bilirubin
kidney
urobilin (stercobilin)
feces
urine
major pathway
minor pathway


Senescent Red Blood Cell
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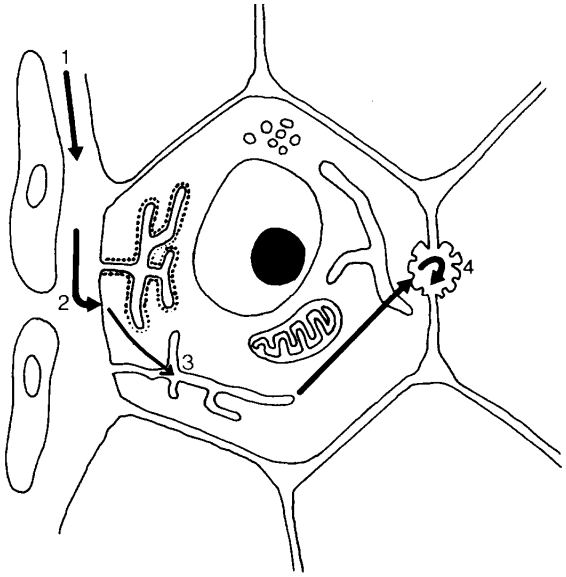
Liver
Conjugated
bilirubin
Portal
circulation
Bile
duct
Intestine
(bacterial flora)
urobilinogen
General
circulation
Feces
Kidney
Urine

Causes of Unconjugated hyperbilirubinaemia:

1) Increased bilirubin formation e.g., Haemolysis , Ineffective erythropoiesis , Blood transfusion and Haematoma.
2) Decreased bilirubin uptake by hepatocyte e.g., Drugs like Rifampicin , Gilbert's syndrome
3) Deficit in conjugation - Gilbert's syndrome , Crigler Najjar , Drugs

Causes of Unconjugated hyperbilirubinaemia:

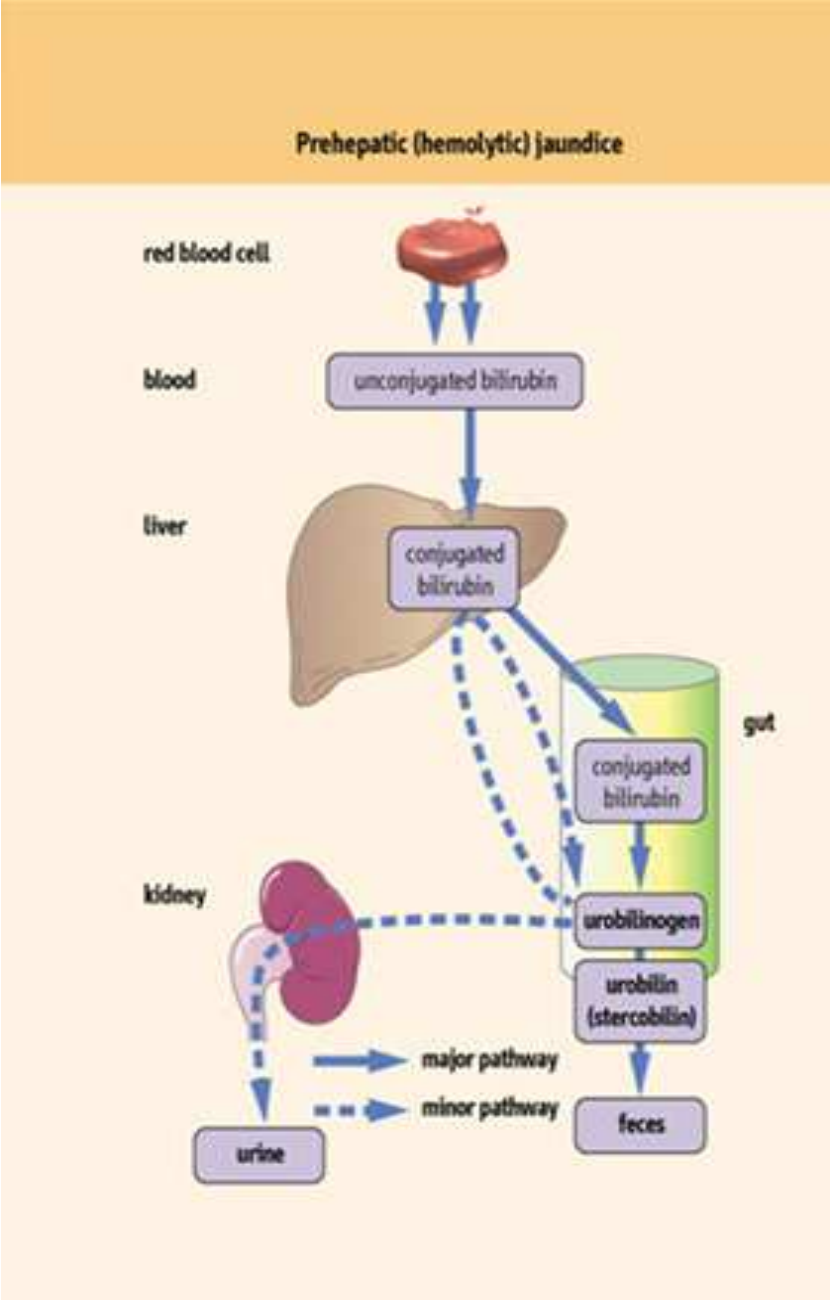
1) Dubin-Johnson syndrome, Rotor's syndrome
2) Hepatocellular dysfunction
3) Hepatic disorder with prominent cholestasis
4) Biliary duct obstruct



PREHEPATIC JAUNDICE

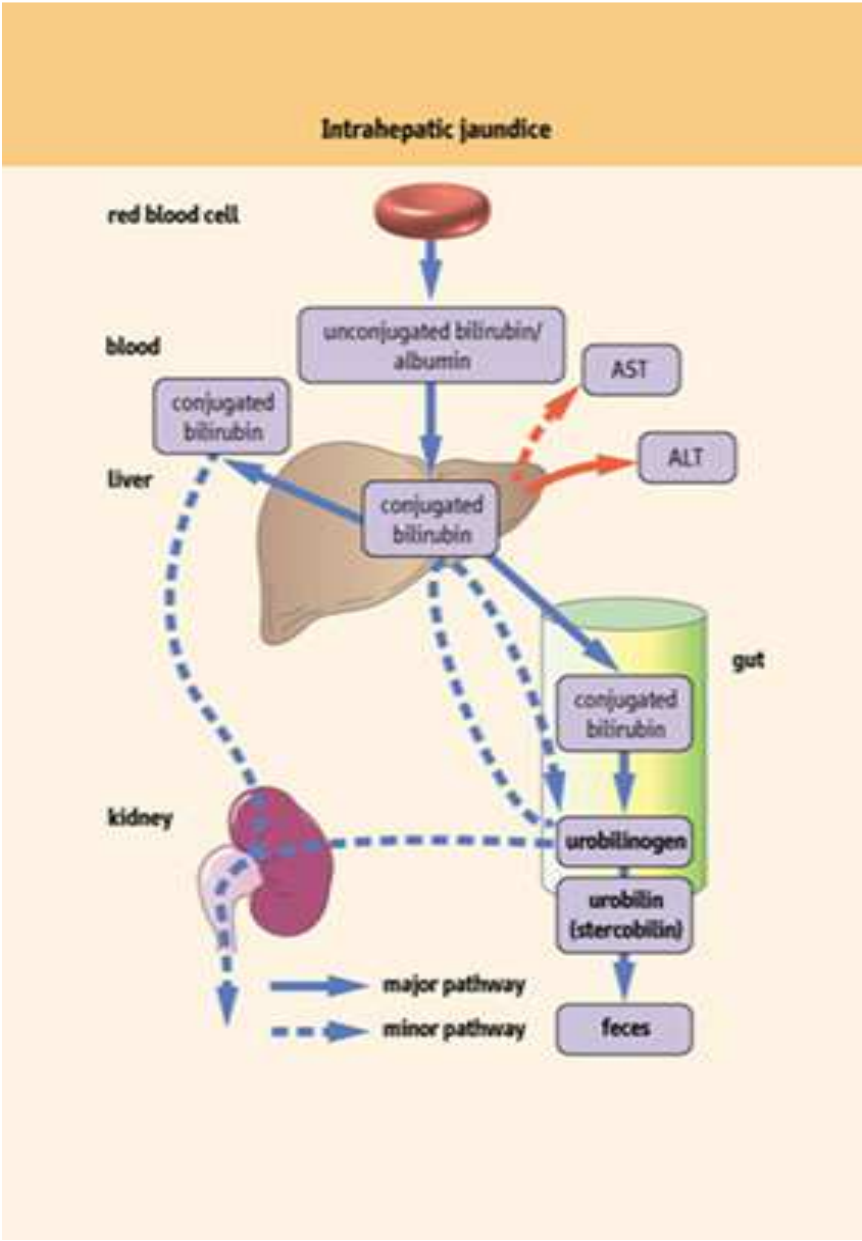
Prehepatic jaundice results from excessive RBC lysis as in haemolytic anaemia .

Unconjugated bilirubin level is increased.



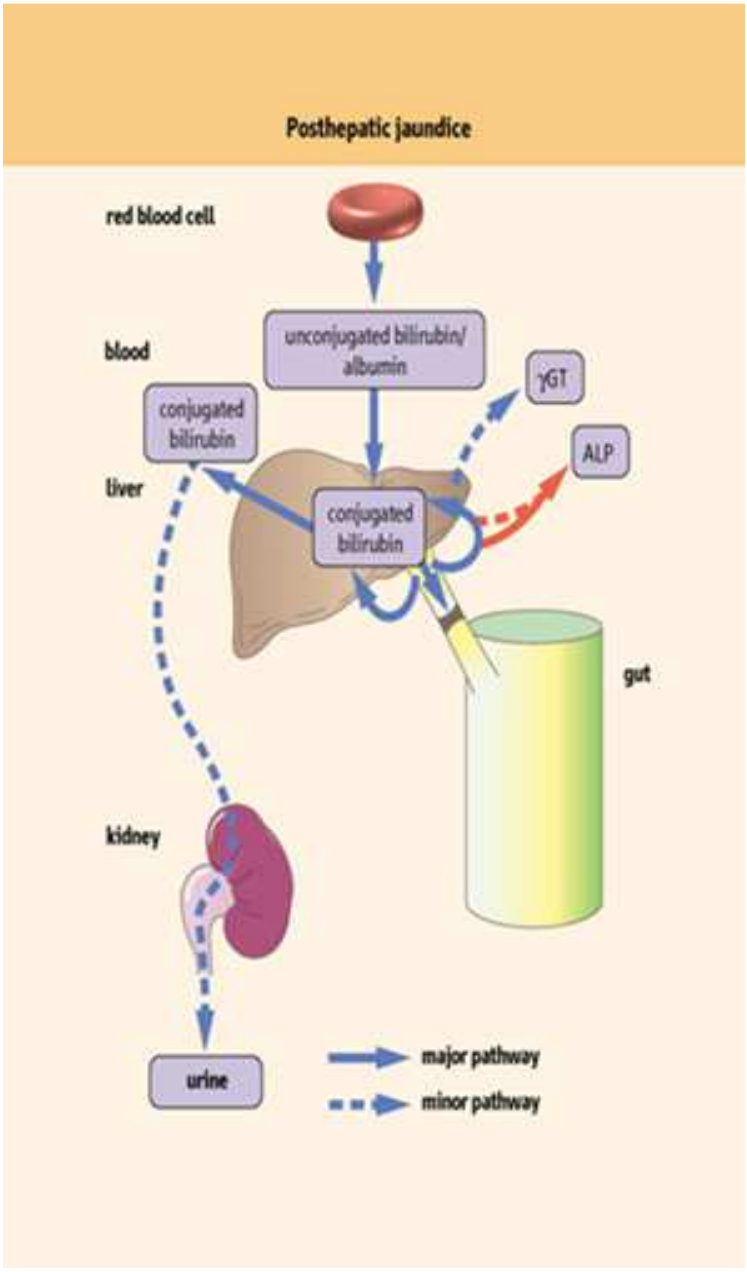
HEPATIC JAUNDICE

As a result of liver dysfunction there is impairment of uptake , conjugation and secretion of bilirubin , hence both direct and indirect bilirubin level increases.



POSTHEPATIC JAUNDICE

When there is obstruction to biliary tree , the conjugated biliribin level gets elevated.



CAUSES	JAUNDICE	BILIRUBIN IN SERUM	BILIRUBIN IN URINE	UBG IN URINE	UBG IN FAECES
Prehepatic	**Haemolytic**	**↑ indirect**	**No**	↑	↑
Hepatic	**Hepatic**	**↑Both direct and indirect**	**Yes**	↓	↓
Postheapatic	**Obstructive**	**↑Direct**	**Yes**	**No**	**No**

PHYSIOLOGIC CHANGES IN LIVER FUNCTION TESTS DURING PREGNANCY

Liver Test Results	Physiologic Changes Compared With Normal Range
Increased	Alkaline phosphatase , fibrinogen, fetoprotein, white blood cell, ceruloplasmin, cholesterol , alpha or/and beta globulins, triglycerides
Unchanged	Aminotransferases, prothrombin time
Decreased	Bilirubin, g-globulin, hemoglobin

DISEASE PRESENTATION AND THE TIMING OF ONSET DURING PREGNANCY

Disease Categories	First Trimester	Second Trimester	Third Trimester
Preexisting Liver Diseases	Chronic hepatitis B or C, autoimmune hepatitis, primary sclerosing cholangitis, Wilson disease, primary biliary cirrhosis, cirrhosis		
Newly Acquired Liver Diseases	Viral hepatitis, gallstones, drugs, sepsis, Budd-Chiari syndrome		
Diseases Related to Pregnancy	Hyperemesis gravidorum	Intrahepatic cholestasis	ICP, AFLP , preeclampsia, HELLP

AIMS

AND

OBJECTIVES

AIMS AND OBJECTIVES OF OUR STUDY :

- To analyze the maternal outcome in terms of mode of termination of pregnancy, maternal complications and mortality of jaundice complicating pregnancy.
- To identify the relation of maternal morbidity and mortality in relation to admission serum bilirubin level.
- To assess fetal outcome by perinatal mortality and morbidity.
- To identify the various etiologies and distribution of jaundice with reference to age , parity and trimesters.

Inclusion criteria : pregnant women affected by jaundice treated in Government Rajaji Hospital, Madurai.

Exclusion criteria : jaundice in pregnant women occurring due to septic etiology

REVIEW

OF LITERATURE

REVIEW OF LITERATURE

LIVER DISEASES RELATED TO PREGNANCY

HYPEREMESIS GRAVIDARUM

HG is defined as intractable nausea and vomiting during the first trimester of pregnancy. Most severe illness in the spectrum of vomiting in pregnancy.[1] In 0.3% of pregnancies it may lead to dehydration and electrolyte imbalance. But usually resolves by 16 to 18 weeks. In up to 10% of women, symptoms continues throughout pregnancy and resolve only with delivery of the fetus.[2,3]

The mechanism for Hyperemesis remains unclear. Proposed mechanisms include hormonal imbalance with elevated levels of human chorionic gonadotropin (HCG) and estrogen and decreased levels of prolactin, along with overactivity of the hypothalamic-pituitary-adrenal axis.[4,5] High level of HCG stimulates the thyroid gland and upregulate the secretary processes of the upper gastrointestinal tract.[4]

High levels of TNF a, IgG, IgM, C3, C4, natural killer cells, and extrathymic T cells observed in these women suggest that cytokine, T cell–mediated immune reactivation, immunoglobulin and complement play an important role in Hyperemesis.[6,7]

Risk factors includes –

a. Molar pregnancy

b. Multiple pregnancies
c. Preexisting diabetes or hyperthyroidism
d. Psychiatric disorders

Hyperemesis is a clinical diagnosis and based on exclusion of other underlying or newly acquired liver diseases.[2]

An abnormal LFT is seen in up to 50% of cases.[8] Transaminases are usually 2 to10 fold elevated. Rarely can be up to 20 times the normal with mild jaundice.

HG resolves in most patients with the replacement of electrolytes and glucose, rehydration, and nutritional support.

Parenteral hydrocortisone may lead to rapid resolution in severe cases, with slow improvement. Serious complications like malnutrition, esophageal tear ,[9,10] hyperthyroidism,[3] or even Wernicke encephalopathy caused by vitamin B12 deficiency can occur rarely.[11]

INTRAHEPATIC CHOLESTASIS OF PREGNANCY

Elevated bile acid (BA) levels during the late second or third trimester of pregnancy, with resolution after delivery associated with pruritus.

Risk factors for ICP are advancing maternal age, twin pregnancies, multiparity and oral contraceptive use. Prevalence is 1 in 1000 to 1 in 10000.

Cause of ICP is multifactorial, involving genetic, hormonal, and exogenous factors. Sex hormones can cause cholestatic effects through inhibition of the hepatocellular bile salt export pump (Bsep).[12] Also , pregnancy is associated with an abnormal metabolic response with impaired sulfation. The hepatic transport systems for biliary excretion are affected and saturated by the large amount of sulfated progesterone metabolites.[13,14] Genetic studies suggests that at least 10 different multidrug resistance–associated protein (MDR) 3 mutations have been identified in progressive familial intrahepatic cholestasis.[15]

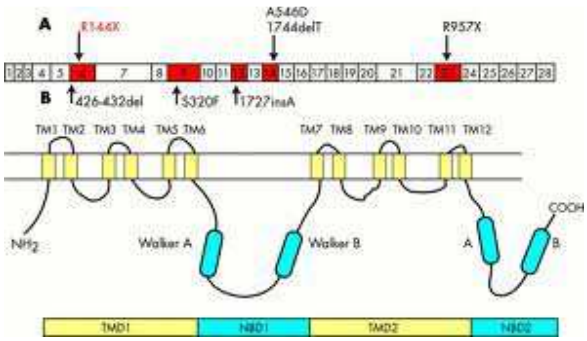
Pruritus usually starts during weeks 25 to 32 of pregnancy and resolves after delivery , excoriations caused by scratching are often noted on physical examination. Serum abnormalities detected 4 weeks after the onset of pruritus.[16] Early and specific abnormality is the elevation of serum total bilirubin acid level, and it is usually less than 5 mg/dL. Fetal morbidity and mortality are correlated with maternal BA levels.

Jaundice occurs in 10%to25%of patients and some may have diarrhea. Clinical improvement in ICP with the administration of cholestyramine,[17] dexamethasone,[18] and S-adenosyl-L-methionine.

Antihistamines,benzodiazepines, phenobarbital, and epomediol have no benefit. [19].

UDCA is the treatment of choice for ICP.

Close monitoring and early delivery after confirming fetal lung maturity may be the best way to prevent sudden antenatal death. Glantz and colleagues [24] have suggested using maternal BA levels of 40 mmol/L as a threshold for early delivery.



HELLP SYNDROME

HELLP syndrome is a multisystem disorder characterized by hemolysis, elevated levels of liver enzymes, and low platelet counts with or without preeclampsia.

The prevalence is estimated to be 0.6% of deliveries.[21] Risk factors are advanced maternal age, Whites and multiparity. It occurs in the third trimester in two-thirds of the Patients. Microangiopathic hemolytic anemia is associated with vascular endothelial injury, fibrin deposition in blood vessels, and platelet activation with platelet consumption. It is the hallmark of the syndrome but is not specific to this entity.

Periportal or focal parenchymal necrosis with hyaline deposition of fibrin material in the sinusoids is the characteristic histopathological finding.[22,23] Clinical symptoms includes epigastric or right upper quadrant pain, malaise, headache, nausea, and vomiting.

On physical examination, hypertension,generalized edema, and weight gain are common signs.

Three laboratory criteria:

- Thrombocytopenia
- Elevated aminotransferase levels
- Hemolysis. Several different classifications have been proposed.

Platelet count less than 100,000/mm3, aspartate aminotransferase levels greater than 70 U/L, and L-lactate dehydrogenase (LDH) levels greater than or equal to 600 U/L are helpful to make the diagnosis. The Mississippi classification is based on the degree of thrombocytopenia and the elevation of transaminase and LDH levels. Mississippi classification is for assessing the severity of the pathologic process. Frequent complications are disseminated intravascular coagulopathy (30%), abruptio placentae (16%), acute kidney injury (7.7%), aspiration pneumonia (7%), pulmonary edema (6%), acute respiratory distress syndrome, cardiopulmonary arrest (4%), cerebral hemorrhage (1.2%), and retinal detachment (0.9%). Rarely, severe ascites, subcapsular hematoma, hepatic failure, and hepatic rupture can occur (0.015%).[24,25]

Proteinuria is not required to make the diagnosis.

Computed tomography (CT) may show subcapsular hematomas, intraparenchymal hemorrhage, hepatic rupture, or infarction.

Transfer to a tertiary care center is advocated. If the pregnant woman is at or beyond 34 weeks' gestation or if there is any evidence of multiorgan dysfunction or severe complication, immediate induction of labor is recommended.

Close monitoring of the mother should be continued after delivery also.

Most laboratory values normalize in 48 hours after delivery of the fetus.

Causes of perinatal mortality include abruptio placenta,asphyxia, and prematurity. Subsequent pregnancies in patients with HELLP syndrome carry a high risk of complications including recurrent HELLP.[26,27]

ACUTE FATTY LIVER OF PREGNANCY

AFLP occurs in 1 in 7000 to 16000 pregnancies.[28,29]

Associated with microvesicular fatty infiltration of the liver, hepatic failure, and encephalopathy. It occurs commonly in the third trimester of pregnancy.

Deficiencies of the enzymes of mitochondrial fatty acid beta oxidation (FAO) results in fatty infiltration of the liver. The most commonly enzyme deficiency is long-chain 2-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency. The defect is in the alpha subunit of the mitochondrial protein is associated with G1528C or E474Q mutations.[30,31] Natarajan and colleagues [32] demonstrated that placental mitochondrial function is compromised in AFLP, which may lead to free radical production and accumulation of fatty acids in the placenta, resulting in maternal hepatocyte stress and mitochondrial dysfunction leading to acute liver failure.

40% to 50% of patients with AFLP are nulliparous with an increased incidence in twin pregnancies.[33] Symptoms includes anorexia, nausea, emesis, malaise, fatigue, and headache.

On physical examination, the patient may have jaundice,hypertension, edema, and hepatic encephalopathy.

Serum aminotransferase levels vary from 300 to 500 U/L. The total bilirubin concentration is usually less than 5 mg/dL. Other laboratory abnormalities include anemia, leukocytosis, normal or low platelet counts, coagulopathy with or without DIC, hypoalbuminemia, hypoglycemia,and acute kidney injury.

Management includes hospitalization for stabilization of hypertension and DIC, seizure prophylaxis. Fetal monitoring followed by immediate delivery of the fetus or termination of the pregnancy along with intensive support. The aminotransferase levels and encephalopathy improve within 72 hours of delivery. Most recover in 1 to 4 weeks post partum.

Maternal mortality is 3% to 12% and fetal mortality is 15% to 66%. The strong association of AFLP with LCHAD deficiency in the fetus suggests a necessity of neonatal testing for enzymatic defects of FAO. Women who are carriers of the LCHAD mutation have an increased risk of recurrence of AFLPin 20%to 70%of pregnancies.

Elevated level of serum bilirubin causes vasoconstrictive effect on the placental vessels and cardiotoxic effect resulting in fetal asphyxia and intrauterine

death . Also elevated bilirubin produce cellular effect which stimulates uterine contractility and sensitizes myometrium to oxytocin resulting in preterm labour.

The fetomaternal complications of jaundice in pregnancy are not only due to jaundice alone but largely due to the underlying causes of these conditions. Thus the management depends on the underlying cause.

Careful examination and vigilance is required to detect the early sign of hepatic dysfunction and to differentiate these from the physiological changes during pregnancy. Management of jaundice in pregnancy requires a combined effort of physician, gastroenterologist, obstetrician and on rare occasions, a liver transplant team.

MATERIALS

AND METHODS

MATERIALS AND METHODS

Sixty five women with jaundice complicating pregnancy admitted and treated at Government Rajaji Hospital , Madurai from september 2011 to september 2012 were studied.

- A detailed history including patient's age , socioeconomic status , booking , parity and details of menstrual history to arrive at the expected date of delivery was obtained.
- Patients were enquired in detail about their complaints and duration like nausea , vomiting , pruritus , anorexia , yellow coloured urine , pale stools , edema legs , bleeding tendency , joint pain , fever and others.
- Past history of jaundice especially in previous pregnancy and history of blood transfusion were elicited.
- systemic and obstetric examinations were carried out.
- Investigations included liver function tests, serum billirubin, SGOT, SGPT, alkaline phosphatase, Viral markers, prothrombin time (PT), partial thromboplastin time (PTT), bleeding time (BT), clotting time (CT), platelet count and ultrasound abdomen were carried out as and when required.
- HIV screeing was done in all patients.
- Medical gastroenterologist opinion was obtained for all cases.

- Labour was closely monitored. Jaundice perse was not an indication for cesarean section. Vaginal delivery with close monitoring was preferred and cesarean sections were done only for obstetric indication. After cross matching fresh blood was kept ready as alteration in coagulation profile was expected in jaundice complicating pregnancy.
- Atonicity was managed with oxytocin drip , injection methergin and injection 15 methyl PGF2α.
- Patient were kept in the labour ward for close observation. Clotting time was repeated hourly if it was prolonged till it becomes normal.
- Soon after delivery all babies were assessed by paediatrician. Alive or dead , sex , gestational age at birth , weight , apgar score and presence or absence of any congenital anomalies were looked for and noted. As per paediatrician opinion sick babies were admitted in preterm ward for intensive care.
- Of the 65 women, 28 had viral hepatitis,8 AFLP, 6 HELLP , 3 cholestatic , 1 hyperemesis ,1 haemolytic anaemia , 1 cirrhosis and 1 Gilberts.
- The maternal outcome was noted in terms of the mode of termination of pregnancy, maternal complications and maternal mortality.The relation of maternal morbidity and mortality to the admission serum bilirubin level was analysed.

- To identify the various etiologies and distribution of jaundice with reference to age , parity and trimesters.
- Fetal outcome was assessed by perinatal morbidity and mortality

OBSERVATIONS

AND RESULT

OBSERVATIONS AND RESULT

The incidence of jaundice in India varies from 0.4 to 0.9/1000 deliveries. According to this study the incidence of jaundice is 2/1000 deliveries. Singh et al **1** reported 1.03/1000 incidence while Kamalajayaram and Rama Devi **2** reported 0.4/1000 incidence. Of the 65 women studied 58.5% were in 21 to 25 yrs of age (TABLE 1). Mean age is 23 yrs. About 47.7% were primi and 41.5% were second gravida (TABLE 3). 70.7% were in third third trimester (TABLE 4).

Etiologies for jaundice

Of the 65 women, 28 had viral hepatitis (43.1%), 8 AFLP (12.3%), 6 HELLP (9.2%), 3 cholestatic , 1 hyperemesis ,1 haemolytic anaemia , 1 cirrhosis and 1Gilberts.***(table 10)*** Among the 28 viral hepatitis 60.7% was due to hepatitis E, hepatitis B 28.6% and hepatitis A 10.7. Two deaths were due to hepatitis E. Of the 6 HELLP cases 3 expired (50% mortality) .

Relation with serum bilirubin level

Maternal mortality was directly related to the level of serum bilirubin as shown in (Table 9). 59 women were discharged in improved condition - 21 undelivered and 38 delivered. Initial serum bilirubin level of > 13 lead to 50%

mortality. About 33.8% of women had an initial serum bilirubin level of about 5 – 10 mg / dl.

Pregnancy outcome

Among the 65 women studied 43 women got delivered (66.2%). 22 women remained undelivered (TABLE 4). Of these 22 women, one expired due to hepatic encephalopathy during 3rd trimester. Others improved and were discharged. Of the 43 women delivered , 28 was vaginal delivery (65.1) , 1 outlet , 1 VBAC , 12 LSCS (25.6%) and 1 spontaneous expulsion.

4 cases of atonic PPH was observed. 3 were following vaginal delivery and one following outlet forceps delivery.

One atonic PPH ended up in hysterectomy she subsequently deteriorated and expired due to hepatic encephalopathy.

Maternal outcome

28% developed hepatic encephalopathy, 28% ARF , 22% atonic PPH , 17% abruption and 5% DIC. (table 12)

Cause of death

In our study , six women expired . Hepatic encephalopathy (66.7) was the cause in 4 out of 6 women who died. One women died of hepatorenal failure (16.7) and one due to acute cholangitis (16.7).(table 13)

Fetal outcome

0f the 43 women delivered , one was a twin delivery but only one baby survived. 86.4% born alive and 11..4 % were intrauterine death (table 8). 52.3 % were male babies and 47.4 % were female babies (table 7). 68.2 % were preterm babies and 29.5 % were term babies (table 6). 52.3% of babies weighed < 2.5kg and 47.7 % of babies weighed 2.5 – 3 kg.

ANALYSIS

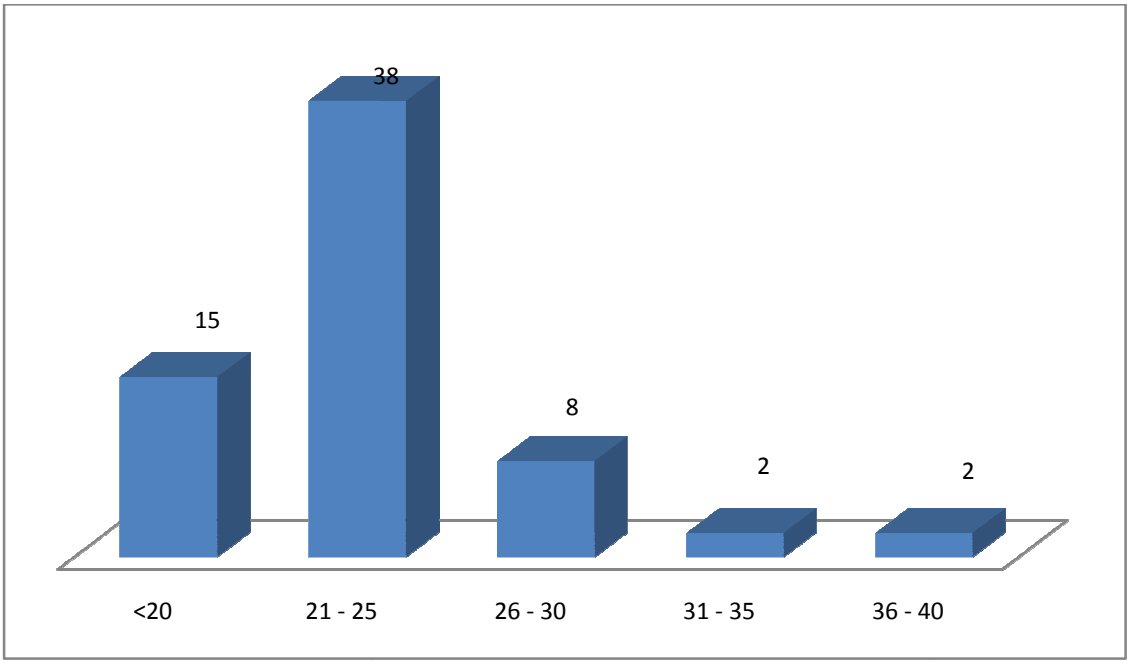
AGE DISTRIBUTION (TABLE 1)

AGE		PERCENTAGE
<20	15	23
21-25	38	58.5
26-30	8	12.3
31-35	2	3.07
36-40	2	3.07

Of the 65 women studied 58.5% were in 21 to 25 yrs of age . Mean age is 23 yrs.

Mean	23.65
Median	23.00
Mode	24[a]
Std. Deviation	3.797
Variance	14.420
Range	18

AGE DISTRIBUTION (CHART 1)



Of the 65 women studied 58.5% were in 21 to 25 yrs of age . Mean age is 23 yrs.

AGE

	Frequency	Percent	Valid Percent	Cumulative Percent
18	4	6.2	6.2	6.2
19	4	6.2	6.2	12.3
20	6	9.2	9.2	21.5
21	4	6.2	6.2	27.7
22	6	9.2	9.2	36.9
23	9	13.8	13.8	50.8
24	10	15.4	15.4	66.2
25	10	15.4	15.4	81.5
26	1	1.5	1.5	83.1
27	2	3.1	3.1	86.2
28	4	6.2	6.2	92.3
29	1	1.5	1.5	93.8
31	1	1.5	1.5	95.4
32	1	1.5	1.5	96.9
36	2	3.1	3.1	100.0
Total	65	100.0	100.0	

TRIMESTER DISTRIBUTION (TABLE 2)

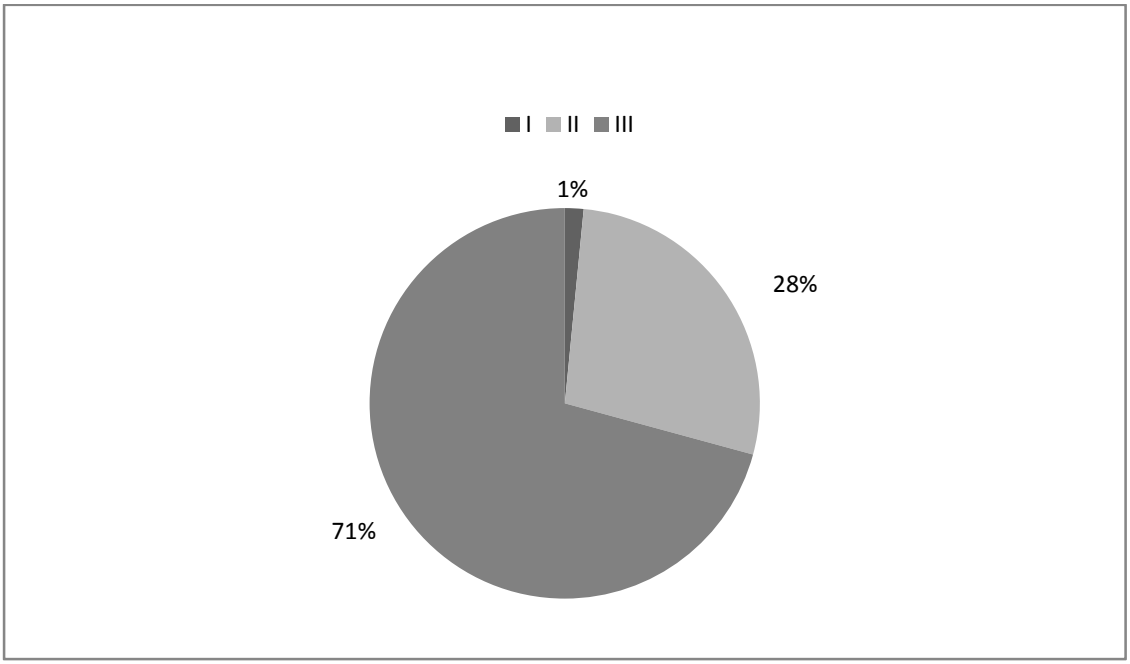
TRIMESTER	TOTAL	PERCENTAGE
I	1	1.53
II	18	27.7
III	46	70.7

The occurrence of jaundice was high during third trimester . 70.7% were in third trimester.

TRIMESTER DISTRIBUTION

	Frequency	Percent	Valid Percent	Cumulative Percent
I	1	1.5	1.5	1.5
II	18	27.7	27.7	29.2
III	46	70.8	70.8	100.0
Total	65	100.0	100.0	

TRIMESTER DISTRIBUTION(CHART 2)



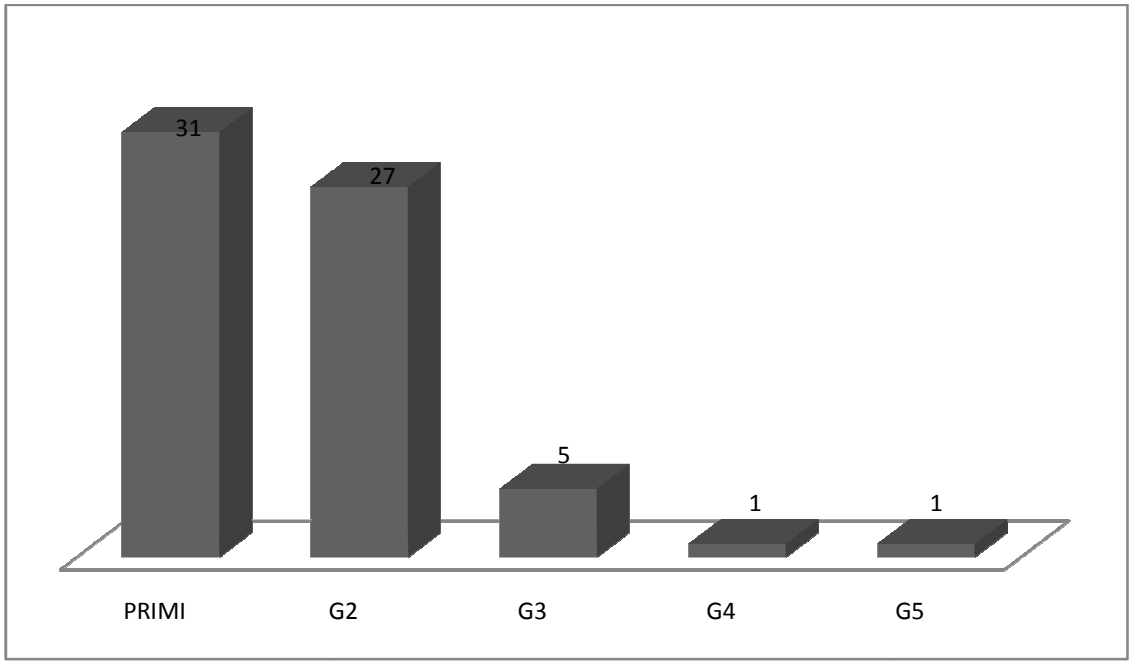
PARITY DISTRIBUTION (TABLE 3)

PARITY		PERCENTAGE
PRIMI	31	47.7
G2	27	41.5
G3	5	7.7
G4	1	1.53
G5	1	1.53
TOTAL	65	

Of the total 65 women studied , 47.7% were primigravida and 41.5% were second gravida.

	Frequency	Percent	Valid Percent	Cumulative Percent
G3	5	7.7	7.7	49.2
G4	1	1.5	1.5	50.8
G5	1	1.5	1.5	52.3
PRIMI	31	47.7	47.7	100.0
Total	65	100.0	100.0	

PARITY DISTRIBUTION (CHART 3)



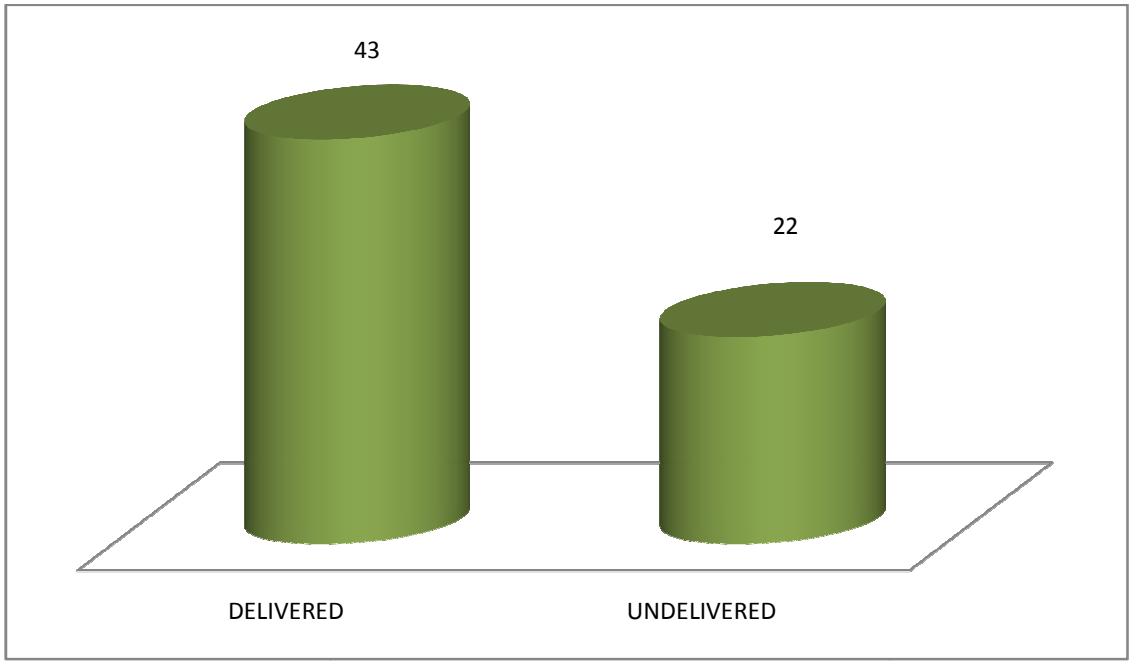
Of the total 65 women studied , 47.7% were primigravida and 41.5% were second gravida.

PREGNANCY OUTCOME (TABLE 4)

	TOTAL	PERCENTAGE
DELIVERED	43	66.2
UNDELIVERED	22	33.8
TOTAL	65	

43 out of 65 patients delivered (66.2%). 22 remained undelivered. Of these 22, one expired due to hepatic encephalopathy during 3rd trimester. Others improved and were discharged.

PREGNANCY OUTCOME (CHART 4)



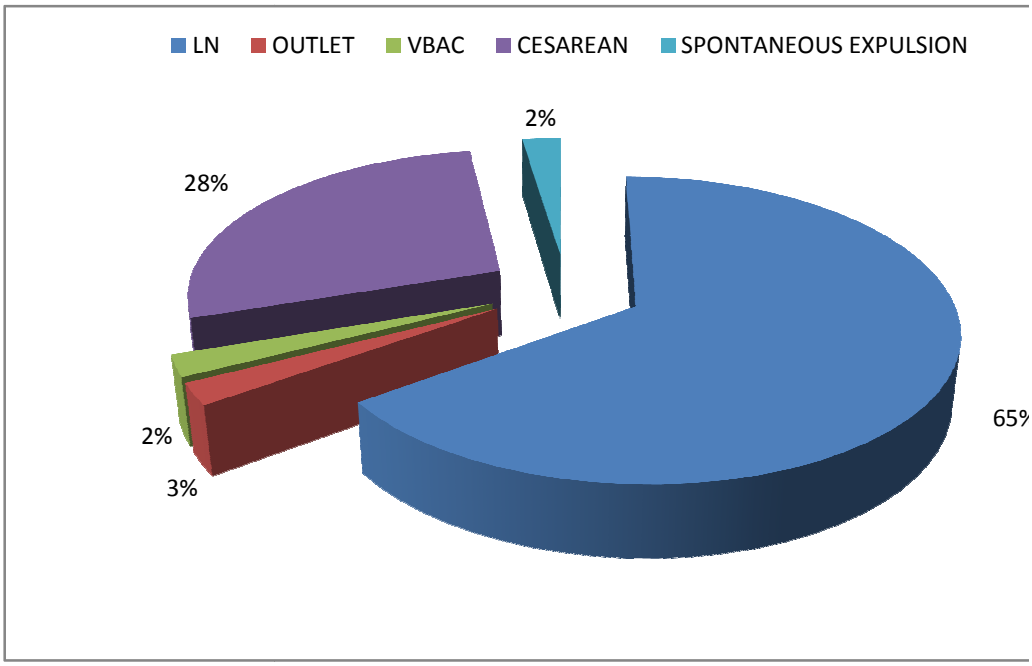
MODE OF DELIVERY (TABLE 5)

MODE OF DELIVERY		PERCENTAGE
LABOUR NATURAL	28	65.1
OUTLET	1	2.3
VBAC	1	2.3
CESAEREAN	12	25.6
SPONTANEOUS EXPULSION	1	2.3
TOTAL	43	

Of the 43 delivered , 28 was labour natural (65.1) , 1 outlet , 1 VBAC , 12 LSCS (25.6%) and 1 spontaneous expulsion.

4 cases of atonic PPH was observed. 3 were following labour natural and one following outlet forceps delivery.

MODE OF DELIVERY (CHART 5)

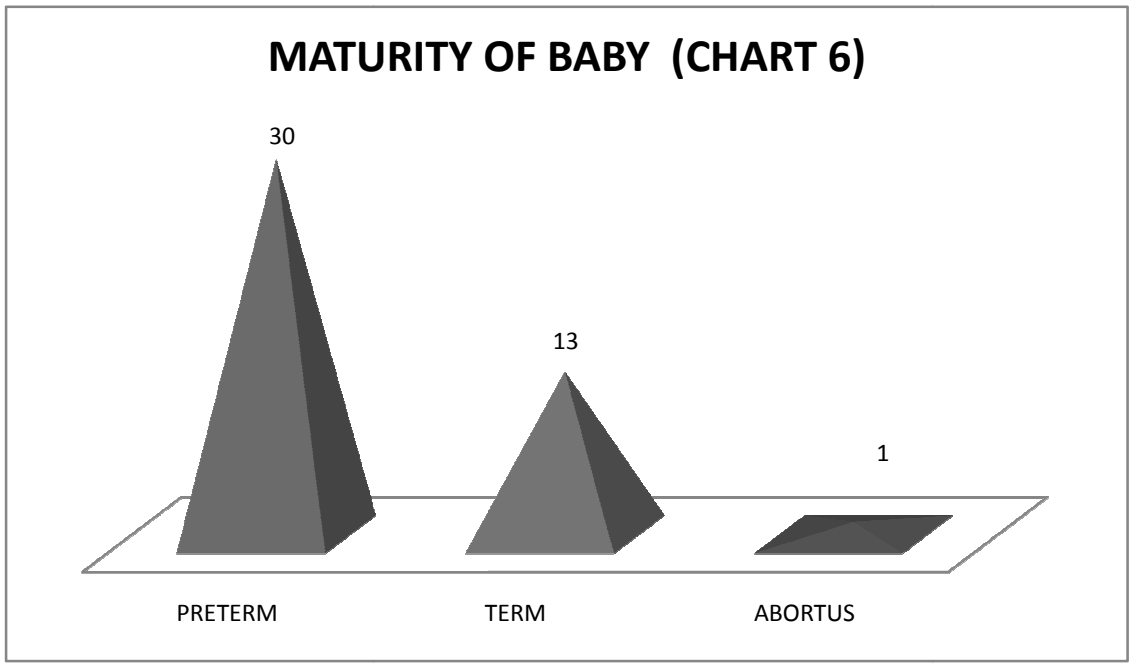


MODE OF DELIVERY	Frequency	Percent	Valid Percent	Cumulative Percent
LABOUR NATURAL	28	43.1	43.1	43.1
LSCS	12	18.5	18.5	61.5
OUTLET FORCEPS	1	1.5	1.5	63.1
SPONTANEOUS EXPULSION	1	1.5	1.5	64.6
UNDELIVERED	22	33.8	33.8	98.5
VBAC	1	1.5	1.5	100.0
TOTAL	65	100.0	100.0	

MATURITY OF BABY (TABLE 6)

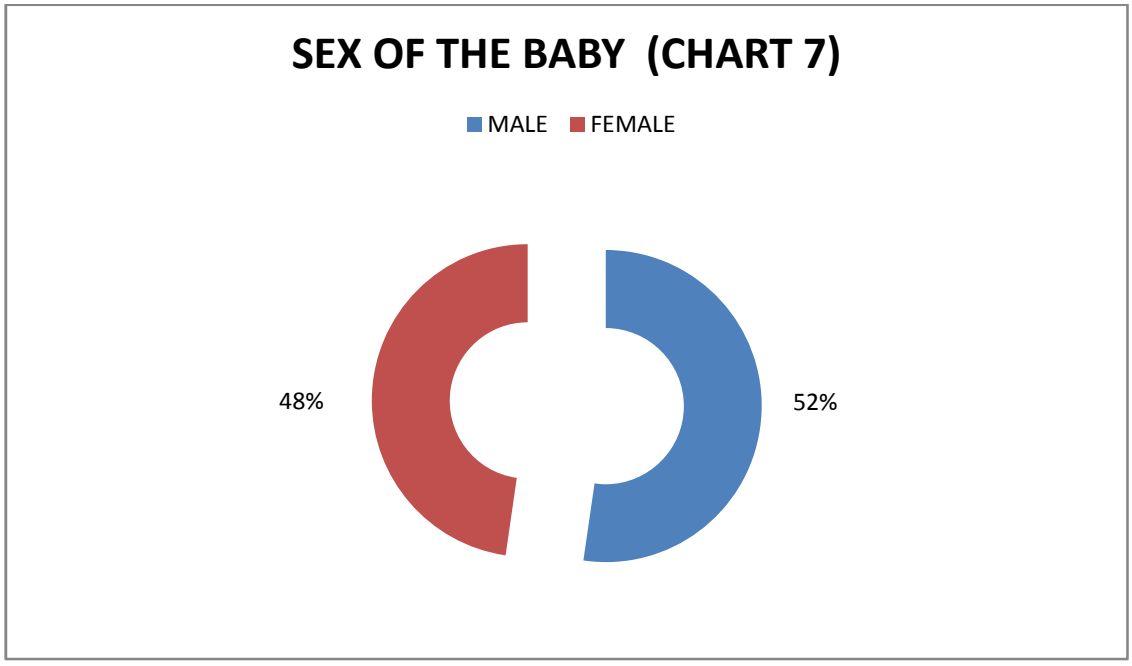
MATURITY OF BABY	TOTAL	PERCENTAGE
PRETERM	30	68.2
TERM	13	29.5
ABORTUS	1	2.3
TOTAL	44	

30 Babies out off 44 were premature. 2 out of this 30 babies were intrauterine deaths. Remaining 28 admitted in preterm ward for intensive care. Inspite of intensive care 4 out of 28 babies expired.



SEX OF THE BABY (TABLE 7)

SEX OF THE BABY	TOTAL	PERCENTAGE
MALE	23	52.3
FEMALE	21	47.7
TOTAL	44	



FETAL OUTCOME (TABLE 8)

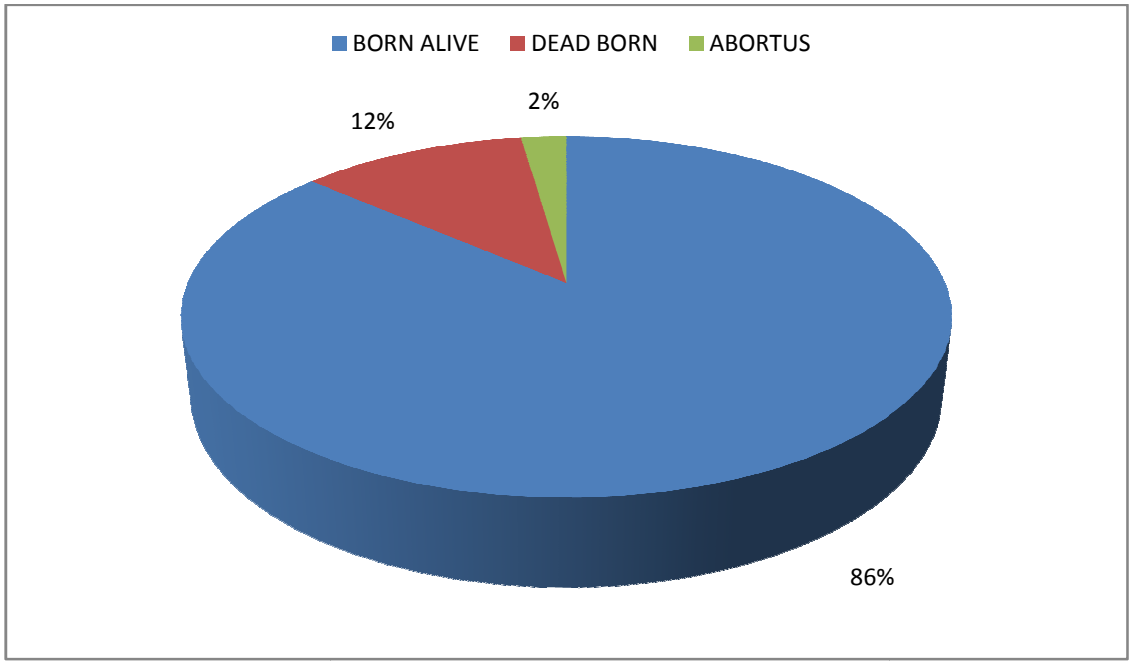
FETAL OUTCOME	TOTAL	PERCENTAGE
BORN ALIVE	38	86.4
DEAD BORN	5	11.4
ABORTUS	1	2.3

0f the 43 delivered , one was a twin delivery but only one baby survived. 86.4% born alive and 11..4 % were intrauterine death . 52.3 % were male babies and 47.4 % were female babies. 68.2 % were preterm babies and 29.5 % were term babies.

Of 12 babies expired, 6 babies were preterm and 6 babies were term. 2 out of 6 preterm babies were dead born and 3 out of 6 term babies were dead born.

Inspite of intensive neonatal care 7 babies expired.

FETAL OUTCOME (CHART 8)



LEVEL OF INITIAL BILIRUBIN (TABLE 9)

INITIAL BILIRUBIN	TOTAL	PERCENTAGE
<5	31	47.7
5-10	22	33.8
10-15	10	15.4
>15	2	3.1

Maternal mortality and morbidity was directly related to the initial level of serum bilirubin . Initial serum bilirubin level of > 13 lead to 50% mortality. About 33.8% of women had an initial serum bilirubin level of about 5 – 10 mg / dl.

Keeping the initial bilirubin level at admission as 10 mg/dl , the maternal outcome was poor and high mortality rate was seen when the bilirubin level exceeds 10 mg/dl. It is statistically significant.

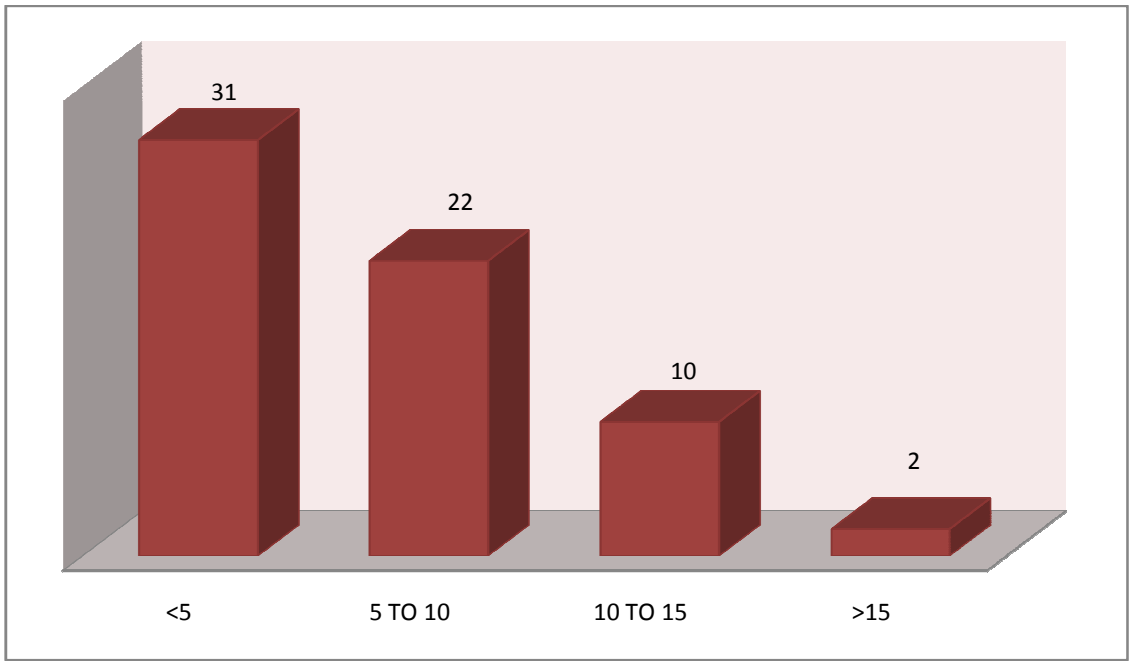
LEVEL OF INITIAL BILIRUBIN

When the initial serum bilirubin level is > 13the mortality rate was upto 50%.

Bilirubin level	Frequency	Percent	Valid Percent	Cumulative Percent	Deaths
<13	57	87.7	87.7	87.7	3
>13	8	12.3	12.3	100.0	3
Total	65	100.0	100.0		

Bilirubin level	Frequency	Percent	Valid Percent	Cumulative Percent
<10	55	84.6	84.6	84.6
>10	10	15.4	15.4	100.0
Total	65	100.0	100.0	

INITIAL BILIRUBIN LEVEL (CHART 9)



STATISTICAL SIGNIFICANCE OF INITIAL BILIRUBIN LEVEL

(using chi square test)

BILIUBIN LEVEL	EXPIRED	RECOVERED	TOTAL
A (<10)	3	52	55
B (>10)	3	7	10
TOTAL	6	59	65
	0.092	0.907	

INITIAL BILIRUBIN LEVE L	EXPIRED	RECOVERED	TOTAL
< 10	O = 3 E = 5.06 2.06	0 = 52 E = 49.9 2.1	55
> 10	O = 3 E = 0.92 2.08	0 = 7 E = 9.07 2.07	10
TOTAL	6	59	65

$\chi^2 = 6.104$

From the probability table of X^2, probability (P) value is found to be around 0.01.

Hence the difference in the recovery pattern between the two groups is statistically significant.

HENCE THE INITIAL BILIRUBIN LEVEL AT ADMISSION > 10 IS ASSOCIATED WITH POOR MATERNAL OUTCOME AND HIGH MATERNAL MORTALITY.

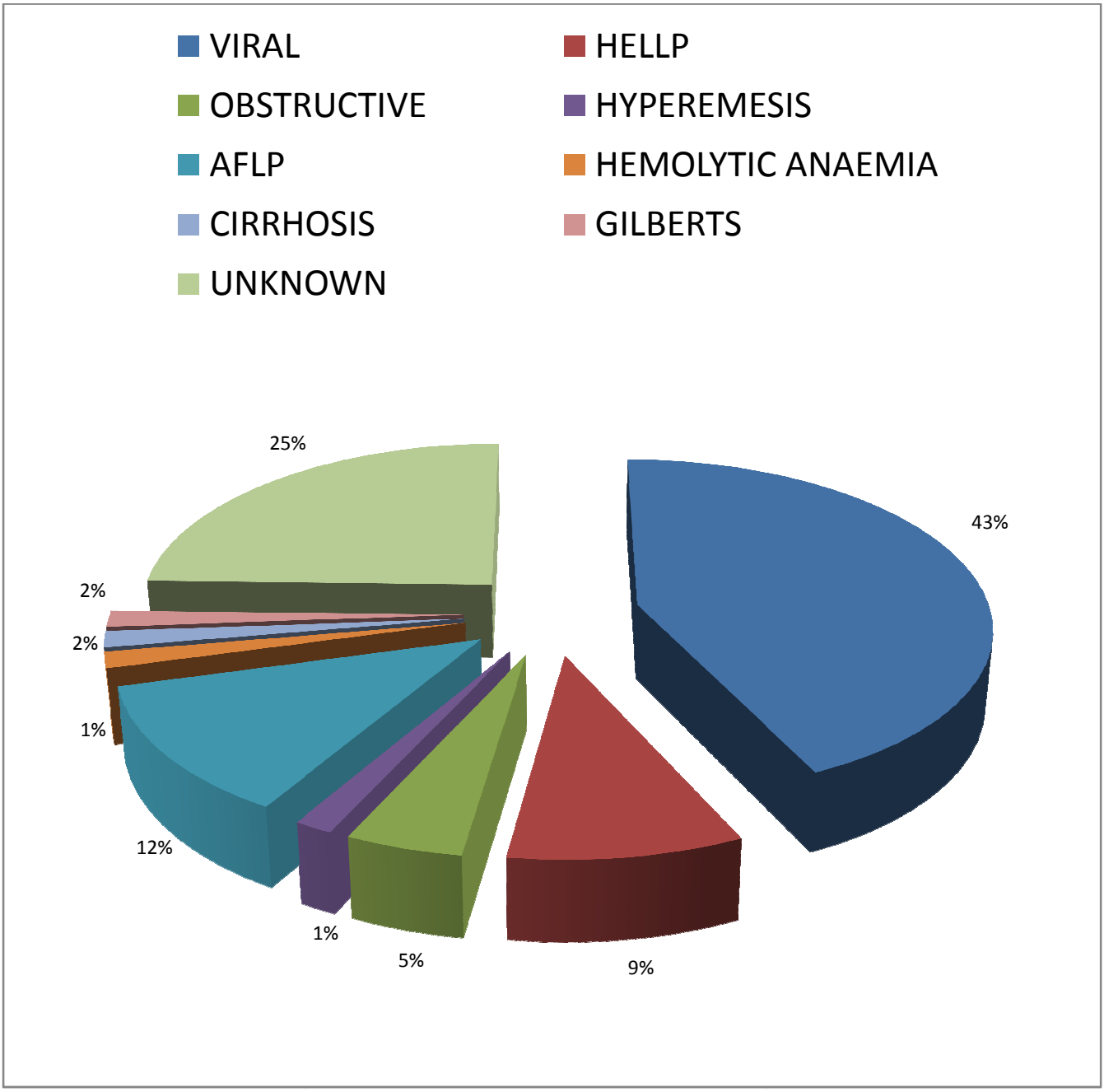
ETIOLOGY OF JAUNDICE (TABLE 10)

ETIOLOGY OF JAUNDICE	TOTAL	PERCENTAGE
VIRAL	28	43.1
AFLP	8	12.3
HELLP	6	9.2
OBSTRUCTIVE	3	4.62
HYPEREMESIS	1	1.5
HAEMOLYTIC ANAEMIA	1	1.5
CIRRHOSIS	1	1.5
GIBERTS	1	1.5
UNKNOWN	16	

Of the 65 women studied , 28 had viral hepatitis (43.1%), 8 AFLP (12.3%), 6 HELLP (9.2%), 3 cholestatic , 1 hyperemesis ,1 haemolytic anaemia , 1 cirrhosis and 1Gilberts syndrome.

ETIOLOGY	FREQUENCY	PERCENT	VALID PERCENT	CUMULATIVE PERCENT
AFLP	8	12.3	12.3	12.3
CHOLESTATIC	3	4.6	4.6	16.9
CIRRHOSIS WITH PORTAL HYPERTENSENSION	1	1.5	1.5	18.5
GILBERT	1	1.5	1.5	20.0
HAEMOLYTIC	1	1.5	1.5	21.5
HELLP	6	9.2	9.2	30.8
HEPATITIS A	3	4.6	4.6	35.4
HEPATITIS B	8	12.3	12.3	47.7
HEPATITIS E	17	26.2	26.2	73.8
HYPEREMESIS	1	1.5	1.5	75.4
UNKNOWN	16	24.6	24.6	100.0
TOTAL	65	100.0	100.0	

ETIOLOGY OF JAUNDICE (CHART 10)



TYPES OF VIRAL ETIOLOGIES (TABLE 11)

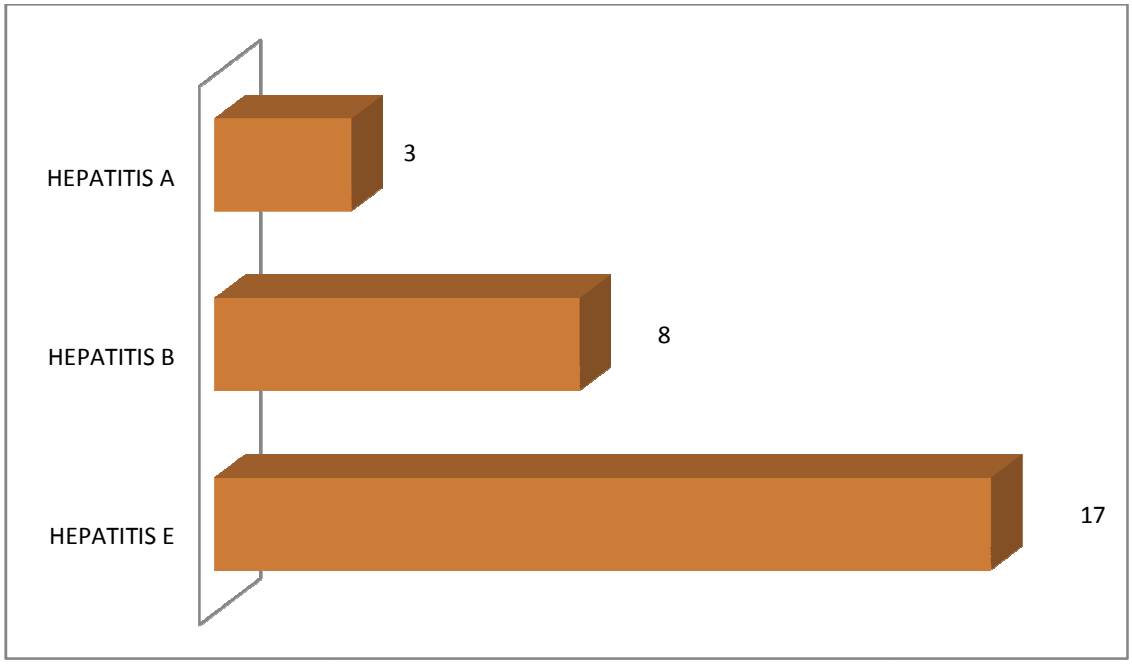
VIRAL	TOTAL	PERCENTAGE
E	17	60.7
B	8	28.6
A	3	10.7

Of the 65 women studied , 28 had viral hepatitis (43.1%) . Among the 28 viral hepatitis 60.7% was due to hepatitis E , hepatitis B 28.6% and hepatitis A 10.7.

Out of 12 fetal deaths , 5 babies were born to women with viral hepatitis. Of these 5 , 4 were due to viral hepatitis E.

Of the 28 women with viral hepatitis two women expired due to hepatic encephalopathy. And both were hepatitis E virus.

TYPE OF VIRAL ETIOLOGIES (CHART 11)

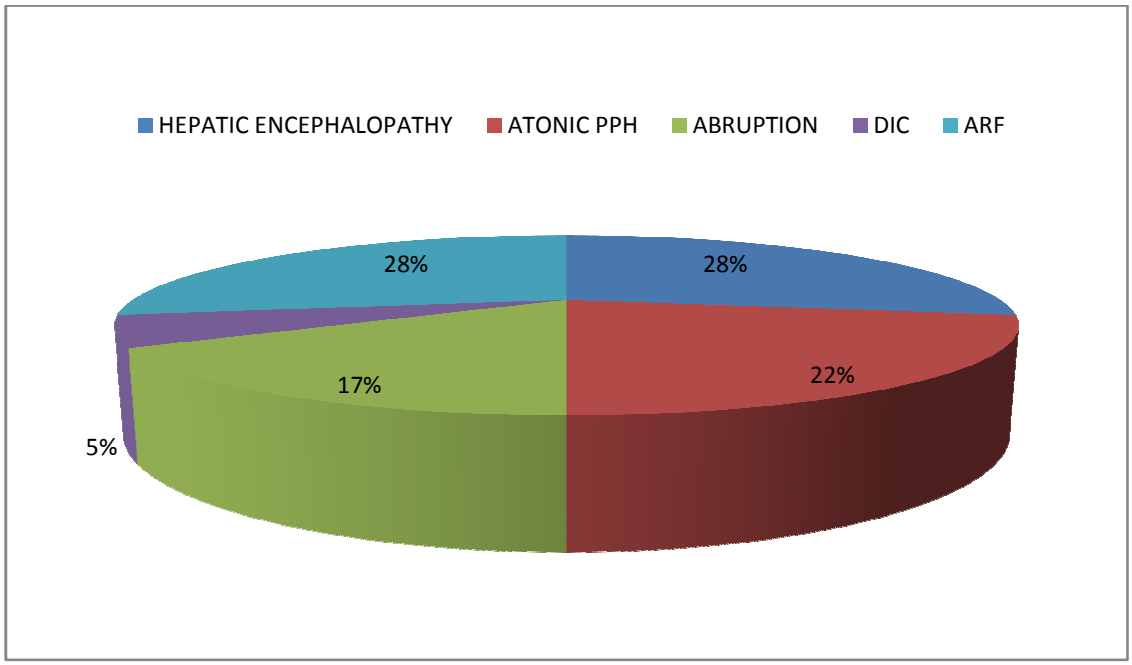


MATERNAL COMPLICATIONS (TABLE 12)

MATERNAL COMPLICATIONS		PERCENTAGE
HEPATIC ENCEPHALOPATHY	5	28
ARF	5	28
ATONIC PPH	4	22
ABRUPTION	3	17
DIC	1	5

28% developed hepatic encephalopathy, 28% ARF , 22% atonic PPH , 17% abruption and 5% DIC.

MATERNAL COMPLICATIONS (CHART 12)

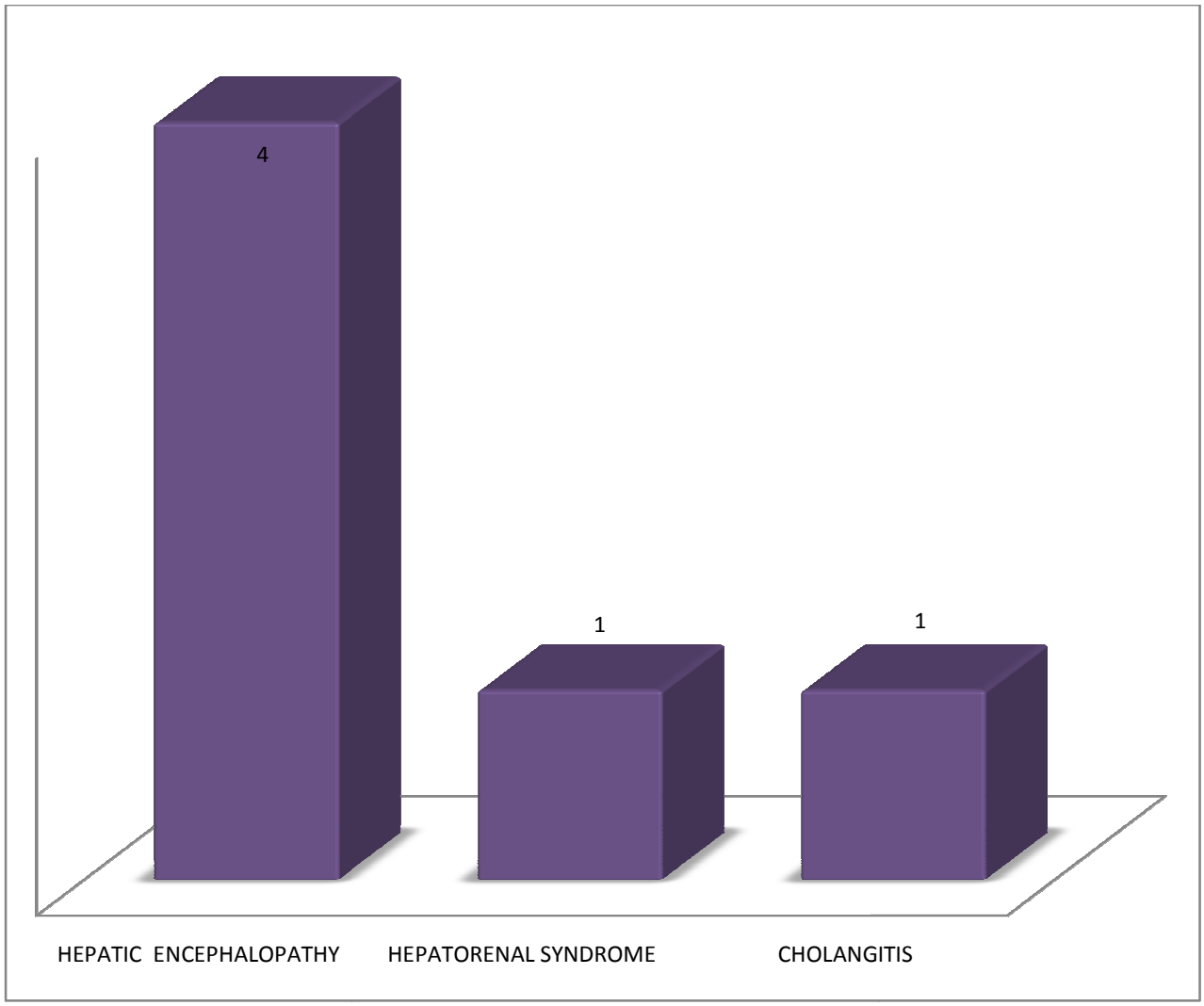


CAUSE OF DEATH (TABLE 13)

CAUSE OF DEATH		PERCENTAGE
HEPATIC ENCEPHALOPATHY	4	66.7
HEPATORENAL SYNDROME	1	16.7
CHOLANGITIS	1	16.7

Six women expired. Hepatic encephalopathy (66.7 %) was the cause in 4 out of 6 women who died. 0ne women died of hepatorenal failure (16.7 %) and one due to acute cholangitis (16.7 %)

CAUSE OF DEATH (TABLE 13)

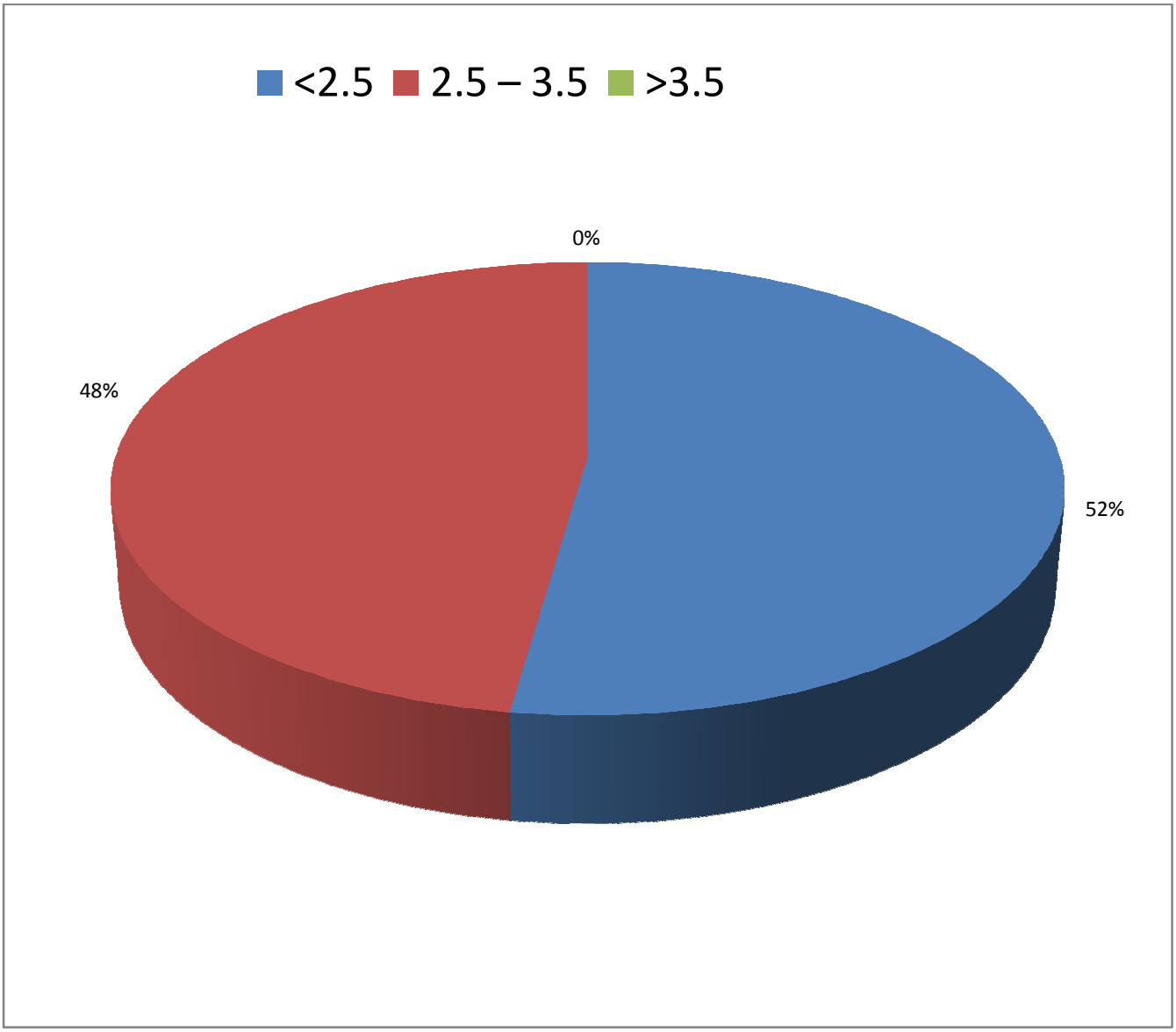


BIRTH WEIGHT (TABLE 13)

BIRTH WEIGHT		PERCENTAGE
<2.5	23	52.3
2.5 – 3.5	21	47.7
>3.5	-	

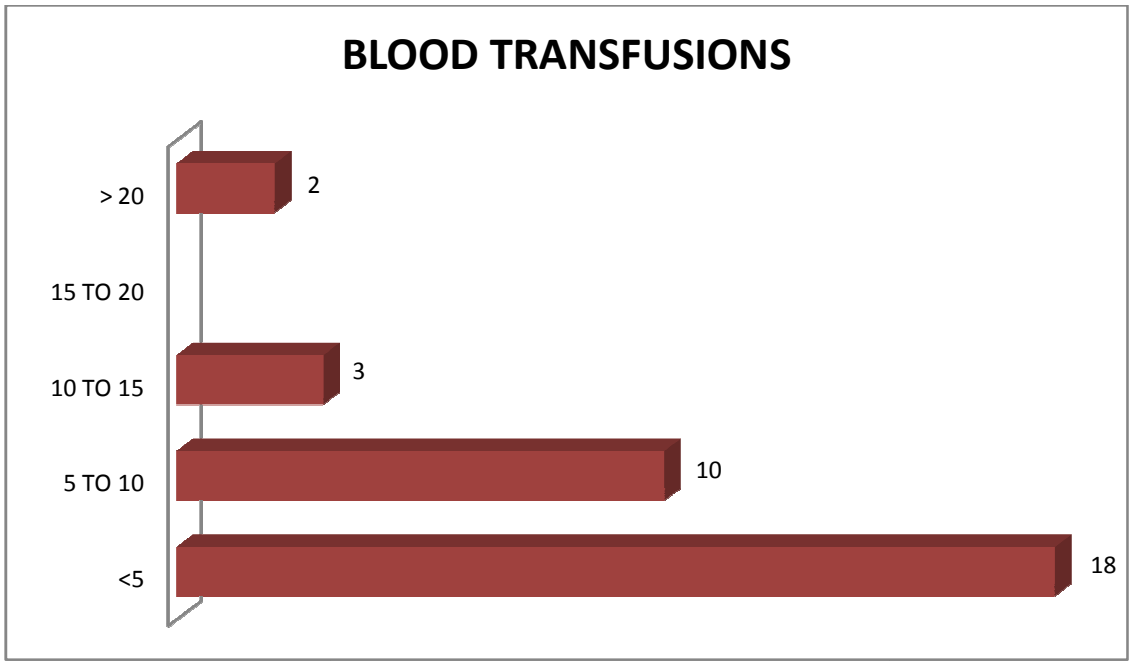
Maximum weight among the 44 babies was 3.5 kg

BIRTH WEIGHT (CHART 13)



BLOOD TRANSFUSIONS (TABLE 14)

TRANSFUSIONS	
<5	18
5-10	10
10-15	3
15-20	
>20	2



PERINATAL DEATHS (TABLE 15)

ETIOLOGY	PRETERM / TERM	MODE OF DELIVERY	SEX OF THE BABY	SERUM BILIRUBIN
VIRAL 5 (41.7 %)	PRETERM -2 TERM -3 (2 OUT 3 WAS DEAD BORN)	LABOUR NATURAL -4 OUTLET 1	MALE - 2 FEMALE -3	<5 - 1 5 to10 -2 10 to15 -2
HELLP 3 (25 %)	PRETERM - 3 (2 OUT 3 WAS DEAD BORN)	LABOUR NATURAL – 2 LSCS - 1	MALE -3	5 to10- 1 10 to15 -1 >15 -1
CHOLESTASIS 1 (8.3 %)	PRETERM - 1	LSCS - 1	MALE - 1	5 to10- 1
AFLP 2 (16.7 %)	TERM -2	LABOUR NATURAL – 1 LSCS - 1	MALE -1 FEMALE -1	<5 -1 10 to15 -1

UNKNOWN 1 (8.3 %)	TERM - 1 (DEAD BORN)	LSCS	MALE - 1	10 to15-1

Among the 12 babies expired 6 babies were term and 6 babies were preterm. 2 out of 6 preterm babies were dead born and 3 out of 6 term babies were dead born.

Out of 12 fetal deaths , 5 babies were born to women with viral hepatitis. Of these 5 , 4 were due to viral hepatitis E.

30 babies admitted for intensive care. Inspite of intensive neonatal care 7 babies expired.

5 deaths have occurred when the maternal initial bilirubin was in the range of 10 - 15 mg/dl.

COMPARISON WITH REPORTED MATERNAL DEATHS DUE TO JAUNDICE

Authors	Year	Percentage of deaths due to jaundice amongst total maternal deaths
Kamalajayaram and Rama Devi	1988	12.4
Rao and Rudra	2001	15.8
Roychowdhary et al	1990	13.37
Bera and Sengupta	1992	19.9
Sapre and Joshi	1999	04.99
Trivedi et al	2003	29.3
According to this study	2012	7.9

DISCUSSION

DISCUSSION

The incidence of jaundice in India varies from 0.4 to 0.9/1000 deliveries. According to this study incidence is 2/1OOO deliveries. Singh et al [34] reported 1.03/1000 incidence while Kamalajayaram and Rama Devi [35] reported 0.4/1000 incidence. Jaundice occurring in pregnancy can be due to acute yellow atrophy of liver due to infective hepatitis of A, B, C, D or E type. Cholestatic jaundice is also common during pregnancy, in which serum bilirubin levels of up to 6 mg% are seen with either minimal or no increase in serum enzyme levels. It is associated with prematurity in 19.5% and a perinatal mortality rate of 30%.

HELLP syndrome is present in 3-10% of preeclamsia. It is associated with weight gain and edema in 60%, maternal mortality of 20%, DIC in 4-38%, neonatal mortality rate of 31%, and rupture and hematoma of the liver in 2% [36].

Acute fatty liver during pregnancy usually occurs in the 3rd trimester. Preeclampsia is associated in 50-100% of cases. There is moderately increased liver enzyme level of <1000 IU/mL, bilirubin level of 1-10mg% and hypoglycemia. The maternal mortality is 18% while preterm labor is increased and the perinatal mortality is 23% [36].

Jaundice in pregnancy is associated with high maternal and perintal mortality

rates.High perinatal mortality rate of 45.45% was observed by Singh et al [34].

According to this study percentage of fetal deaths due to jaundice amongst total perinatal deaths is 2%. Maternal mortality reported by various authors. Kamalajayaram and Rama Devi [35] reported 33.3% maternal mortality and Singh et al [34] reported 10%.

According to this study percentage of maternal deaths due to jaundice amongst total maternal deaths is 7.9 % which is nearly comparable with the study conducted by Sapre and Joshi .

Hepatorenal failure, encephalopathy.DIC and postpartum hemorrhage were responsible for the deaths. According to this study hepatic encephalopathy (66.7 %) was the cause in 4 out of 6 women who died , one women died of hepatorenal failure (16.7 %) and one due to acute cholangitis (16.7 %)

Various studies also report jaundice as one of the major indirect cause of maternal death, responsible for 5 to 30% of all maternal deaths [35,37-41] . Maternal deaths were directly proportional to the level of the serum bilirubin. Trivedi et al [41] also observed the same. According to this study the initial bilirubin level at admission > 10 is associated with poor maternal outcome and high maternal mortality.

CONCLUSION

CONCLUSION

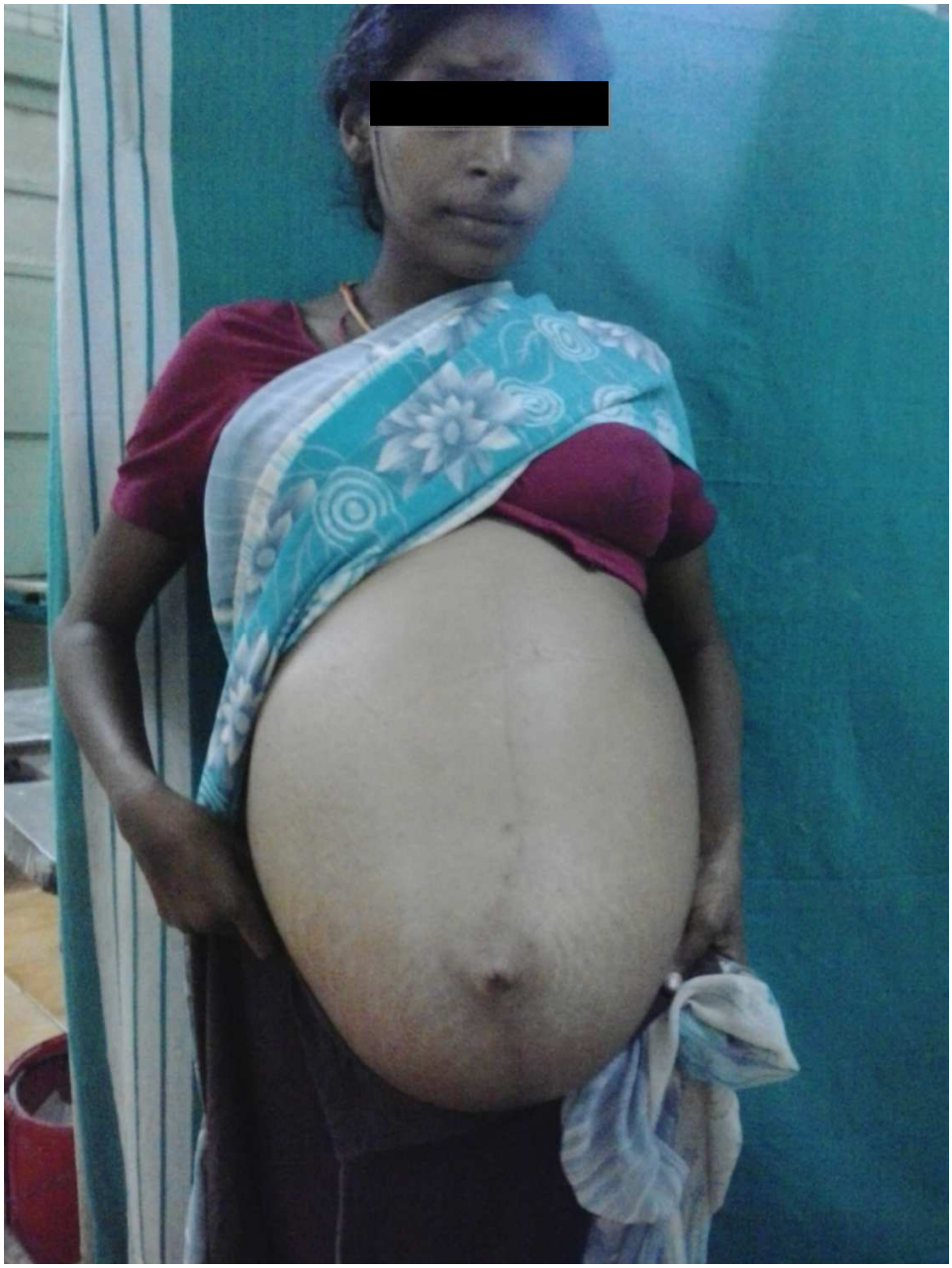
Jaundice in pregnancy is associated with high maternal and perintal mortality rates.

Viral hepatitis is the leading cause of jaundice according to our study with hepatitis E being the predominant virus. Hepatic encephalopathy and renal failure are the two important maternal complications. Hepatic encephalopathy is the common cause of death according to our study.

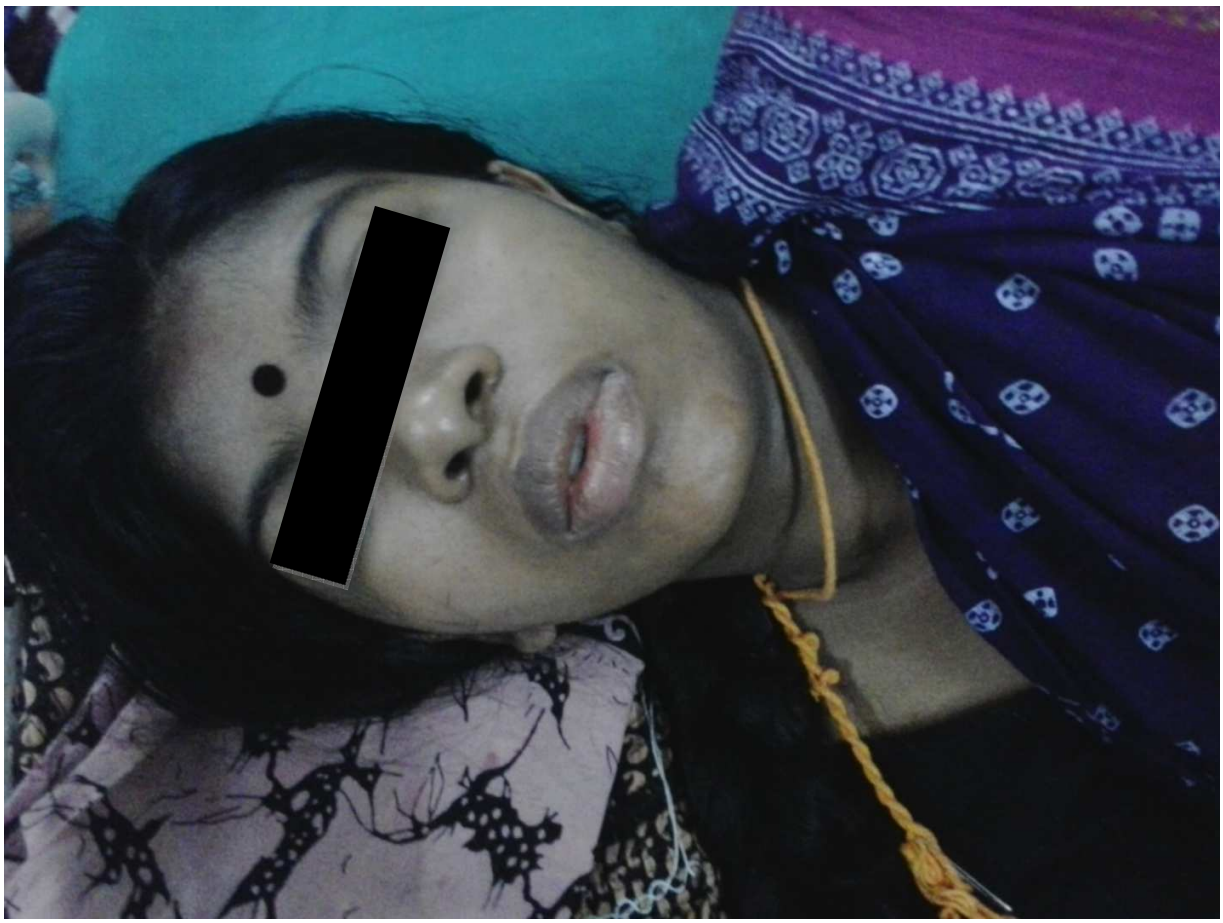
According to this study the initial bilirubin level at admission > 10 is associated with poor maternal outcome and high maternal mortality.

The factors responsible for a high maternal mortality in our country may be poor nutrition and hygiene, prevalence of anemia, delay in seeking medical advice, and delay in referral to the hospital. Many of the patients when brought to the tertiary health care system are already in moribund condition and often, do not respond to treatment.

CIRRHOSIS WITH PORTAL HYPERTENSENSION



Patient recovered from hepatic encephalopathy



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PROFORMA

MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY

NAME: AGE: IPNO:

D.O.A:

D.O.DELIVERY:

D.O.DISCHARGE:

PARITY:

L.M.P: E.D.D:

COMPLAINTS:

HOPI:

PAST H/O:

MENSTRUAL H/O:

MARITAL H/O:

OBSTETRICS H/O:

ON EXAMINATION: CONSCIOUS/ORIENTED - ANAEMIC

TEMPERATURE- PEDAL EDEMA- ICTERIC – MILD/SEVERE

PR- BP- CVS- RS-

P/A-

P/V-

INVESTIGATIONS:

HB% -

URINE ALBUMIN

SUGAR

DEPOSIT

BS/BP

BLOOD SUGAR

UREA

S.CREATININE

LFT T.BILIRUBIN

DIRECT

INDIRECT

SGOT

SGPT

ALP

LDH

PLATLET COUNT

CLOTTING TIME

BLEEDING TIME

PROTHROMBIN TIME

ACTIVATED PARTIAL THROMBOPLASTIN TIME

VIRAL MARKERS:

ULTRASOUND:

MANAGEMENT:

LIVER SUPPORTIVES

BLOOD AND BLOOD PRODUCTS TRANSFUSION:

ICU CARE:

MATERNAL OUTCOME:

MODE OF DELIVERY:

PERINATAL OUTCOME:

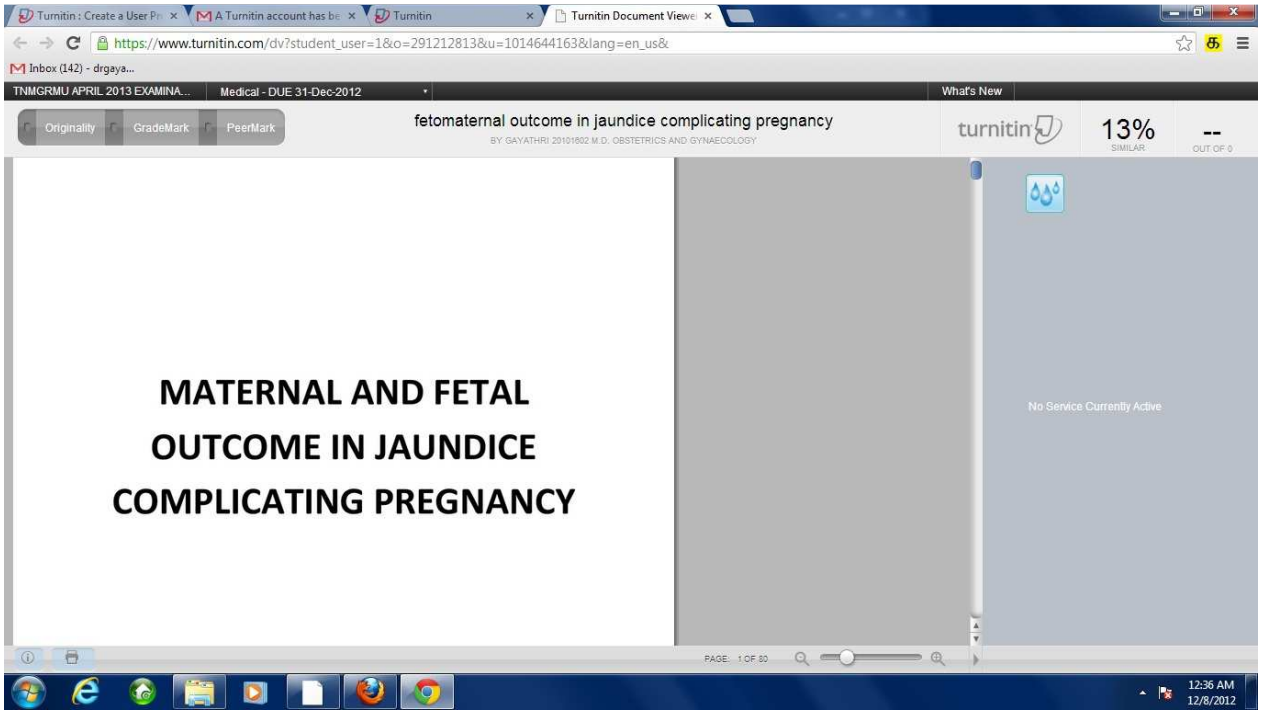
SEX: TERM/PRETERM: ALIVE/DEAD BORN/STILL BIRTH

APGAR 1MIN 5 MIN CONGENITAL ANOMALIES:

MASTER CHART

S.NO	NAME	AGE	PARITY	TRIMESTER	INITIAL BILIRUBIN	ETIOLOGY	MATERNAL OUTCOME				PERINATAL OUTCOME			
							TRANSFUSIONS	MODE OF DELIVERY	ICU CARE	COMPLICATIONS	TERM/ PRETERM	APGAR	SEX	B.WT IN KG
1.	MAHESWARI	21	PRIMI	III	1	VIRAL (HEV)	2 FFP	LN WITH EPI	-	-	PRETERM	2/10 5/10	FCH	2.2
2.	INDUJA	25	PRIMI	III	1	VIRAL (HBV)	-	LN WITH EPI	-	-	PRETERM	5/10 7/10	FCH	2.3
3.	SHANTHI	28	G4P2L2 A2	III	1	VIRAL (HBV)	-	LN WITH LP 1	-	-	TERM	5/10 6/10	FCH	2.7
4.	PONNUTHAI	21	PRIMI	III	5.4	VIRAL (HEV)	5 FFP	OUTLET FORCEPS WITH EPI	-	ATONIC PPH TENDERNESS ON BOTH LL	TERM SEVERLY ASPHYXIATES	0/10(BABY COULD NOT BE REVIVED)	MCH	3.5
5.	KARTHIGA	32	G2P1L1	III	9.5	VIRAL (HBV)	-	REPEAT LSCS	-	-	TERM	5/10 6/10	FCH	2.9
6.	MUTHUMARI	23	PRIMI	III	8	VIRAL (HEV)	2 FFP , I WB	UNDELIVERED	-	EXPIRED (HEPATIC ENCEPHALOPATHY)	-	-	-	-
7.	SYED ALI	24	G2P1L1	II	5.1	CHOLESTATIC	2 PC	UNDELIVERED	-	-	-	-	-	-
8.	ASHINA BANU	29	G3P2L2	III	3	VIRAL (HEV)	1 FFP	LNWITH EPI	-	-	TERM	6/10 8/10	MCH	3.3
9.	SUMATHI	24	G2P1L0	III	15	VIRAL (HAV)	I PC	LN WITH EPI	-	-	TERM	6/10 7/10	MCH	3
10.	PETCHIYAMMAL	36	G2P1L0	II	3.5	CIRRHOSIS WITH PORTAL HYPERTENSENSION	5 PC 7 FFP 1 WB	UNDELIVERED	-	-	-	-	-	-
11.	ALAGULAKSGMI	24	PRIMI	III	10.1	AFLP	I WB 6 FFP	LSCS (PRIMI / CPD / FETAL DISTRESS)	-	-	TERM	2/10 5/10	MCH	2.8
12.	MUTHUMARI	20	G2P1L0	III	1	-	-	LN WITH EPI	-	-	TERM	5/10 7/10	MCH	2.1
13.	MAHALAKSHMI	18	PRIMI	III	5.5	VIRAL (HAV)	5 FFP	LN WITH EPI	-	-	TERM	IUD	FCH	1.6
14.	VIJAYALAKSHMI	27	G5P1L1 A5	II	12.5	VIRAL (HAV)	-	LN	-	K/C/O ARNOLD CHIARI MALF TYPE1/ SYRINGOMYELIA	TERM	4/10 5/10	MCH	2.3
15.	AMUTHESWARI	22	PRIMI	III	8	AFLP	-	LN WITH EPI	-	-	TERM	6/10 7/10	FCH	2.2
16.	MUTHULAKSHMI	24	PRIMI	III	3	-	-	UNDELIVERED	-	-	-	-	-	-
17.	ANGELMARY	25	G3P1L1 A1	III	0.9	-	-	UNDELIVERED	-	-	-	-	-	-
18.	SELVI	24	G2P1L1	III	8.9	AFLP	1 WB 1 PLATLET	LN WITH EPI	-	-	TERM		FCH	2.6
19.	DEEPA	22	G2P1L1 (PREV LSCS)	II	9.5	-	-	UNDELIVERED	-	-	-	-	-	-
20.	KARPAGAVALI	24	G2P1L1 (PREV LSCS)	II	9.1	-	-	UNDELIVERED	-	-	-	-	-	-
21.	RAJESWARI	18	PRIMI	III	16.6	-	2 FFP	LN WITH EPI	-	ELEVATED RENAL PARAMETERS	TERM		MCH	2.6
22.	PREMA	23	G2P1L1	III	7.8	-	-	LSCS	-	GR I ABRUPTION				2.3
23.	TAMIL ELAKYA	25	PRIMI	III	1	VIRAL (HEV)	-	LN WITH EPI	-	-	TERM		FCH	3
24.	NAGAJOTHI	19	PRIMI	II	0.8	VIRAL (HEV)	-	UNDELIVERED	-	-	-	-	-	-
25.	NAGALAKSHMI	23	G2P1L1	III	0.8	VIRAL (HEV)	2 FFP	LN WITH EPI	-	-	TERM		MCH	3.5
26.	PUSPAVALLI	28	G2P1L1	II	5	VIRAL (HEV)	-	UNDELIVERED	-	-	-	-	-	-
27.	LAKSHMI	22	PRIMI	II	7	AFLP	-	UNDELIVERED	-	-	-	-	-	-
28.	UMA MAHESWARI	26	PRIMI	II	2.5	VIRAL (HBV)		UNDELIVERED	-	-	-	-	-	-
29.	SARAMMAL	23	G2P1L1	II	2.5	-	-	UNDELIVERED	-	-	-	-	-	-
30.	VEERALAKSHMI	24	G2P1L1 (PREV LSCS)	III	3	-	-	VBAC	-	-	TERM	6/10 8/10	FCH	2.3
31.	MEERAJ BANU	22	PRIMI	III	3	VIRAL (HBV)	-	LN WITH EPI	-	-	PRETERM		FCH	2.1
32.	KALESWARI	25	G2P1L1	II	2.5	-	-	UNDELIVERED	-	-	-	-	-	-
33.	BHAVANI	25	G2P1L1	II	3	VIRAL (HEV)	-	UNDELIVERED	-	-	-	-	-	-
34.	JEGADAMMAL	22	PRIMI	II	1.9	-	-	UNDELIVERED	-	-	-	-	-	-
35.	MUTHULAKS	21	PRIMI	II	7	VIRAL	-	UNDELIVERED	-	-	-	-	-	-

	HMI					(HEV)								
36.	DHANALAKSHMI	23	PRIMI	III	4.5	HELLP	3 WB , 3 FFP ,1 PLATLET	LSCS	Y	EXPIRED ON 7TH POD- HEPATIC ENCEPHALOPATHY	TERM	8/10 9/10	FCH	2.6
37.	KAVITHA	28	PRIMI	III	6.5	-	2 PC , 2 FFP	LN /LP 1	-	?HAEMOLYTIC ANAEMIA, ELEVATED RENAL PARAMETER, MR GR I	PRETERM	5/10 6/10	FCH	1.8
38.	MAHALAKSHMI	25	G2P1L1 / PREV LSCS	III	12.5	-	3 PC	LSCS	IRCU	RUPTURE UTERUS	TERM	IUD	MCH	2.7
39.	KARTHIGA DEVI	18	PRIMI	III	3	HELLP	7 FFP 1 PC 4 HUMAN ALBUMIN	LSCS	MICU	GDM	TERM	5/10 7/10	FCH	2.9
40.	LAKSHMI PRIYA	22	G2P1L1	I	3	HG	-	UNDELIVERED	-	-	-	-	-	-
41.	PANDIMEENA	19	PRIMI	III	2	VIRAL (HBV)	3 PC	LN / EPI	-	-	TERM	4/10 7/10	MCH	2.25
42.	CHINNAPONNU	20	PRIMI	II	3.5	AFLP	1 FFP 1 PLATLET	UNDELIVERED	-	-	-	-	-	-
43.	MEENA	19	PRIMI	III	10.2	VIRAL (HEV)	1WB 3 FFP 1 PLATLET	LN/EPI	-	-	TERM	6/10 7/10	FCH	2.6
44.	SUMATHI	23	G2P1L1	III	1	-	1 WB	REPEAT LSCS	-	-	TERM	6/10 7/10	MCH	2.5
45.	NAGAJOTHI	27	G2P1L1 / IUD	III	19.1	HELLP	7 FFP 2 WB 4 PLATLETS	REPEAT LSCS	IMCU	? HEPATORENAL SYND – I HD DONE EXPIRED ON	PRETERM	IUD	MCH	1.8 KG
46.	THULASIMANI	23	G2P1L1	III	1.3	VIRAL (HBV)	-	LN/EPI	-	-	TERM	6/10 7/10	FCH	2.3
47.	CHELLAMMAL	18	PRIMI	III	3	-	-	LN/EPI	-	-	TERM	5/10 6/10	MCH	2.6
48.	MUTHUKANNU	25	G2P1L1 / TWINS	II	2.6	-	-	UNDELIVERED	-	-	-	-	-	-
49.	MAHALAKSHMI	20	PRIMI	II	2.5	HELLP	-	SPONT. EXP	-	-	ABORTION	3/10 4/10	FCH	900
50.	BANUPRIYA	21	PRIMI	III	5	HELLP	1 PC 2 FFP	LN	-	-	PRETERM	IUD	MCH	1.5
51.	SEETHALAKSHMI	25	G2P1L1	III	3.5	AFLP	2 WB, 1 PC, 1 FFP, 1 PLT	REPEAT LSCS	-	-	TERM	7/10 8/10	FCH	2.8
52.	PARAMESWARI	28	G3P2L2 TWINS	III	13.5	VIRAL (HEV)	6 PC , 1O FFP, 4 WB , 2 PLATLET	LN/EPI	IRCU	ATONIC PPH HYSTERECTOMY DONE EXPIRED – HEPATIC ENCEPHALOPATHY	PRETERM	1-5/10 7/10 2-1/10 3/10	BOTH MCH	I – 1.86 II – 2.2
53.	CHANDRA	36	PRIMI	III	15	GILBERTS	5 FFP 1 WB	LSCS	MICU	-	PRETERM	6/10 7/10	MCH	2
54.	NAGALAKSHMI	25	G3P1L1 A1	II	10.6	VIRAL (HEV)	-	UNDELIVERED	-	-	-	-	-	-
55.	SHANTHI	23	PRIMI	III	8	VIRAL (HBV)	-	LN/EPI	-	-	LATE PRETERM	6/10 7/10	MCH	2
56.	MANIMALA	25	G2P1L1 PREV LSCS	III	8	VIRAL (HEV)	-	UNDELIVERED	-	-	-	-	-	-
57.	SABARNISHA	20	PRIMI	III	9	VIRAL (HEV)	5 FFP 1 PLATLET 1 WB	LN/EPI	-	ATONIC PPH	TERM	6/10 7/10	MCH	2.75
58.	KARTHIGA	23	G2P1L1	III	13	HELLP	18 WB 6 FFP 4 PLATLET	LN	IMCU	ATONIC PPH/DIC/ARF EXPIRED DUE TO HEPATIC ENCE	PRETERM	2/10 5/10	MCH	1.9
59.	KALAIVANI	24	G2P1L1	III	1.6	AFLP	2 FFP	LN/EPI	-	-	TERM	RESUS FAILURE	FCH	3.2
60.	THILAGAVATHY	20	PRIMI	III	9.5	VIRAL (HEV)	1WB 6 FFP	LN/EPI	IMCU	HEPATIC ENCEPHALOPATHY	IUD	-	FCH	2
61.	AMUTHA	24	G2P1L1	III	2	HAEMOLYTIC	2 FFP	REPEAT LSCS	MICU	PIGMENTED NEPHROPATHY	TERM	5/10 7/10	MCH	2.7
62.	CHITHRADEVI	24	G3P2L2	III	9	VIRAL (HEV)	1 WB 2 FFP	LN	-	-	PRETERM	5/10 6/10	MCH	1.75
63.	MEENATCHI	19	PRIMI	III	12	AFLP	1 WB 2 FFP	LN/EPI	-	-	TERM	5/10 6/10	FCH	3
64.	AMUTHA	20	PRIMI	III	7.2	CHOLESTATIC	I FFP	UNDELIVERED	-	-	-	-	-	-
65.	KAMATCHI	31	G2P1L1	III	8	CHOLESTATIC	2 FFP	REPEAT LSCS	IRCU	EXPIRED (ACUTE CHO;ANGITIS)	PRETERM	3/10 4/10	MCH	1.5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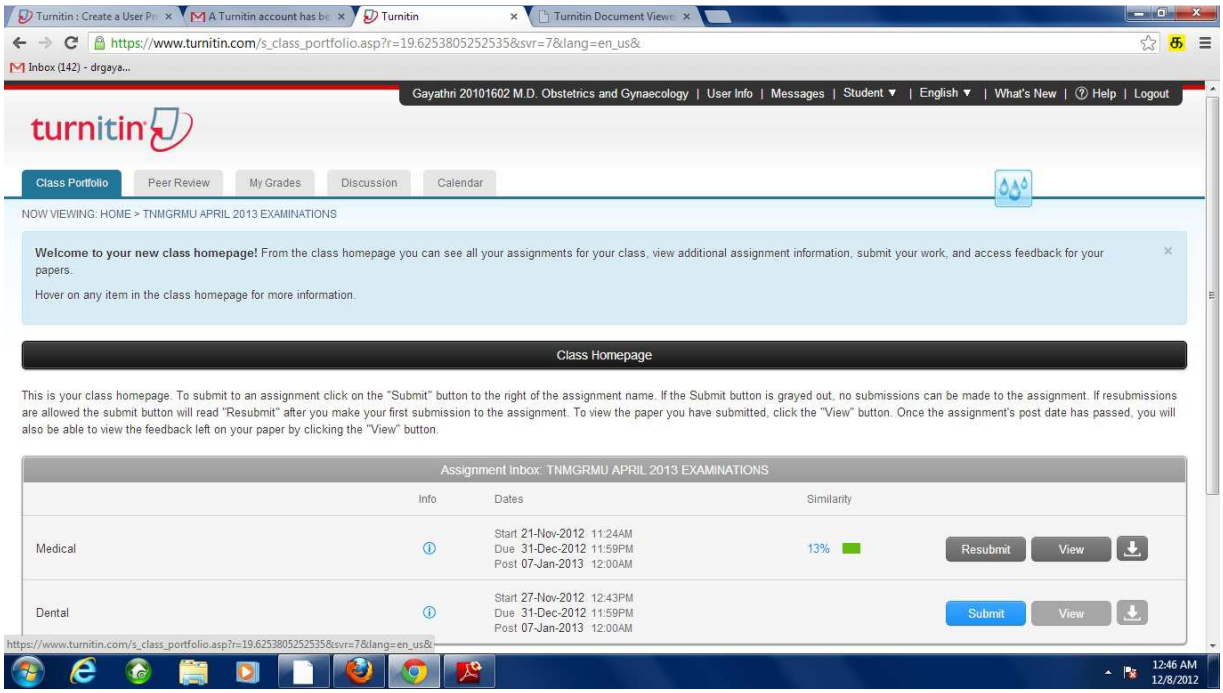
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MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY CONTENTS INTRODUCTION AIMS AND OBJECTIVES REVIEW OF LITERATURE MATERIALS AND METHODS OBSERVATION AND RESULTS DISCUSSION CONCLUSION BIBLIOGRAPHY INTRODUCTION The incidence of jaundice in India varies from 0.4 to 0.9/1000 deliveries. Jaundice in pregnancy carries a grave prognosis for both the mother and the fetus , and is responsible for 10% of maternal deaths. Liver disease in pregnancy is an important medical disorder seen more often in developing countries than in developed ones. The present study analyzes the causes and the fetomaternal outcome in pregnancies affected with jaundice. Abnormal liver test results are obtained in...

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Govt Rajaji Hospital, Madurai 625020.
Convenor
grhethicssecy @gmail.com.

Sub: Establishment-Govt. Rajaji Hospital, aMadurai-20-
Ethics committee-Meeting Agenda-communicated-regarding.

The Ethics Committee meeting of the Govt. Rajaji Hospital, Madurai was held at 11.00 Am to 1.00Pm on 29.03.2012 at the Dean Chamber, Govt. Rajaji Hospital, Madurai. The following members of the committee have been attended the meeting.

1. Dr.N.Vijayasankaran,M.ch(Uro.) 094-430-58793 0452-2584397	Sr.Consultant Urologist Madurai Kidney Centre, Sivagangai Road,Madurai	Chairman
2. Dr.P.K. Muthu Kumarasamy, M.D., 9843050911	Professor & H.O.D of Medical, Oncology(Retired)	Member Secretary
3. Dr.T.Meena,MD 094-437-74875	Professor of Physiology, Madurai Medical College	Member
4. Dr. S. Thamilarasi, M.D (Pharmacol)	Professor of pharmacology	
5. Dr.Moses K.Daniel MD(Gen.Medicine) 098-421-56066	Professor of Medicine Madurai Medical College	Member
6. Dr.M.Gobinath,MS(Gen.Surgery)	Professor of Surgery Madurai Medical College	Member
7. Dr.S. Dilshadh, MD(O&G) 9894053516	Professor of OP&Gyn Madurai Medical College	Member
8. Dr.S.Vadivel Murugan., M.D, 097-871-50040	Professor of Medicine Madurai Medical College	Member
9. Shri.M.Sridher,B.sc.B.L. 099-949-07400	Advocate, 2, Deputy collectors colony 4th street KK Nagar, Madurai-20.	Member
10. Shri.O.B.D.Bharat,B.sc., 094-437-14162	Businessman Plot No.588, K.K.Nagar,Madurai.20.	Member
11.Shri. S.sivakumar,M.A(Social) Mphil 093-444-84990	Sociologist, Plot No.51 F.F, K.K Nagar, Madurai.	Member

Following Projects were approved by the committee

Sl. No	Name of P.G.	Course	Name of the Project	Remarks
1.	Gayathri. S	PG, M.D (ob gyn)	Jaundice complicating pregnancy and fetal-maternal outcome	Approved

Please note that the investigator should adhere the following: She/He should get a detailed informed consent from the patients/participants and maintain Confidentially.

1. She/He should carry out the work without detrimental to regular activities as well as without extra expenditure to the institution to Government.
2. She/He should inform the institution Ethical Committee in case of any change of study procedure site and investigation or guide.
3. She/He should not deviate for the area of the work for which applied for Ethical clearance. She/He should inform the IEC immediately, in case of any adverse events pr Serious adverse reactions.
4. She/he should abide to the rules and regulations of the institution.
5. She/He should complete the work within the specific period and apply for if any Extension of time is required She should apply for permission again and do the work.
6. She/He should submit the summary of the work to the Ethical Committee on Completion of the work.
7. She/He should not claim any funds from the institution while doing the word or on completion.
8. She/He should understand that the members of IEC have the right to monitor the work with prior intimation.

DEAN

To
All the above members and Head of the Departments concerned.
All the Applicants.