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A STUDY ON FETOMATERNAL OUTCOMES OF JAUNDICE IN PREGNANCY WOMEN

GYNAECOLOGY

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ABSTRACT

The incidence of jaundice in pregnancy varies throughout the world.
Objective of my study is Prospective Study of Jaundice in Pregnancy Admitted in a Tertiary Care Centre with Special Reference to the Fetomaternal Outcome and thus to evaluate outcome in order to reduce maternal and fetal morbidity and mortality by early diagnosis , regular follow up and prompt intervention.
100 antenatal patients with jaundice and serum bilirubin more than 3mg/dl. The proposed study will be conducted in the department of obstetrics &gynaecology in COMJNMH HOSPITAL, KALYANI, West Bengal from March 2019 to February 2020 (One year)
Acute viral hepatitis is the most common cause of jaundice in pregnancy with HEV being the predominant cause with highest maternal and fetal complication rate and even mortalities. We found in our 15(15%) patients had PPH. 3(3%) patients had DIC. 2(2%) patients had Heaptorenal Syndrome. 6(6%) patients had Hepatic encephalopathy. 11(11%) patients had Maternal Mortality. 30(30%) patients had MSL. 7(7%) patients had instrumental delivery, 28(28%) patients had LSCS, 65(65%) patients had vaginal delivery. 66 (66%) patients had no Viral Hepatitis, 34(34%) patients had Viral Hepatitis.
It was concluded that availability of antenatal care for early detection and well-equipped hospitals for intensive care will help in the reduction of viral hepatitis in pregnancy and also its associated maternal and perinatal mortality and morbidity.

KEYWORDS

Jaundice, Pregnancy, Fetomaternal Outcomes

INTRODUCTION

Jaundice is defined as yellowish discoloration of skin and sclera due to increase in serum bilirubin levels. Pregnancy is characterized by number of physiological changes in various organs including liver. High levels of serum estrogen and progesterone affect the metabolic, synthetic, and excretory function so of the liver duringpregnancy. Liver disorders complicate 3-5% of pregnancies and constitute an important cause of neonatal and maternal morbidity and mortality.[1]It accounts for 60% perinatal and 14% of maternal mortality.[2]

The incidence of jaundice in pregnancy varies throughout the world. It is around 0.1% in developed countries and ranges from 3-20% or higher in developing countries. Incidence of jaundice in pregnancy is 0.4-0.9/1000 in India.[2] Viral hepatitis is the most common cause of jaundice in pregnancy followed by cholestasis. The most common viruses responsible for viral hepatitis are hepatitis A (HAV), hepatitis B (HBV), hepatitis C (HCV), hepatitis E virus (HEV). Jaundice is the most common symptom of acute hepatitis. In developing countries like India, hepatitis E is the commonest cause of fulminant hepatic failure in pregnancy, mostly occurring in the third trimester of pregnancy leading to high maternal mortality ranging from 15-45%.[3]

Jaundice in pregnancy whilst relatively rare, has potentially serious consequences for maternal and fetal health.[4] Incidence of jaundice in pregnancy in developing country is much higher, due to poor nutrition and poor sanitation.

Jaundice in pregnancy can be caused by viral hepatitis, intrahepatic cholestasis of pregnancy, choledocholithiases, HELLP syndrome (hemolysis, elevated liver enzymes, and a low platelet count), severe preeclampsia, and acute fatty liver of pregnancy.[5] Course of hepatitis is unaltered by pregnancy the exception is hepatitis E, where the pregnant women who contract the disease exhibit fatality rates of 10-15%.[6] Jaundice in the pregnancy can be a grave prognosis for both mother and fetus, causing maternal mortality in 10%.[7,8] Most common cause of jaundice is viral hepatitis, hepatitis B is most commonly involved.

When red blood cells have completed their life span of approximately 120 days, or when they are damaged, their membranes become fragile and prone to rupture. As each red blood cell traverses through the reticuloendothelial system, its cell membrane ruptures when its membrane is fragile enough to allow this. Cellular contents, including hemoglobin, are subsequently released into the blood. The hemoglobin is phagocytosed by macrophages, and split into its heme and globin portions. The globin portion, a protein, is degraded into amino acids and plays no role in jaundice. Two reactions then take place with the heme molecule. The first oxidation reaction is catalyzed by the microsomal enzyme hemeoxygenase and results in biliverdin (green color pigment), iron and carbon monoxide. The next step is the reduction of biliverdin to a yellow colortetrapyrol pigment called bilirubin by cytosolic enzyme biliverdinreductase. This bilirubin is "unconjugated," "free" or "indirect" bilirubin. Approximately 4 mg of bilirubin per kg of blood is produced each day.[6]The majority of this bilirubin comes from the breakdown of heme from expired red blood cells in the process just described. However approximately 20 percent comes from other heme sources, including ineffective erythropoiesis, and the breakdown of other heme-containing proteins, such as muscle myoglobin and cytochromes.[6]

Objective of my study is Prospective Study of Jaundice in Pregnancy Admitted in a Tertiary Care Centre with Special Reference to the Fetomaternal Outcome and thus to evaluate outcome in order to reduce maternal and fetal morbidity and mortality by early diagnosis , regular follow up and prompt intervention.

MATERIALS AND METHODS

100 antenatal patients with jaundice and serum bilirubin more than 3mg/dl. The proposed study will be conducted in the department of obstetrics &gynaecology in COMJNMH HOSPITAL, KALYANI, West Bengal from March 2019 to February 2020(One year)

Exclusion Criteria

All antenatal patients with serum bilirubin <3mg/dl
All antenatal patients complicated by any other medical disorder.

Statistical Analysis:

For statistical analysis data were entered into a Microsoft excel spreadsheet and then analyzed by SPSS 20.0.1 and GraphPad Prism version 5. Data had been summarized as meanand standard deviation for numerical variables and count and percentages for categorical variables. p-value ≤ 0.05 was considered for statistically significant.

RESULTS AND ANALYSIS

Our study showed that the mean age (mean± s.d.) of patients was 25.1900 ± 3.7489 years with range 18.00-32.00 years and the median age was 26.00 years.58 (58.0%) patients were 1 st gravida, 25(25%) patients were 2 nd gravida, 10(10%) patients were 3 rd gravida, 7(7%) patients were 4 th gravida.75(75%) patients were no living issue, 14(14%) patients were 1 living issue, 7(7%) patients were 2 living issue, 4(4%) patients were 3 living issue.58(58%) patients had no parity, 25(25%) patients had 1 parity, 10(10%) patients had 2 parity,7(7%) patients had 3 parity.

We found in our study the mean height (mean± s.d.) of patients was 156.3900±156.3900 cm with range 140.0000168.0000 cm and the median age was 158.0000 cm.The mean weight (mean± s.d.) of patients was 59.9300 ± 15.2966 kg with range 46.0000-155.0000 kg and the median age was 56.0000 kg.The mean BMI (mean± s.d.) of patients was 59.9300 ± 15.2966 kg with range 46.0000-155.0000 kg and the median age was 56.0000 kg.The mean SBP (mean± s.d.) of patients was 117.7200 ± 13.8827 mmHg with range 100.0000-170.0000 mmHg and the median age was 116.0000 mmHg.The mean DBP (mean± s.d.) of patients was 73.6000 ± 8.8169 mmHg with range 60.0000 -100.0000 mmHg and the median age was 72.0000mmHg.

We showed that the mean Hemoglobin (mean± s.d.) of patients was 10.9130 ± 1.3461(g/dL) with range 7.8000- 13.1000 (g/dL) and the median age was 11.2000(g/dL).24(24%) patients had edema, 76(76%) patients had no edema. 98(98%) patients had no H/O of nausea vomiting, 2(2%) patients had H/O of nausea vomiting.88(88%) patients had no H/O of cholestasis, 12(12%) patients had H/O cholestasis.30(30%) patients had no H/O of Booked case, 70(%) patients had Un booked case.24(24%) patients had no H/loss of Appetite, 76(76%) patients had H/loss of Appetite.

Our study showed that the 99(99%) patients had no H/O convulsion, 1(1%) patient had H/O convulsion.100(100%) patients had no H/o diabetes.91(91%) patients had no H/O essential hypertension, 9(9%) patients had H/O essential hypertension.100(100%) patients had no H/O gall bladder stone.89(89%) patients had no H/o hyperemesis, 11(11%) patients had H/o hyperemesis.94(94%) patients had no H/O hypertension, 6(6%) patients had H/O hypertension.96(96%) patients had no Past H/O jaundice, 4(4%) patients had Past H/O jaundice.

We found in our study 42(42%) patients had no H/O pruritus, 58(58%) patients had Past H/O pruritus.The mean Total bilirubin (mean± s.d.) of patients was 7.7694 ± 3.0000 mg/dL with range3.0000-22.1000 mg/dL and the median age was 5.2000 mg/dL.46(46%) patients had 1 T bilirubin, 19(19%) patients had 2 T bilirubin, 21(21%) patients had 3 T bilirubin, 14(14%) patients had 4 T bilirubin.The mean SGOT (mean± s.d.) of patients was 175.9900 ± 355.2067 IU/LT with range 28.00-180.00 IU/LT and the median age was 50.00 IU/LT.The mean SGPT (mean± s.d.) of patients was 182.1400 ± 360.5947 IU/LT with range 30.00-1600.00 IU/LT and the median age was 46.00 IU/LT.

Our study showed that the mean ALP (mean± s.d.) of patients was 285.2800 ± 42.4269 IU/L with range 140.00- 380.00 IU/L and the median age was 280.00 IU/L.The mean Albumin (mean± s.d.) of patients was 3.8140 ± .6207 g/dL with range 2.5000 - 5.0000 g/dLand the median age was 4.0000 g/dL.The mean TP (mean± s.d.) of patients was 5.6470 ± .4815 g/dL with range 4.8000 - 6.2000 g/dL and the median age was 5.8000 g/dL.The mean LDH (mean± s.d.) of patients was 183.42 ± 130.00 U/L with range 130.00 - 280.00 U/L and the median age was 180.00 U/L.100(100%) patients had no ANTI ACV.

We showed that 79(79%) patients had no HBEAG, 21(21%) patients had HBEAG. 79(79%) patients had no ANTI HEV, 21(21%) patients had ANTI HEV 84 (84%) patients had no HBSAG, 16(16%) patients had HBSAG. The mean PT sec (mean± s.d.) of patients was 14.4700 ± 3.6858 sec with range 12.00 - 28.00 sec and the median age was 14.00 sec. The mean APPT sec (mean± s.d.) of patients was 32.4700 ± 5.4057sec with range 28.00 - 52.00 sec and the median age was 30.50 sec. The mean INR (mean± s.d.) of patients was 1.2320 ± .4836 sec with range 1.00 - 2.80 sec and the median age was 1.10 sec. The mean CBG (mean± s.d.) of patients was 75.9100 ± 15.7455(mg/dl) with range 40.00 - 102.00 (mg/dl) and the median age was 78.01(mg/dl).

We found in our study 85 (85%) patients had no PPH, 15(15%) patients had PPH. 97 (97%) patients had no DIC, 3(3%) patients had DIC. 98 (98%) patients had no Heaptorenal syndrome, 2(2%) patients had Heaptorenal Syndrome. 94 (94%) patients had no Hepatic encephalopathy, 6(6%) patients had Hepatic encephalopathy. 34 (34%) patients had no Jaundice resolve after delivery, 66(66%) patients had Jaundice resolve after delivery. 89 (89%) patients had no Maternal Mortality, 11(11%) patients had Maternal Mortality. 70 (70%) patients had no MSL, 30(30%) patients had MSL. 7(7%) patients had instrumental delivery, 28(28%) patients had LSCS, 65(65%) patients had vaginal delivery. 66 (66%) patients had no Viral Hepatitis, 34(34%) patients had Viral Hepatitis.

DISCUSSION

Jaundice in pregnancy is a high risk pregnancy and requires a tertiary centre care for management of the same and prompt management of maternal complications.[9]

Obstetric cholestasis is the most common liver disorder in pregnancy. [10] We found that 12(12%) patients had H/O cholestasis.

Our study found that most common cause of Jaundice in pregnancy is viral Hepatitis and obstetric cholestasis is the second most common liver disorder in pregnancy.

The second most common cause of jaundice in pregnancy from our study was cholestatic jaundice which accounted for 23.6% of the cases. Pruritus is the most common complaint in these patients. Pruritus with elevated serum bile acids seen in second half of pregnancy resolving soon after delivery is considered as intrahepatic cholestasis. The recurrence of this complication is frequently seen in subsequent pregnancies. Since maternal morbidity associated with it is low, the effects are seen on the fetus. One study reported 5 out of 48 cases of intrahepatic cholestasis in pregnancy. All the cases of intrahepatic cholestasis of pregnancy were observed in the third trimester of pregnancy similar to the study by one study.[9] OC is associated with increased risk of prematurity, fetal distress, and stillbirth10 depending upon disease severity.[10]

The most common cause of jaundice was viral hepatitis accounting for 62% of the cases.39 The similar finding was noted by one study found that the incidence of viral hepatitis causing jaundice was 57% while Ching et al reported half of patient out of 48 were of viral hepatitis in pregnancy.6-10 We found that 32% jaundice was viral hepatitis.

Hepatitis E is the most prevalent and most dangerous type of viral hepatitis, contributing to 71 % of the cases of viral hepatitis in our case series. The incidence reported by a study done by [9]. We found 21% of the cases of viral hepatitis.

ICMR is as high as 80-90% in cases of viral hepatitis in pregnancies. Totally, hepatitis E contributed to 44 % cases of jaundice. Hepatitis E is a self-limiting enterically transmitted acute viral hepatitis. In non-pregnant women the disease has a case fatality rate of <0.1%. [9]

The course of events in other viral causes of hepatitis and that of hepatitis E differs in the fact that hepatitis E has a more fulminant course in pregnancy. Pregnancy appears to be a potential risk factor for viral replication and extreme low immune status of Indian or rather Asian pregnant women. The reason for this phenomenon is unclear, yet certain reasons have been put forward to prove the same. There is a shift in the T Helper cell balance in pregnancy more towards a TH2 response leading to reduced CD4 and increase in CD8 count thus causing a moderate amount of immunosuppression. This is superadded to the immunosuppressed state of pregnancy. Also, estrogen and progesterone secreted in high amounts during pregnancy impair the cell mediated immunity by triggering the adapter protein of HEV virus (ORF3) which also facilitates replication of the virus and further catastrophe.[9]

Other studies with larger sample size like Shukla et.al (2011), Jethwa et al (2015), Patra et al and others shows comparative results though acutely infected patients with HBV and HCV has been seen in their

studies, current study does not. Prevalence rate of current study {HEV (84%) and HAV (16%)} and Patra et al study (HEV 60%) and Jaiswal et al 57.5% are comparable. Majority of patients with mean age of 23.3±3.9 years were primigravida (58%) in their third trimester (75%). Our findings are similar to the observations seen in other studies by Jaiswal et al(72%), Singh S et al (72%) and Patra et al (72%) who reported more than 70% of pregnant women with acute viral hepatitis presented in their third trimester and were primigravida.[11-16]

There were totally 78 maternal deaths in that institution during the study period, 12 maternal deaths due to jaundice (15.3%). Case fatality rate of jaundice in pregnant woman is 21.8%.[17]

In our study we had three patients of HELLP. Ching et al reported 2 cases out of 48 of HELLP syndrome in pregnancy. Complications of HELLP include DIC, abruption placenta, acute renal failure, pulmonary edema and rarely hepatic rupture.[17]

We found 3 patients of HELLP syndrome and agree with that study.

CONCLUSION

Acute viral hepatitis is the most common cause of jaundice in pregnancy with HEV being the predominant cause with highest maternal and fetal complication rate and even mortalities. Till then increased availability of antenatal care for early detection and well equipped hospitals for intensive care will help in the reduction of viral hepatitis in pregnancy and also its associated maternal and perinatal mortality and morbidity.

In a country like India, beginning from health education to the pregnant mother regarding warning signs and immediate visit to the doctor, to medical personnel at primary health centre for early transfer can go long way in lowering maternal and perinatal mortality and morbidity due to jaundice in pregnancy.

Table1 : Distribution Of Mean In Clonicopathological Parameters

	Number	Mean	SD	Minimum	Maximum	Median
Age	100	25.1900	3.7489	18.0000	32.0000	26.0000
Height(cm)	100	156.3900	156.3900	140.0000	168.0000	158.0000
Weight (kg)	100	59.9300	15.2966	46.0000	155.0000	56.0000
BMI	98	24.3420	3.0740	20.0000	34.6000	23.5000
SBP	100	117.7200	13.8827	100.0000	170.0000	116.0000
DBP	100	73.6000	8.8169	60.0000	100.0000	72.0000
Haemoglobin(g/DL)	100	10.9130	1.3461	7.8000	13.1000	11.2000
TBilirubin	100	7.7694	5.0179	3.0000	22.1000	5.2000
SGOT	100	175.9900	355.2067	28.0000	1800.0000	50.0000
SGPT	100	182.1400	360.5947	30.0000	1600.0000	46.0000
ALP	100	285.2800	42.4269	140.0000	380.0000	280.0000
AlB	100	3.8140	.6207	2.5000	5.0000	4.0000
TP	100	5.6470	.4815	4.8000	6.2000	5.8000
LDH	100	183.4200	30.9226	130.0000	280.0000	180.0000
PTsec	100	14.4700	3.6858	12.0000	28.0000	14.0000
APTTsec	100	32.4700	5.4057	28.0000	52.0000	30.5000
INR	100	1.2320	.4836	1.0000	2.8000	1.1000
CBGmg/dl	100	75.9100	15.7455	40.0000	102.0000	78.0000

Table2:Distribution Of All Parameters

		Frequency	Percent
Parity	**0**	58	58.0%
	1	25	25.0%
	2	10	10.0%
	3	7	7.0%
Edema	**Positive**	24	24.0%
	Nil	76	76.0%
H/O Nauseavomiting	**NO**	98	98.0%
	YES	2	2.0%
H/o cholestasis	**NO**	95	95.0%
	YES	5	5.0%
Booked/Unbooked	**Booked Case**	30	30.0%
	Unbooked Case	70	70.0%
H/loss of Appetite	**NO**	24	24.0%
	YES	76	76.0%
H/O convulsion	**NO**	99	99.0%
	YES	1	1.0%
H/odiabetes	**NO**	100	100.0%
H/Oessential hypertension	**NO**	91	91.0%
	YES	9	9.0%
H/Ogall bladder stone	**NO**	100	100.0%
H/o hyperemesis	**NO**	89	89.0%
	YES	11	11.0%
H/O hypertension	**NO**	94	94.0%
	YES	6	6.0%
H/O jaundice	**NO**	96	96.0%
	YES	4	4.0%
H/Opruritus	**NO**	42	42.0%
	YES	58	58.0%
T bilirubin1	**1**	46	46.0%
	2	19	19.0%
	3	21	21.0%
	4	14	14.0%
ANTIACV	**NO**	100	100.0%
HBEAG	**NO**	90	90.0%
	YES	10	10.0%
ANTIAEV	**NO**	96	96.0%
	YES	4	4.0%
ANTIHAV	**NO**	95	95.0%
	YES	5	5.0%
HBEAG	**NO**	100	100.0%
HBSAG	**NO**	91	91.0%
	YES	9	9.0%
PPH	**NO**	85	85.0%
	YES	15	15.0%
DIC	**NO**	97	97.0%
	YES	3	3.0%
Heaptorenal syndrome	**NO**	98	98.0%
	YES	2	2.0%
Hepaticencephalo pathy	**NO**	94	94.0%
	YES	6	6.0%
Jaundice resolve after delivery	**NO**	34	34.0%
	YES	66	66.0%
Maternal Mortality	**NO**	89	89.0%
	YES	11	11.0%
MSL	**NO**	70	70.0%
	YES	30	30.0%
MOD	**Instrumental**	7	7.0%
	LSCS	28	28.0%
	VD	65	65.0%

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