# "To Study the Effect of Jaundice in Pregnancy and Pregnancy Outcome"

**PROF. Uma Pandey[1]**

**Dr. Devesh Prakash Yadav[2]**

**Dr. Nikhila saite[3]**

1. Professor, Former HOD, Department of Obstetrics and Gynaecology, Institute of Medical Science, Banaras Hindu University, Varanasi

2. Associate Professor and Head of Development of Gastroenterology, Institute of Medical Science, Banaras Hindu University, Varanasi

3. Junior Resident, Department of Obstetrics and Gynaecology, Institute of Medical Science, Banaras Hindu University, Varanasi

**Introduction:** Jaundice during pregnancy, characterized by the yellow discoloration of the skin, sclera, and mucous membranes, results from elevated serum bilirubin levels exceeding 2 mg/dL. Jaundice in pregnancy may arise from conditions unique to pregnancy or from pre-existing liver diseases. Globally, the prevalence and causes of jaundice in pregnancy vary. It is more frequently observed in low- and middle-income countries (LMICs), where infections, limited access to healthcare, and delayed diagnosis heighten its burden. Conversely, in high-income settings, cases are typically linked to autoimmune, metabolic, or genetic disorders. Timely diagnosis and targeted management are essential for improving outcomes (1).

Hormonal changes, particularly elevated estrogen and progesterone, can impair bile secretion and predispose pregnant individuals to intrahepatic cholestasis. Cholestasis results in the accumulation of conjugated bilirubin due to disrupted bile flow. In contrast, hemolytic processes—such as those observed in HELLP syndrome, malaria, or inherited conditions like sickle cell anemia—lead to

increased levels of unconjugated bilirubin due to accelerated breakdown of red blood cells. Hepatocellular damage caused by viral hepatitis or metabolic liver diseases impairs both bilirubin conjugation and excretion, while biliary obstruction from gallstones or strictures can further compromise bile flow and raise direct bilirubin levels (2).

Pregnancy-specific hepatic disorders include intrahepatic cholestasis of pregnancy (ICP), acute fatty liver of pregnancy (AFLP), and HELLP syndrome. ICP, typically presenting in the third trimester, is driven by hormonal disruption of bile transport and manifests as pruritus and mild jaundice. Though maternal outcomes are generally favorable, ICP increases risks of preterm labor, fetal distress, and stillbirth. HELLP syndrome, a severe form of preeclampsia, is defined by hemolysis, elevated liver enzymes, and low platelet counts. It is often accompanied by jaundice and can lead to life-threatening complications if not managed in a timely manner. AFLP, a rare but life-threatening condition, is characterized by microvesicular fat accumulation in hepatocytes, leading to hepatic failure, coagulopathy, and multisystem involvement.(3)

Management of jaundice in pregnancy depends on identifying the underlying cause and implementing timely, individualized treatment strategies.

## Aims and Objective:

- This study aims to assess the prevalence, etiology, maternal morbidity and mortality and fetal outcomes in pregnancies complicated by jaundice.
- It also evaluates the effect of etiological treatment on clinical and biochemical parameters.

## Materials and Methods:

- This prospective observational study was conducted over two years (January 2023 to December 2024) at the Institute of Medical Sciences, BHU. Eighty pregnant women with jaundice (cases) and eighty without jaundice (controls) were enrolled.
- Relevant history, clinical examination, hematological / biochemical and fetal outcome parameters were assessed in cases and controls. Statistical analyses were performed using t-tests and Chi-square tests.

### Study Participants:

- **Inclusion Criteria:**
  - Pregnant women with increased bilirubin levels.
  - Pregnant patients already diagnosed with jaundice and undergoing treatment.
- **Exclusion Criteria:**
  - Non-pregnant individuals diagnosed with jaundice.

## RESULTS :

Our findings show that the **mean pulse rate** was significantly higher in cases (100.65 bpm) than in controls (95.23 bpm), indicating a statistically significant difference. This elevated pulse rate may suggest hemodynamic stress or a systemic compensatory response in affected pregnancies.

The t-test results ($t = 4.1072$, $p = 0.0001$) indicate a statistically significant difference in pulse rates between cases and controls.

The comparison of **systolic and diastolic blood pressure** between cases and controls shows significantly higher values in the cases group.The t-tests for both systolic ($t = 10.93$, $p < 0.0001$) and diastolic ($t = 10.06$, $p < 0.0001$) BP confirm highly significant differences, suggesting a strong association between elevated BP and the condition under study.

To compare **Haemoglobin** levels Case and Control from two groups ($n_1 = 80$ and $n_2 = 80$). The two-tailed p-value for $t = -1.6143$ is approximately 0.1085. Since p-value (0.1085) > 0.05, the null hypothesis cannot be rejected. This means there is no statistically significant difference in haemoglobin levels between Case and Control groups.(Table: 1)

To compare the **White Blood Cell (WBC)** distributions between the Case and Control groups, we can use an **independent two-sample t-test**. Since the p-value (0.0679) is slightly above the common significance level (0.05), we fail to reject the null hypothesis at the 5% significance level. This suggests that there is **no strong statistical evidence** to conclude a significant difference in mean WBC counts between the Case and Control groups.(Table: 1)

The mean **platelet count** in the case group was 135,692.5 , whereas in the control group, the mean was significantly higher at 169,677.5. The independent t-test yielded a t-statistic of -1.96768 and

a p-value of 0.05, indicating a borderline statistically significant difference in platelet levels between the two groups.(Table: 1)

In our study it was seen that there were about 40% cases having jaundice had thrombocytopenia with platelet counts below 1,00,000.

In our study **creatinine** level were slightly elevated in cases (0.98) compared to controls(0.907), though difference was not statistically significant. To compare the **Urea** distributions between the Case and Control groups, we can use an **independent two-sample t-test**. Since p-value (0.0000) is less than the common significance level (0.05), we reject the null hypothesis at the 5% significance level.

To compare **SGPT and SGOT** in Case and Control from two groups ($n_1 = 80$ and $n_2 = 80$), the appropriate statistical method is the Independent Sample t-test. Since the p-value (0.0007) and (0.0013) respectively , the null hypothesis rejected. This means there is statistically significant difference in SGPT and SGOT between Case and Control groups.

our findings show significantly elevated liver enzymes indicating hepatocellular injury and conforming hepatic dysfunction in jaundiced pregnancy.(Table: 2)

To compare **Total Bilirubin (TB) and Direct Bilirubin (DB)** between Case and Control, the appropriate statistical method is the Independent Sample t-test. For TB, The two-tailed p-value for t=15.6150 is approximately 0.0000 and for DB The two-tailed p-value for t = 8.5173 is approximately 0.0013. Since p-value (0.0000) < 0.05, the null hypothesis rejected. This means there is statistically significant difference in Total Bilirubin (TB) and Direct Bilirubin (DB) between Case and Control groups.(Table: 2)

To compare the **Alkaline Phosphatase (ALP)** distributions between the Case and Control groups, we can use an **independent two-sample t-test**. Since the t-test is 2.119 and p-value (0.0356) is <0.05, we reject the null hypothesis at the 5% significance level. This suggests that there is **strong**

**statistical evidence** to conclude a significant difference in mean Alkaline Phosphatase (ALP) counts between the Case and Control groups.

To compare the **Serum Uric Acid and LDH** distributions between the Case and Control groups, we can use an **independent two-sample t-test**. Since the p-value (0.0019) and 0.0034 respectively, which is <0.05, the null hypothesis is rejected. This suggests that there is **strong statistical evidence** to conclude a significant difference in mean Uric Acid and LDH counts between the Case and Control groups.

compare the **International Normalized Ratio (INR)** distributions between the Case and Control groups, we can use an **independent two-sample t-test**. Since the t-test is 1.339 and p-value (0.1823) is greater than the common significance level (0.05), we fail to reject the null hypothesis at the 5% significance level. This suggests that there is no **strong statistical evidence** to conclude a significant difference in mean **International Normalized Ratio (INR)** counts between the Case and Control group

Although, in our study it was observed that more that 30% cases had increased INR levels, which signifies that jaundice leads to deranged coagulation profile in cases than normal INR levels in controls.

Out of 80 cases only 72% patients was vitality stable and got discharged, a large number (~20%) patients required Gastroenterology referral.

## Final outcome of mother (Out of 80 cases and controls)

Our study shows a significant number of case (~15%) required Gastroenterology referral and transfer. Although, many cases (~70%) respond to etiological treatment and discharged with stable vitals, around ~5% patient's conditions deteriorated even after intensive care and treatment then compared to controls.(table :3)

**NICU Admissions:**

The table shows a significantly higher rate of NICU admissions in the Case group (32 out of 73) compared to the Control group (4 out of 80). The Chi-square statistic is 41.73 with a p-value of 0.0001, indicating a highly significant difference ($p < 0.05$). This suggests that cases were more likely to require NICU care than controls.(Figure:2)

**Birth Weight :**

The table shows a significant difference in **birth weight distribution** between Case and Control groups (Chi-square = 12.6504, *p* = 0.0055). Cases had more low birth weight infants (1.5–2.5 kg: 25 cases vs. 9 controls), while controls had more infants in the 2.5–3 kg range (52 vs. 42). This suggests cases were associated with lower birth weights.(Figure:3)

**DISCUSSION**

Jaundice in pregnancy, marked by yellowing of skin and mucosa from bilirubin >2 mg/dL, poses serious maternal-fetal risks. More common in low-income settings due to infections, its causes vary by region and include ICP, HELLP, and liver disease. This study explores demographics, causes, outcomes, and effects of targeted treatments.

Our findings revealed a significantly higher mean pulse rate in cases (100.65 bpm) compared to controls (95.23 bpm), suggesting hemodynamic stress or systemic compensation. Tavakolizadeh et al. (2018) and Aracil Moreno et al. (2024) similarly emphasized the link between maternal cardiovascular status and neonatal outcomes. Mean hemoglobin levels were slightly lower in cases (9.23 g/dL vs. 9.39 g/dL; p = 0.1085), while WBC counts were higher (15,732 vs. 13,025.75; p = 0.0679), indicating possible inflammation. These trends align with findings by Jiang et al. (2023) and Luo et al. (2022), who associated elevated maternal inflammatory markers with adverse pregnancy outcomes (9-12).

Mean WBC counts were higher in cases (15,732) than controls (13,025.75; p = 0.0679), suggesting inflammation. Jiang et al. (2023) and Luo et al. (2022) associated elevated WBC, MCV, NLR, and PLR with neonatal jaundice and intrahepatic cholestasis. Post-treatment WBC significantly declined (p = 0.021), and platelet counts were significantly lower (p < 0.0001), with a further drop after treatment (39,369 to 20,660; p = 0.0022), indicating persistent thrombocytopenia. Creatinine

levels were slightly elevated and stable post-treatment, consistent with findings by Zhang et al. (2019), Ciobanu et al. (2016), Reese et al. (2018), Taber-Hight et al. (2020), emphasizing the role of hematologic and renal markers in jaundiced pregnancies (11-13).

Our findings revealed significantly higher serum urea levels in cases (24.24 mg/dL) than controls (21.29 mg/dL; $p < 0.0001$), suggesting renal stress or catabolism, with no significant post-treatment change ($p = 0.8736$). SGPT and SGOT levels were also markedly elevated ($p = 0.0007$ and 0.0013), indicating hepatic dysfunction. These results align with studies by Tesfa et al. (2022), Wu et al. (2022), Mishra et al. (2016), and Mou et al. (2025), which linked elevated urea and liver enzymes to adverse pregnancy outcomes. PT and SGOT levels remained unchanged post-treatment ($p > 0.9$), echoing findings from Ambreen et al. (2015) suggest limited hepatic recovery in jaundiced pregnancies (14-16).

Our findings showed significantly elevated total (5.42 mg/dL) and direct bilirubin (4.74 mg/dL) levels in cases compared to controls (1.44 and 0.86 mg/dL; $p < 0.0001$ and $p = 0.0013$), indicating cholestasis or hepatocellular dysfunction. Post-treatment bilirubin levels declined but not significantly ($p = 0.1451$ for TB, $p = 0.0941$ for DB), suggesting variable therapeutic response. These results align with studies by Kant et al. (2018), Joshi et al. (2022), Bansal et al. (2023), supporting bilirubin's role as both diagnostic and prognostic in jaundiced pregnancies (17-18).

Our findings showed significantly elevated ALP levels in cases (460.21 IU/L) compared to controls (407.13 IU/L; $p = 0.0356$), indicating cholestatic or hepatic involvement, though post-treatment changes were non-significant ($p = 0.4646$), limiting its utility as a response marker. Serum total protein was higher in cases (4.76 g/dL vs. 4.39 g/dL; $p = 0.008$), while albumin was lower (3.45 vs. 3.61 g/dL; $p = 0.1398$). Post-treatment, total protein significantly decreased ($p = 0.0193$) and albumin slightly increased ($p = 0.2516$), suggesting hepatic recovery. These trends are supported by Titaux et al. (2023), Arbib et al. (2021), KARE et al. (2024), Khatun et al. (2020), Gohel et al. (2013) (19-21).

Our findings showed significantly higher uric acid (5.85 vs. 5.34 mg/dL; $p = 0.0019$) and LDH levels (467.2 vs. 408.3 U/L; $p = 0.0034$) in cases, suggesting oxidative stress and tissue damage; LDH. INR levels were slightly higher in cases but not significant ($p = 0.1823$). Adverse outcomes included 4 maternal deaths, 11 gastroenterology referrals, and 32 NICU admissions. These findings

align with Hassen et al. (2022), Burwick et al. (2018), Joshi et al. (2022), and Sharma et al. (2016), highlighting the clinical severity of jaundiced pregnancies (22-24).

Our findings revealed significantly more abnormal USG findings (FGR, IUGR, oligohydramnios, altered Doppler; $p = 0.039$) and low birth weights (<2.5 kg; $p = 0.0055$) in cases, suggesting compromised fetal well-being. These align with Varghese et al. (2016) and Houri et al. (2024), who linked such abnormalities to adverse neonatal outcomes. Age and gestational age were comparable between groups ($p = 0.8721$ and 0.3628), and blood group showed no association ($p = 0.5941$), consistent with Changede et al. (2019), Kanwal et al. (2022), Ayalew et al. (2024), (25-27).

## CONCLUSION

This two-year study, conducted in the Departments of Obstetrics & Gynecology and Gastroenterology at IMS BHU, involved 80 pregnant women with jaundice and 80 controls. It revealed that jaundice in pregnancy is linked to significant maternal complications, including hypertension, hepatic dysfunction, inflammatory responses, and thrombocytopenia. Elevated liver enzymes, bilirubin, and INR levels indicated systemic involvement. It is found that the inflammatory response is notably higher in cases with jaundice then compared to controls. Severe cases can result in sepsis, multi-organ failure, and increased maternal morbidity and mortality.

Our findings shows that jaundice in pregnancy leads to increased risk of FGR, Oligohydrominos, low birth weight and preterm birth which further leads to increased NICU admissions and fetal morbidity and mortality.

The study emphasizes the need for early screening, multidisciplinary care, and larger multicentric research, while noting limitations like small sample size, single center study and lack of long-term follow-up.

**REFERENCES:**

1. Obeagu EI, Katya MCJMUjoM, - HSI. A Systematic Review on Physiological Jaundice: Diagnosis and Management of the Affected Neonates. 2022;2(3):25-41.
2. Privitera G, Agarwal B, Jalan RJEH. Acute liver failure: pathophysiologic basis, and the current and emerging therapies. 2014;1:99-107.
3. Kumari K, Pahuja SK, Kumar SJCSTT. An evolution of bilirubin physiology and analysis. 2023;18(2):1-13.
4. Bigna JJ, Modiyinji AF, Nansseu JR, Amougou MA, Nola M, Kenmoe S, et al. Burden of hepatitis E virus infection in pregnancy and maternofoetal outcomes: a systematic review and meta-analysis. 2020;20:1-11.
5. Alanazi NO, Alharbi FS, Almotairi MMH, Almutairi MSM, Alotaibi NHN, Alshehri OMZ, et al. Bile Tract Emergencies: An Updated Data for Emergency Healthcare Professionals, Nursing, and Clinical Pathologists. 2025;68(2):145-64.
6. Yu X, Yang H, Qi H, Medicine PMBoCMAJM-F. Clinical Management Guidelines for Intrahepatic Cholestasis of Pregnancy. 2023:10.1097.
7. Tavakolizadeh R, Izadi A, Seirafi G, Khedmat L, Mojtahedi SYJEjotm. Maternal risk factors for neonatal jaundice: a hospital-based cross-sectional study in Tehran. 2018;28(3):7618.
8. Aracil Moreno I, Prieto-Arévalo R, Ortega-Abad V, Martín-Manzano V, Pérez-Burrel L, Fraile-López A, et al. Maternal and Perinatal Outcomes in Pregnant Women with Heart Disease: A Case—Control Study. 2024;13(17):5084.
9. Jiang N, Qian L, Lin G, Zhang Y, Hong S, Sun B, et al. Maternal blood parameters and risk of neonatal pathological jaundice: a retrospective study. 2023;13(1):2627.
10. Luo M, Wang L, Yao H, Wen Y, Cao D, Shen W, et al. Diagnostic and prognostic value of blood inflammation and biochemical indicators for intrahepatic cholestasis of pregnancy in Chinese pregnant women. 2022;12(1):20833.
11. Zhang L, Zhang C, Meng Z, Gong L, Pang C, Liu X, et al. Serum bilirubin is negatively associated with white blood cell count. 2019;74:e775.
12. Ciobanu AM, Colibaba S, Cimpoca B, Peltecu G, Panaitescu AMJM. Thrombocytopenia in pregnancy. 2016;11(1):55.
13. Reese JA, Peck JD, Deschamps DR, McIntosh JJ, Knudtson EJ, Terrell DR, et al. Platelet counts during pregnancy. 2018;379(1):32-43.
14. Tesfa E, Munshea A, Nibret E, Mekonnen D, Sinishaw MA, Gizaw STJH. Maternal serum uric acid, creatinine and blood urea levels in the prediction of pre-eclampsia among pregnant women attending ANC and delivery services at Bahir Dar city public hospitals, northwest Ethiopia: A case-control study. 2022;8(10).
15. Wu L, Liu Y, Liu Z, Chen H, Shen S, Wei Y, et al. Serum urea acid and urea nitrogen levels are risk factors for maternal and fetal outcomes of pregnancy: a retrospective cohort study. 2022;19(1):192.
16. Mou AD, Ali NJSR. Investigating the prevalence and associated factors of elevated liver enzymes and dyslipidemia during pregnancy. 2025;15(1):3967.
17. Kant A, Goswami S, Gupta U, Razdan A, Amle DJIJRCOG. Maternal and perinatal outcome in cholestasis of pregnancy: a study in tertiary care hospital in North India. 2018;7(12):5067.

18. Bansal S, Jain S, Rathi PM, Chandnani S, Debnath P, Nawghare PJmjafi. Effects of high bilirubin level in pregnancy in Crigler–Najjar syndrome type 2: An extremely rare but important clinical entity to recognize. 2023;79(5):597-600.
19. Titaux C, Ternynck C, Pauchet M, Stichelbout M, Bizet G, Maboudou P, et al. Total alkaline phosphatase levels by gestational age in a large sample of pregnant women. 2023;132:32-7.
20. Arbib N, Sacks KN, Salman L, Sneh-Arbib O, Berezowsky A, Krispin E, et al. Normal distribution of alkaline phosphatase levels during pregnancy. 2021;48(6):1393-9.
21. Gohel MG, Joshi AG, Anand JS, Makadia JS, Kamariya CPJIJoR, Contraception, Obstetrics, Gynecology. Evaluation of changes in liver function test in first, second and third trimester of normal pregnancy. 2013;2(4):616-21.
22. Hassen FS, Malik T, Dejenie TAJPo. Evaluation of serum uric acid and liver function tests among pregnant women with and without preeclampsia at the University of Gondar Comprehensive Specialized Hospital, Northwest Ethiopia. 2022;17(8):e0272165.
23. Burwick RM, Rincon M, Beeraka SS, Gupta M, Feinberg BBJH. Evaluation of hemolysis as a severe feature of preeclampsia. 2018;72(2):460-5.
24. Sharma S, Aherwar R, Jawade SJIJoR, Contraception, Obstetrics, Gynecology. Maternal and fetal outcome in jaundice complicating pregnancy: a prospective study. 2016;5(4):1084-8.
25. Varghese S, Kour G, Manchanda K, Dhar TJIJoR, Contraception, Obstetrics, Gynecology. Abnormal umbilical artery Doppler in third trimester and perinatal outcome: a retrospective study. 2016;5(7):2196-200.
26. Houri O, Yoskovitz MS, Walfisch A, Pardo A, Geron Y, Hadar E, et al. Neonatal outcomes of infants diagnosed with fetal growth restriction during late pregnancy versus after birth. 2024;13(13):3753.
27. Ayalew T, Molla A, Kefale B, Alene TD, Abebe GK, Ngusie HS, et al. Factors associated with neonatal jaundice among neonates admitted at referral hospitals in northeast Ethiopia: a facility-based unmatched case-control study. 2024;24(1):150.

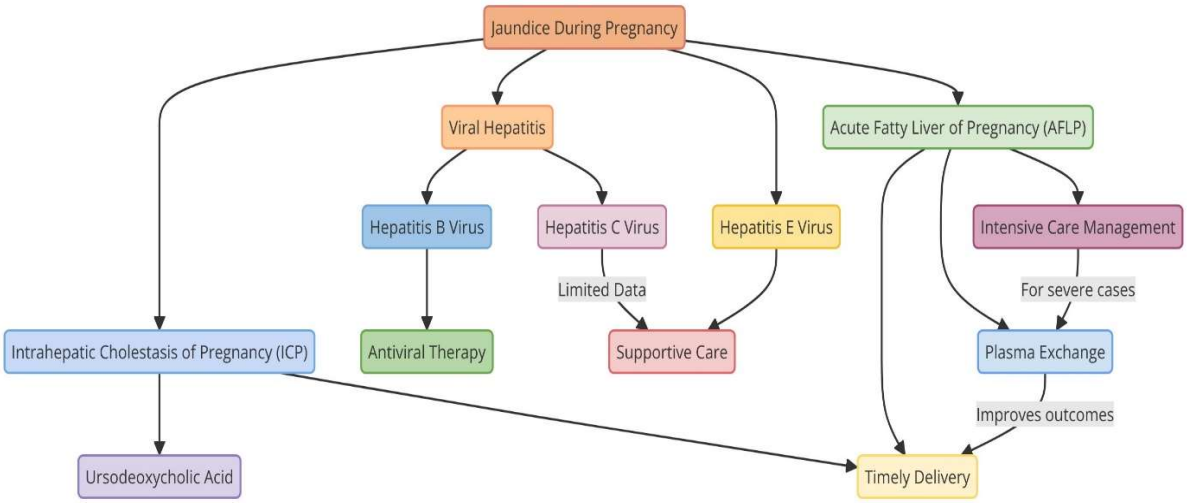

Jaundice During Pregnancy
Viral Hepatitis
Acute Fatty Liver of Pregnancy (AFLP)
Hepatitis B Virus
Hepatitis C Virus
Hepatitis E Virus
Intensive Care Management
Limited Data
For severe cases
Intrahepatic Cholestasis of Pregnancy (ICP)
Antiviral Therapy
Supportive Care
Plasma Exchange
Improves outcomes
Ursodeoxycholic Acid
Timely Delivery

**Figure :1**

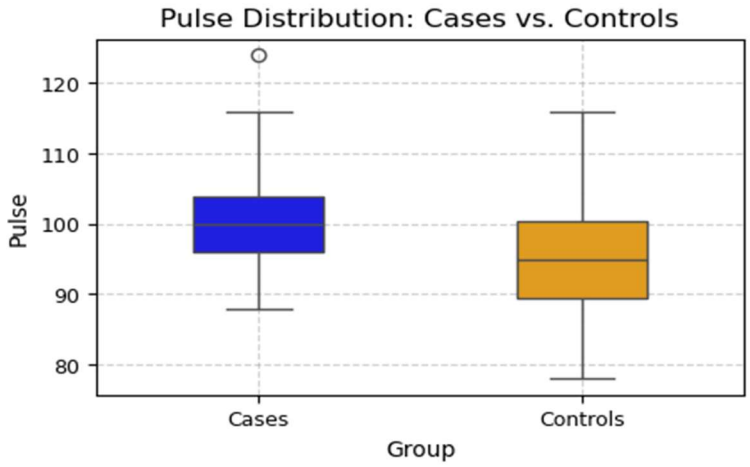



**Figure :2 Pulse Distribution Comparison**

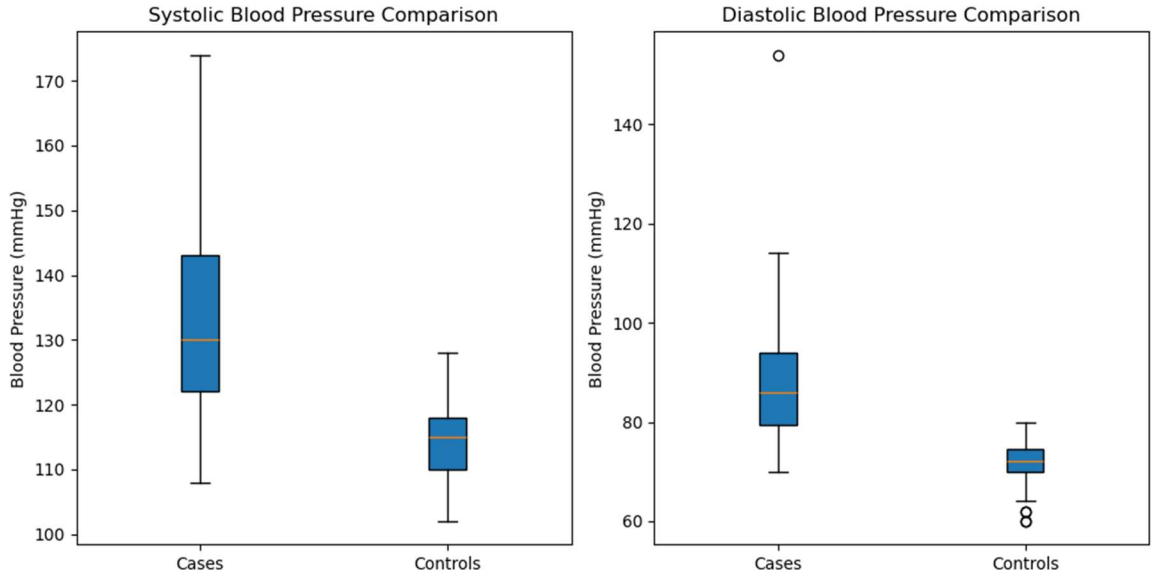



**Figure 3 : Blood pressure Distribution Comparison**

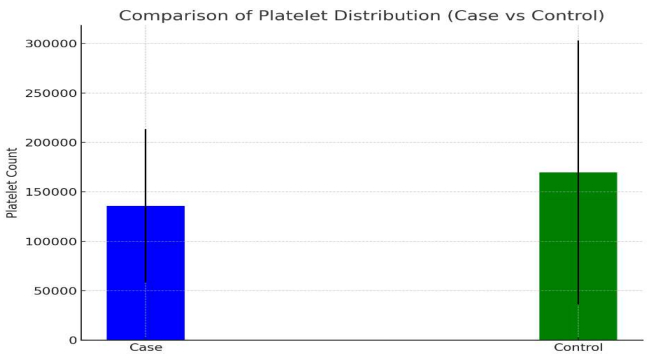



**Figure 4 : Platelets Distribution Comparison**

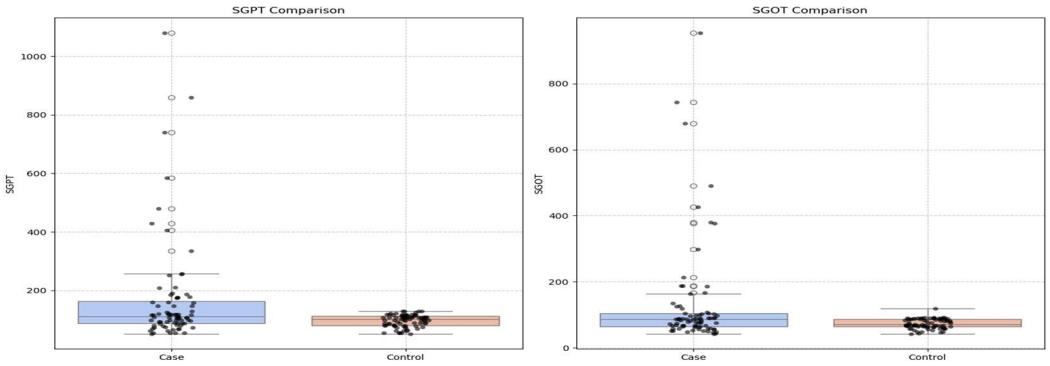



**Figure 5 : SGPT/SGOT Distribution Comparison**

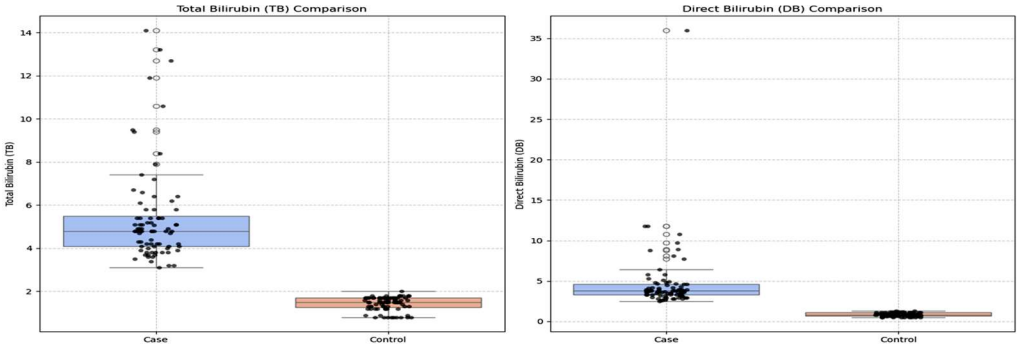
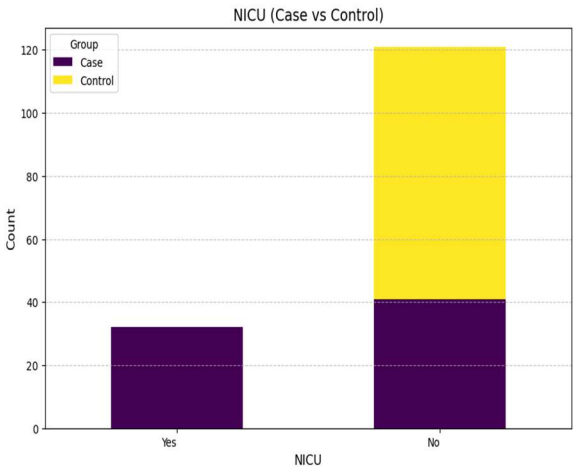



**Figure 6 : TB/DB Distribution Comparison**

**Figure 7 : NICU Admissions**

**Figure 8 : Baby Weight at birth**

Table:



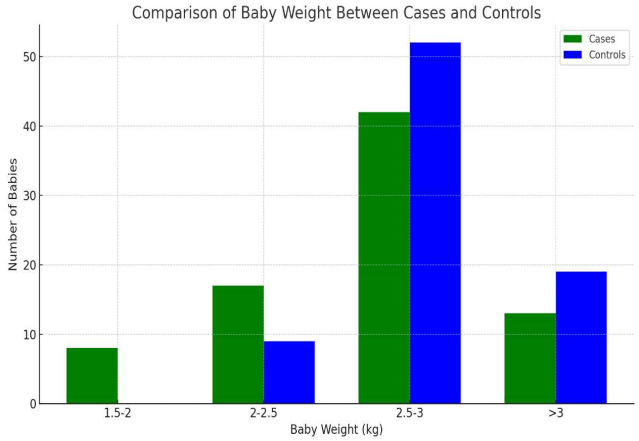



**Table 1 Comparison**

**arison of Hemoglobin, WBC and Platlets Distribution between cases and controls**

| | HEMOGLOBIN | | WBC | | PLATELETS | |
|---|---|---|---|---|---|---|
| | Case | Control | Case | Control | Case | Control |
| COUNT | 80 | 80 | 80 | 80 | 80 | 80 |
| MEAN | 9.23 | 9.38 | 15,732 | 13,025.75 | 1,35,692 | 1,69,677 |
| STANDARD | 1.32 | 1.26 | 12,071.04 | 5,146.89 | 77,627 | 1,33,561 |
| MINIMUM | 5.10 | 5.90 | 6,400 | 8,400 | 40,000 | 1,01,000 |
| MAXIMUM | 11.20 | 11.20 | 1,13,000 | 27,600 | 6,40,000 | 1,31,000 |

t -statistics= -1.6143 and p-value= 0.1085 (Hemoglobin)

t-statistics=1.8445 and p-value= 0.0679(WBC)

t-statistic =1.96768 and p-value = 0.05(Platelet)

**Table :2 Comparison of SGPT, SGOT Distribution between cases and controls**

| | SGPT | | SGOT | |
|---|---|---|---|---|
| | Case | Control | Case | Control |
| Count | 80 | 80 | 80 | 80 |
| Mean | 166.86 | 97.21 | 132.18 | 73.14 |
| Std | 174.79 | 20.49 | 157.34 | 14.71 |
| Min | 52 | 52 | 42 | 42 |
| Max | 1080 | 130 | 954 | 119 |

t-statistic = 3.5398 and p-value= 0.0007(SGPT)

t-statistic = 3.3416 and p-value= 0.0013(SGOT)

**Table :3 Comparison of TB, DB & ALP Distribution between cases and controls**

| | TB | | DB | | ALP | |
|---|---|---|---|---|---|---|
| | Case | Control | Case | Control | Case | Control |
| Count | 80 | 80 | 80 | 80 | 80 | 80 |
| Mean | 5.42 | 1.44 | 4.74 | 0.86 | 460.2125 | 407.125 |
| Std | 2.26 | 0.32 | 4.07 | 0.23 | 160.3104 | 156.607 |
| Min | 3.10 | 0.80 | 2.50 | 0.50 | 205 | 190 |
| Max | 14.10 | 2.00 | 36 | 1.30 | 1120 | 880 |

t-statistic = 1.4658 and p-value= 0.1451(TB)

t-statistic = 1.6861 and p-value= 0.0941(DB)

t-statistic = 2.119 and p-value= 0.0356(ALP)

**Table : 4 Comparison of UREA & LDH Distribution between cases and controls**

| | UREA | | LDH | |
|---|---|---|---|---|
| | Case | Control | Case | Control |
| count | 80 | 80 | 80 | 80 |
| mean | 5.85 | 5.34 | 467.225 | 408.30 |
| std | 1.232 | 0.74 | 138.737 | 109.71 |
| min | 4.3 | 4.2 | 240 | 254 |
| max | 9.0 | 8.0 | 990 | 695 |

t-test = 3.18 and p-value =0.0019(UREA)

t-test = 2.979 and p-value =0.0034(LDH)

**Table : 5 Final outcome of cases and Controls**

| Final Outcome | Case | % | Control | % |
|---|---|---|---|---|
| DISCHARGED | 58 | 72% | 80 | 100% |
| DISCHARGED AND REFERRED TO GASTROENTEROLOGY | 11 | 13.75% | 0 | - |
| DISCHARGED ON PATIENT REQUEST | 1 | 1.25% | 0 | - |
| EXPIRED | 4 | 5% | 0 | - |
| LAMA | 1 | 1.25% | 0 | - |
| TRANSFERRED TO GASTROENTEROLOGY | 5 | 6.25% | 0 | - |
| Chi-square Statistics: 25.507 and p-value: 0.0001 | | | | |

**Table :6 NICU Admissions**

| NICU | CASES | % | Control | % |
|---|---|---|---|---|
| **Yes** | **32** | **40%** | **4** | **5%** |
| **No** | **41** | **51.2%** | **76** | **95%** |
| **Total** | **73** | **91.2%** | **80** | **100%** |

**Table :7 Birth Weight**

| Weight(kg) | Case | Control |
|---|---|---|
| 1.5-2 | 8 | 0 |
| 2-2.5 | 17 | 9 |
| 2.5-3 | 42 | 52 |
| >3 | 13 | 19 |
| Total | 80 | 80 |
| Chi-square statistics: 12.6504 and p-value: 0.0055 | | |