# STUDY OF LABORATORY PARAMETERS IN NEONATAL JAUNDICE

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## ABSTRACT

### INTRODUCTION

Neonatal jaundice is a common phenomenon during the first week of postnatal life affecting almost two thirds of term newborns. Physiological jaundice is seen in about 60% of full term and 70% of preterm babieswhile about 5% of newborn develop pathological jaundice appears usually within 24 hours of age.

### AIMS AND OBJECTIVES

- To study the role of various investigations in diagnosing the etiology of neonatal jaundice.
- To differentiate between physiological and pathological jaundice in new born at earliest.

### MATERIALS AND METHOD

A prospectivestudy was conducted in total 126 neonates having jaundice admitted at Smt. Shardaben general hospital(Smt.NHLMMC) from June 2019 to June 2021.
Investigations like Blood group of baby and mother,Complete blood count of baby, Serum bilirubin level of baby, Direct coomb's test of infant,Indirect coomb's test of mother ,CRP, Glucose-6-phosphate dehydrogenase deficiencywere done.

### RESULT

Preterm neonates were more commonly affected than fullterm neonates.
In our study maximumcases were of physiological jaundice comprising 62.70 % and ABO incompatibility comprising of 23.02%. In the present study total serum bilirubin level was maximum in the case of ABO incompatibility.Direct coomb's test and indirect coomb's test were found to be positive in all 12 cases in Rh incompatibility.

### CONCLUSION

Hematological parameters, blood grouping and serum bilirubin estimation are most important diagnostic methods that assist in etiological diagnosis of neonatal hyperbilirubinemia.Thesehelps in assessing risk of patients and guide the paediatrician for better management of neonatal hyperbilirubinemia.

**KEYWORDS**

Jaundice, ABO, Rh, compatibility, coombs test

## INTRODUCTION

Neonatal jaundice is a common phenomenon during the first week of postnatal life affecting almost two thirds of term newborns[(1)]. The mechanism of neonatal hyperbilirubinemia is multifactorial, comprising primarily processes that contribute to increased bilirubin load, or diminished bilirubin clearance.

The common causes of jaundice in our country in order of frequency include physiological jaundice , premature birth, blood group incompatibility between mother and foetus, infection both intrauterine and postnatal( neonatal septicaemia), G-6 PD deficiency, cephalhematoma, drug induced, RBC disorders and many others. Amongst all of these causes physiological jaundice is seen in about 60% of full term and 70% of preterm babies and appears between 30-72hours of age. While about 5% of newborn babies develop pathological jaundice usually appears within 24 hours of age[(2)].

Though the history and clinical presentation of the new born plays an important role in diagnosing the cause of jaundice. It is also helpful in diagnosing some cases of haemolytic jaundice antenatally by amniocentesis and other recent available modalities and thereby preventing the haemolytic sequelae in the newborn.

## AIMS AND OBJECTIVES

- To study the role of various investigations in diagnosing the etiology of neonatal jaundice.
- To differentiate between physiological and pathological jaundice in new born at the earliest thus to guide paediatrician and facilitating timely interventions.
- To compare the obtained results of the present study with other studies.

## MATERIALS AND METHOD

A prospective observational study was conducted among neonates having jaundice admitted at Smt. Shardaben general hospital(Smt.NHLMMC) from June 2019 to June 2021.
Total 126 cases of neonatal jaundice admitted during the study period mentioned above were included. Detailed history of mother and baby was taken.
Neonates admitted with neonatal jaundice in first 15 days of life are included in this study. Jaundice in preterm and full term neonates both are included in our study. Neonates having serum bilirubin level between 6.3 to 19.9 mg/dl are included.
Following investigations were done in all cases

1. Blood group of baby and mother (ABO / Rh )

Blood group was done by using known anti sera with tube method.

2. Complete blood count of baby:

Including haemoglobin, total WBC count, platelet count on automated 5 part hematoanalyzer by cyanmathhemoglobin methods.

3. Peripheral smear:
Peripheral smear has been stained by field and Giemsa stain. Reticulocyte count has been done by supravital stain-briliantcresyl blue.
4. Serum bilirubin level of baby:

It has been done on auto analyzer Falcon 260 by diazo method ( Modified Jendrassik and Grof's method).
5. Direct coomb's test of infant
6. Indirect coomb's test of mother.
Direct and indirect coomb's test was done by tube method.
7. C- reactive protein:
Detection of C reactive protein has been carried out by Latex agglutination method.
8. Test for Glucose-6-phosphate dehydrogenase deficiency( G-6-PD )
Test for G-6-PD deficiency has been carried out by using Reckon diagnostics reagent kit.

# RESULT

Inthis prospective observational study of 126 neonateswhoadmitted withneonataljaundicefrom June2019toJune2021, laboratory parameterslikeCBC,bloodgroups,serumbilirubinetc.werestudiedandfollowingobservations wereobtained:

**TABLE 1: PRESENTATION OF NEONATAL JAUNDICE ACCORDINGTOAGE INDAYS**

| AGE(INDAYS) | NO.OFCASES | PERCENTAGE |
|---|---|---|
| 1 | 64 | 50.79 % |
| 2 | 5 | 3.97% |
| 3 | 15 | 11.9 % |
| 4 | 17 | 13.49 % |
| 5 | 5 | 3.97% |
| 6 | 7 | 5.56% |
| 7 | 5 | 3.97% |
| 8 | 2 | 1.59% |
| 9 | 0 | 0% |
| 10 | 3 | 2.38% |
| 11 | 2 | 1.59% |
| 12 | 0 | 0% |
| 13 | 0 | 0% |
| 14 | 0 | 0% |
| 15 | 1 | 0.79% |
| TOTAL | 126 | 100% |

Maximum numbers of neonates were of one day age (50.79%). We did notfound asinglecaseofneonatal jaundiceon12$^{th}$,13$^{th}$and14$^{th}$dayofage.

**TABLE 2: GESTATIONAL AGE WISE DISTRIBUTION OFNEONATALJAUNDICE**

| GESTATIONALAGE | NO.OFCASES | PERCENTAGE |
|---|---|---|
| PRETERM | 66 | 52.4% |
| FULLTERM | 60 | 47.6% |
| TOTAL | 126 | 100% |

Pretermneonateswere morecommonlyaffectedthanfulltermneonates.

**TABLE3:GENDERWISEDISTRIBUTIONOFNEONATALJAUNDICE**

| GENDER | NO.OFCASES | PERCENTAGE |
|---|---|---|
| **FEMALE** | 58 | 46.03 % |
| **MALE** | 68 | 53.97 % |
| **TOTAL** | 126 | 100 % |

Incidenceofneonataljaundiceinmalewaspredominantinourstudythanfemale.Male to female ratio is1.17:1 inthisstudy.

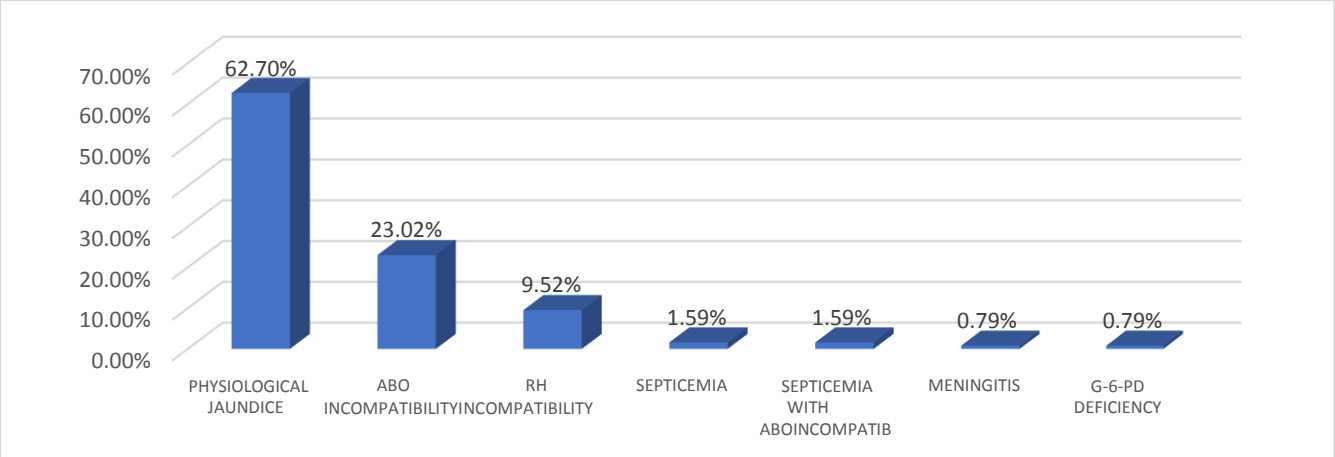
**TABLE 4: TOTAL NUMBER OF PHYSIOLOGICAL ANDPATHOLOGICALJAUNDICE**

| ETIOLOGY | NO.OFCASES | PERCENTAGE |
|---|---|---|
| PHYSIOLOGICALJAUNDICE | 79 | 62.70% |
| PATHOLOGICALJAUNDICE | 47 | 37.30% |
| **TOTAL** | **126** | **100%** |

Physiologicaljaundiceconstituted79cases(62.70%)whichweremorecommonthan pathologicaljaundiceconstituted47cases (37.30%)

**TABLE5:TOTALNUMBEROFCASESANDPERCENTAGEACCORDING TO ETIOLOGY**

| ETIOLOGY | NO.OF CASES | PERCENTAGE |
|---|---|---|
| • PHYSIOLOGICALJAUNDICE | 79 | 62.7 % |
| • PATHOLOGICALJAUNDICE | | |
| 1.ABOINCOMPATIBILITY | 29 | 23.02 % |
| 2.RHINCOMPATIBILITY | 12 | 9.52% |
| 3.SEPTICEMIA | 2 | 1.59% |
| 4.SEPTICEMIA WITH ABOINCOMPATIBILITY | 2 | 1.59% |
| 5.MENINGITIS | 1 | 0.79% |
| 6.G-6-PDDEFICIENCY | 1 | 0.79% |
| **TOTAL** | **126** | **100%** |




Inourstudymaximumnumberofcaseswereofphysiologicaljaundicecomprising 79 cases (62.70 %) and among 47 cases of pathological jaundicemaximumcaseswereofABOincompatibilitycomprisingof29cases(23.02 %).

**TABLE 6:RANGE OF HAEMOGLOBIN LEVEL ACCORDING TOVARIOUSETIOLOGY**

| **ETIOLOGY** | **RANGEOFHAEMOGLOBIN LEVEL(gm/dl)** |
|---|---|
| ABOINCOMPATIBILITY | 11.7-18.6 |
| RHINCOMPATIBILITY | 8.3-19 |
| SEPTICEMIA | 15-19.8 |
| SEPTICEMIA WITH ABO INCOMPATIBILITY | 17.3-20.7 |
| MENINGITIS(SINGLECASE) | 9.8 |
| G-6-PD DEFICIENCY (SINGLECASE) | 16 |
| PHYSIOLOGICALJAUNDICE | 12.5-20.9 |

Thedecreaseinhaemoglobin(gm/dl)levelwasfoundtobemoreinRhincompatibilitycasesfollowedbysingle caseof meningitis.

**TABLE7:RANGEOFBILIRUBINLEVELACCORDINGTOVARIOUSTYPES OF JAUNDICE**

| **ETIOLOGY** | **RANGE OF TOTALS.BILIRUBIN(mg/dl)** |
|---|---|
| ABOINCOMPATIBILITY | 6.3-19.9 |
| RHINCOMPATIBILITY | 9.7-18.7 |
| SEPTICEMIA | 9.7-15.2 |
| SEPTICEMIA WITH ABOINCOMPATIBILITY | 9.2-17.7 |
| MENINGITIS(SINGLECASE) | 10.6 |
| G-6-PD DEFICIENCY (SINGLECASE) | 13.4 |
| PHYSIOLOGICALJAUNDICE | 6.3-18.2 |

Inthepresentstudytotalserumbilirubinlevelwasmaximuminthecase ofABOincompatibilityfollowedbyRhincompatibility.

**TABLE-8 RANGE OF RETICULOCYTE COUNT ACCORDING TOETIOLOGY**

| **ETIOLOGY** | **RANGE OF RETICULOCYTEC OUNT(%)** |
|---|---|
| ABOINCOMPATIBILITY | 2-6% |
| RHINCOMPATIBILITY | 2-9% |
| SEPTICEMIA | 2% |
| SEPTICEMIA WITH ABOINCOMPATIBILI TY | 2-5% |
| MENINGITIS(SINGLECASE) | 4% |
| G-6-PD DEFICIENCY (SINGLECASE) | 4% |
| PHYSIOLOGICALJAUNDICE | 2-6% |

Highestreticulocytecount(9%)wasfoundinRhincompatibility

**TABLE9: RESULTOFDIRECTANDINDIRECTCOOMBS'TEST**

| **ETIOLOGY** | DCT | | | | ICT | | | |
|---|---|---|---|---|---|---|---|---|
| | +VE | -VE | TOTAL | % | +VE | -VE | TOTAL | % |
| NEONATALJAUNDICE DUE TORHINCOMPATIBILITY | 12 | 0 | 12 | 100% | 12 | 0 | 12 | 100% |
| NEONATALJAUNDICE + ABOINCOMPATIBILITY | 5 | 24 | 29 | 17.24% | 5 | 24 | 29 | 17.2% |

Direct coomb's test and indirect coomb's test were found to be positive in all 12cases(100%)inRhincompatibilitywhiletheywerepositivein5cases(17.24%)inA

BOincompatibility.

**TABLE 10: RESULT OF C-REACTIVE PROTEIN IN VARIOUSETIOLOGY**

| ETIOLOGY | NO.OF CASES | CRPPOSITIVE | PERCENTAGE |
|---|---|---|---|
| ABOINCOMPATIBILITY | 29 | 0 | 0% |
| RHINCOMPATIBILITY | 12 | 0 | 0% |
| SEPTICEMIA | 2 | 2 | 100% |
| SEPTICEMIA WITH ABOINCOMPATIBILITY | 2 | 2 | 100% |
| MENINGITIS | 1 | 1 | 100% |
| G-6-PDDEFICIENCY | 1 | 0 | 0% |
| PHYSIOLOGICALJAUNDICE | 79 | 1 | 1.25% |

C-Reactive protein test was positive in the all cases of septicemia, a case ofmeningitis and in a single case among all cases of physiological jaundice in thisstudy.

# DISCUSSION

Jaundice is the most common condition that requires medical attention andhospital admission in newborns. The yellow coloration of the skin and sclera innewborns with jaundice is the result of accumulation of unconjugated bilirubin.In most infants, unconjugated hyperbilirubinemia reflects a normal transitionalphenomenon.Howeverinsomeinfants,serumbilirubinlevelsmayriseexcessively, which can be cause for concern because unconjugated bilirubin isneurotoxic and can cause death in new born and lifelong neurologic sequelae ininfants who survive( kernicterus). For this reason, the presence of neonataljaundiceshouldundergodiagnosticevaluation.

In this study laboratory parameters of various types of neonatal jaundice hasbeen evaluatedand comparedwithotherstudies.

**Table11:Comparisonofetiologyofpresentstudywithvariousotherstudies**

| Etiology | Kassa RT et$_{al}$[3] | ThakkarB[4] | Shah A et$_{al}$[5] | Present Study |
|---|---|---|---|---|
| RHINCOMPATIBILITY | 8.8% | 10% | 8% | 9.52% |
| ABOINCOMPATIBILITY | 35.6% | 16.7% | 15% | 23.02% |
| NEONATALJAUNDICEWITHSEPTICEMIA | 18.8% | 10% | 12% | 3.17% |
| G-6-PDDEFICIENCY | 0% | 6.6% | 3% | 0.79% |
| PHYSIOLOGICAL JAUNDICE | 13.8% | 56.7% | 62% | 62.70% |

In present study percentage of neonatal jaundice due to Rh incompatibility is9.52% which is comparable withThakkar B[4] study in which percentage ofneonatal jaundice due to Rh incompatibility is 10% and with Kassa RT et al[3] inwhich Rh incompatibility comprising 8.8%.Total percentage of physiologicaljaundice in present study is 62.70% is comparable with Shah A et al[5] study(62%)andThakkar B[4] study(56.7%).

Gestationalagewisedistribution:

The present study includes 126 cases of Neonatal jaundice. Among them 66(52.4%) were preterm and 60(47.6%) were term. In a study done by Kumar RKin 1999, they found about 50 percent of term and 80 percent of preterm infantsdeveloped jaundice in the first week of life[6].Bhutani et al[7] in their study foundout that prematurity was a significant risk factor for hyperbilirubinemia and isknown to be a basis for increased biologic vulnerability to risk of bilirubininduced neurotoxicity.

Laboratoryparameters :

InpresentstudyHblevelrangeof8.3-20.9gm/dl.Similarfindingswerenotedin the study carried out by Joshi et al[8]. The findings of their study showed Hblevel8-19.4 gm/dl.The result was almost similar toourstudy.

InourstudyserumbilirubinwashighestinABOincompatibilityandRhincompatibility.Among61.1%ofcasesrangeofserum totalbilirubinwasfound14-19.9mg/dl. Same resultswere observedin the study of NepalD etal[9].Theymentioned thatmaximum number of infants' peak serum bilirubinfell inthe range of15-19.9mg/dl.

In all cases of septicemia CRP was positive in present study. It is an acute phasereactant;issynthesizedbytheliveranditbecomespositiveafteranyinflammation.Itisveryreliableindicator.

Direct coomb's test and indirect coomb's test were found to be positive in allcases in Rh – incompatibility while they were positive in 17.24% of cases inABO incompatibility. The reason for this difference may have been that ''A''and''B'' antigens are weaker antigensand the distance between a/b antigensites on thefetal red cells as comparedtoadult red cellsis more.

# CONCLUSION

Themechanismofneonatalhyperbilirubinemiaismultifactorial,maybeidiopathicorpathological.Increasedlevelofunconjugatedbilirubinisneurotoxic and can cause death in new born or lifelong neurologic deficit. So,early diagnosis and treatment is essential to prevent serious complications ofneonatal hyperbilirubinemia . Hematological parameters, blood grouping andserum bilirubin estimation are simple and most important diagnostic methodsthatassistinetiologicaldiagnosisofneonatalhyperbilirubinemia.Theselaboratoryparametershelpsinassessingriskofpatientsandguidethepaediatricianforbettermanagementofneonatalhyperbilirubinemia.

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