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RISK FACTORS AND SEVERITY OF NEONATAL HYPERBILIRUBINEMIA AMONG ADMITTED NEONATES IN SWAT, KHYBER PAKHTUNKHWA, PAKISTAN

MUHAMMAD ZEESHAN^{1*}, SAYED MUHAMMAD HASSAN², IZHAR UDDIN³, ASHFAQ AHMAD⁴, ASAD KHAN⁵, AKHTAR IQBAL⁶, DR. NAUSHEEN NAZIR⁷, DR. MEHREEN GHIA⁸

^{1, 2, 7}Department of Biochemistry, University of Malakand, Lower Dir, KPK, Pakistan.

³Institute of Basic Medical Sciences, Khyber Medical University, Peshawar, KPK, Pakistan.

³MPhil Haematology, Lecturer and Lab Supervisor at Muhammad medical college, Mirpurkhas, Sindh, Pakistan.

³Nantong university china school of public health, Nantong, China.

³School of Allied Health Sciences, Iqra National University Peshawar, KPK, Pakistan.

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ABSTRACT

Objective: To determine the incidence and levels of total, direct, and indirect serum bilirubin, and to identify the clinical and demographic determinants of neonatal hyperbilirubinemia among jaundiced neonates admitted to a tertiary care hospital in district Swat, Khyber Pakhtunkhwa, Pakistan.

Methodology: This descriptive cross-sectional study was conducted in the NICU of Saidu Group of Teaching Hospitals, Swat, over six months, enrolling 160 neonates with jaundice. Neonates without jaundice and those not admitted at SGTH were excluded. Data on sociodemographic and clinical variables, along with serum bilirubin levels, were collected via a structured questionnaire. Analysis was performed using SPSS version 22, summarizing categorical variables as frequencies and percentages, with associations tested using the chi-square test ($p < 0.05$ significant).

Results: Of 160 neonates studied, 25% were preterm, 75% term, and 59.4% male. High total bilirubin levels (13–18 mg/dL) were found in 50.6%, with severe cases (>18 mg/dL) at 27.5%. Direct bilirubin was moderately elevated in 53.1%, while 52.5% had severely raised indirect bilirubin (>13 mg/dL). Significant associations with total bilirubin included age at admission and mode of delivery ($p < 0.001$), as well as breastfeeding ($p = 0.006$ for total and $p = 0.029$ for indirect bilirubin). Key determinants of hyperbilirubinemia included ABO incompatibility (18.1%), Rh incompatibility (13.8%), neonatal sepsis (15.6%), and breastfeeding (49.4%). Home delivery correlated with cephalohematoma, meconium aspiration syndrome, and neonatal mortality ($p < 0.05$). Overall mortality due to hyperbilirubinemia was 5.6%, significantly linked to higher bilirubin levels ($p = 0.025$ for total and $p = 0.014$ for direct).

Conclusion: Neonatal hyperbilirubinemia remains a substantial burden in this population, with late presentation, non-facility delivery, prolonged/excessive breastfeeding, and blood-group incompatibilities emerging as the principal determinants of severe bilirubin elevation and mortality.

Keywords: Neonatal jaundice; Hyperbilirubinemia, Risk Factor of Jaundice, Bilirubin level, Swat, Pakistan.

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*Corresponding Author

Mr. Muhammad Zeeshan

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INTRODUCTION

Neonatal hyperbilirubinemia, clinically manifesting as one of the most prevalent conditions is jaundice, encountered in newborns and a leading cause of hospital readmission in the first week of life [1,2]. It results from an elevated total serum bilirubin (TSB) that accumulate in the skin and mucous membranes, and it affects up to 60–85% of term and near-term infants to varying degrees [3]. Because neonatal erythrocytes have a shorter lifespan and the hepatic conjugating enzyme uridine diphosphate glucuronosyltransferase (UGT1A1) is

physiologically immature, bilirubin production in newborns exceeds their capacity for clearance, particularly in the first days of life [4].

Most neonatal jaundice is “physiological” and self-limiting, but a minority of infants develop “pathological” jaundice driven by hemolysis, sepsis, birth trauma or prematurity, in whom unconjugated bilirubin can cross the blood–brain barrier and cause acute bilirubin encephalopathy or kernicterus [5]. Recognized risk factors include prematurity, ABO and Rh blood group incompatibility, glucose-6-phosphate dehydrogenase (G6PD) deficiency, cephalohematoma or significant bruising, neonatal sepsis, meconium aspiration syndrome, birth asphyxia, exclusive or excessive breastfeeding, male sex, and delivery outside a healthcare facility [6,7,8]. The relative contribution of these determinants varies substantially by population, and severe hyperbilirubinemia and its neurological

sequelae remain disproportionately common in low- and middle-income countries, where facility-based delivery, timely postnatal screening and phototherapy access are inconsistent [9].

In Pakistan, hospital-based studies have reported a substantial burden of neonatal hyperbilirubinemia, with locally relevant determinants including ABO and Rh incompatibility, G6PD deficiency, prematurity, and delayed presentation [10, 11]. However, data specific to district Swat, Khyber Pakhtunkhwa a region with mixed facility- and home-based delivery practices are lacking. Establishing local incidence figures and determinants of severe bilirubin elevation is essential for guiding screening protocols, staff training and community awareness in this setting.

Thus, the purpose of this investigation was to find out the incidence and severity of total, direct, and indirect serum bilirubin, and to identify the demographic and clinical determinants of hyperbilirubinemia among neonates admitted with jaundice to the Neonatal Intensive Care Unit (NICU) of a tertiary care hospital in Swat, Khyber Pakhtunkhwa, Pakistan.

METHODOLOGY

This descriptive, hospital-based cross-sectional study focused on neonates with clinically apparent jaundice admitted to the Neonatal Intensive Care Unit (NICU) of Saidu Group of Teaching Hospitals (SGTH) in Swat, Khyber Pakhtunkhwa, Pakistan. The study spanned approximately six months, with data collection occurring over a two-month period post-approval from the Department of Biochemistry at the University of Malakand and authorization from the hospital administration. A total of 160 neonates were enrolled, specifically those presenting with jaundice; neonates without jaundice and those treated externally were excluded. Prior to data abstraction, written informed consent was secured from the parents or guardians of each participant, ensuring the confidentiality and anonymization of data. Data were gathered using a structured questionnaire divided into three sections: (A) demographics and obstetric factors, including gestational age, sex, age at admission, birth weight, mode, and place of delivery; (B) clinical determinants of neonatal jaundice such as cephalohematoma, ABO and Rh incompatibility, neonatal sepsis, breastfeeding status, G6PD deficiency, meconium aspiration syndrome (MAS), birth asphyxia, and mortality linked to hyperbilirubinemia; and (C) biochemical measurements encompassing total, direct, and indirect serum bilirubin levels. For analysis, total bilirubin was classified as normal (2–13 mg/dL), high (13–18 mg/dL), or severe (>18 mg/dL); direct bilirubin was categorized as normal (0.0–0.3 mg/dL), moderate (0.4–1.0 mg/dL), high (1.0–3.0 mg/dL), or severe (>3 mg/dL); while indirect bilirubin levels were deemed normal (0.26–5 mg/dL), high (5–13 mg/dL), or severe (>13 mg/dL), adhering to the thresholds established in the associated parent thesis. Data analysis was conducted via the Statistical Package for Social Sciences (SPSS), version 22, utilizing descriptive statistics (frequencies and percentages) presented in tabular format. Additionally, chi-square tests assessed relationships between demographic/clinical factors and bilirubin categories, with a significance threshold set at $p <$

0.05, acknowledging the study's design as purely descriptive and univariate associations without multivariable adjustment.

RESULTS

A total of 160 jaundiced neonates were enrolled. Of these, 40 (25.0%) were preterm and 120 (75.0%) were term; 95 (59.4%) were male and 65 (40.6%) female. At the time of admission, 4 (2.5%) were new-borns (<24 hours), 22 (13.8%) were aged 1–3 days, 99 (61.9%) were aged 4–7 days, and 35 (21.9%) were admitted after 7 days of birth. Birth weight was <2.5 kg in 37 (23.1%), 2.5–4.0 kg in 119 (74.4%), and >4.0 kg in 4 (2.5%) neonates. Delivery was vaginal in 110 (68.8%) and by caesarean section in 50 (31.3%); 133 (83.1%) neonates were delivered in a health facility and 27 (16.9%) at home (Table 01).

Table 01: Demographic and obstetric characteristics of the study neonates (n=160)

Variable	n	%	Variable	n	%
Gestational age – Preterm	40	25.0	Mode of delivery – Vaginal	110	68.8
Gestational age – Term	120	75.0	Mode of delivery – Caesarean section	50	31.3
Sex – Male	95	59.4	Place of delivery – Hospital (facility)	133	83.1
Sex – Female	65	40.6	Place of delivery – Home	27	16.9
Age at admission – Newborn (<24h)	4	2.5	Birth weight – <2.5 kg	37	23.1
Age at admission – 1–3 days	22	13.8	Birth weight – 2.5–4.0 kg	119	74.4
Age at admission – 4–7 days	99	61.9	Birth weight – >4.0 kg	4	2.5
Age at admission – >7 days	35	21.9	Total	160	100.0

On biochemical assessment, 35 neonates (21.9%) had normal total bilirubin, 81 (50.6%) had high total bilirubin (13–18 mg/dL), and 44 (27.5%) had severe rise (>18 mg/dL). Direct bilirubin was normal in 19 (11.9%), moderately high in 85 (53.1%), high in 38 (23.8%), and severely high in 18 (11.3%). Indirect bilirubin was normal in only 4 (2.5%), high in 72 (45.0%), and severely high in 84 (52.5%) neonates (Table 02).

Table 02: Distribution of total, direct and indirect serum bilirubin levels among study neonates (n=160)

Bilirubin category	Total bilirubin n (%)	Direct bilirubin n (%)	Indirect bilirubin n (%)
Normal (Total 2–13 mg/dL; Direct 0–0.3 mg/dL; Indirect 0.26–5 mg/dL)	35 (21.9)	19 (11.9)	4 (2.5)
Moderate/High (Total 13–18 mg/dL; Direct 0.4–1.0 mg/dL; Indirect 5–13 mg/dL)	81 (50.6)	85 (53.1)	72 (45.0)
High (Direct 1.0–3.0 mg/dL only)	—	38 (23.8)	—
Severe (Total >18 mg/dL; Direct >3 mg/dL; Indirect >13 mg/dL)	44 (27.5)	18 (11.3)	84 (52.5)
Total	160 (100)	160 (100)	160 (100)

Determinants recorded among the study neonates were: breastfeeding, 79 (49.4%); ABO incompatibility, 29 (18.1%); neonatal sepsis, 25 (15.6%); Rh incompatibility, 22 (13.8%); cephalohematoma/significant bruising, 16 (10.0%); G6PD deficiency, 13 (8.1%); meconium aspiration syndrome, 7 (4.4%); and birth asphyxia, 3 (1.9%). Nine neonates (5.6%) died as a consequence of hyperbilirubinemia during the study period (Table 03).

Table 03: Frequency of clinical determinants of neonatal hyperbilirubinemia and mortality outcome (n=160)

Determinant	Present, n (%)	Absent, n (%)
Breastfeeding	79 (49.4)	81 (50.6)
ABO incompatibility	29 (18.1)	131 (81.9)
Neonatal sepsis	25 (15.6)	135 (84.4)
Rh incompatibility	22 (13.8)	138 (86.3)
Cephalohematoma / significant bruising	16 (10.0)	144 (90.0)
G6PD deficiency	13 (8.1)	147 (91.9)
Meconium aspiration syndrome (MAS)	7 (4.4)	153 (95.6)
Birth asphyxia	3 (1.9)	157 (98.1)
Neonatal death attributable to hyperbilirubinemia	9 (5.6)	151 (94.4)

On chi-square testing, age at admission was significantly associated with both total bilirubin ($\chi^2=21.64$, $df=6$, $p=0.001$) and indirect bilirubin ($\chi^2=17.14$, $df=6$, $p=0.009$): neonates admitted after 7 days of life had the highest proportion of severe indirect hyperbilirubinemia (65.7%), consistent with a greater likelihood of pathological, late-presenting jaundice in this group. Mode of delivery was significantly associated with total ($\chi^2=13.56$, $p=0.001$), direct ($\chi^2=8.09$, $p=0.044$) and indirect ($\chi^2=8.43$, $p=0.015$) bilirubin, with vaginally delivered neonates showing a higher proportion of severely raised indirect bilirubin (59.1%) than those delivered by caesarean section (38.0%). Breastfeeding was significantly associated with total bilirubin ($\chi^2=10.29$, $p=0.006$) and indirect bilirubin ($\chi^2=7.08$, $p=0.029$); 60.8% of breastfed neonates had severely raised indirect bilirubin compared with 44.4% of non-breastfed neonates (Table 04).

Neonatal mortality attributable to hyperbilirubinemia was significantly associated with total bilirubin ($\chi^2=7.34$, $p=0.025$) and direct bilirubin ($\chi^2=10.69$, $p=0.014$) categories, and with gestational age ($\chi^2=8.83$, $p=0.003$), place of delivery ($\chi^2=10.17$, $p=0.001$) and, on trend, with female sex. Place of (home) delivery was also significantly associated with cephalohematoma/significant bruising ($\chi^2=5.39$, $p=0.020$) and meconium aspiration syndrome ($\chi^2=8.46$, $p=0.004$), while mode of delivery was significantly associated with breastfeeding practice ($\chi^2=6.88$, $p=0.009$). Male sex was significantly associated with G6PD deficiency ($\chi^2=6.36$, $p=0.012$). No statistically significant association was found between gestational age, sex, birth weight, cephalohematoma, ABO incompatibility, Rh incompatibility, neonatal sepsis, G6PD deficiency, meconium aspiration syndrome or birth asphyxia and total bilirubin category ($p>0.05$ for each) (Table 04).

Table 04: Statistically significant associations between demographic/clinical determinants and serum bilirubin categories or mortality (χ^2 test, $p<0.05$)

Determinant	Bilirubin parameter / outcome	χ^2	df	p-value
Age at admission	Total bilirubin	21.64	6	0.001
Age at admission	Indirect bilirubin	17.14	6	0.009
Mode of delivery	Total bilirubin	13.56	2	0.001
Mode of delivery	Direct bilirubin	8.09	3	0.044
Mode of delivery	Indirect bilirubin	8.43	2	0.015
Breastfeeding	Total bilirubin	10.29	2	0.006
Breastfeeding	Indirect bilirubin	7.08	2	0.029
Gestational age	Neonatal death	8.83	1	0.003

Determinant	Bilirubin parameter / outcome	χ^2	df	p-value
Place of delivery	Neonatal death	10.17	1	0.001
Place of delivery	Cephalohematoma/bruising	5.39	1	0.020
Place of delivery	Meconium aspiration syndrome	8.46	1	0.004
Mode of delivery	Breastfeeding practice	6.88	1	0.009
Sex	G6PD deficiency	6.36	1	0.012
Neonatal death	Total bilirubin	7.34	2	0.025
Neonatal death	Direct bilirubin	10.69	3	0.014

The pattern of association between independent variables and each bilirubin fraction (direct, indirect and total) is summarized graphically in Figure 01. Age at admission, mode of delivery, place of delivery, Rh incompatibility and neonatal death showed the lowest p-values ($p < 0.05$) against total and/or direct bilirubin, while ABO incompatibility, birth asphyxia, birth weight, G6PD deficiency, gestational age, meconium aspiration syndrome and neonatal sepsis did not reach significance for any bilirubin fraction.

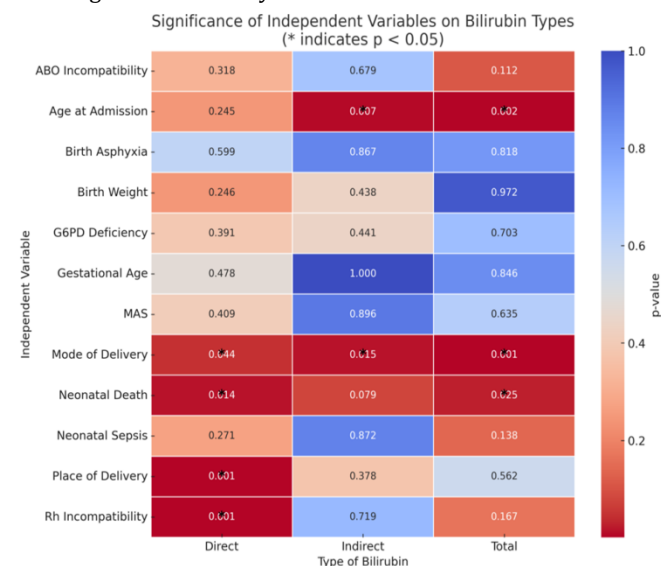


Figure 01: Heatmap of p-values for the association of independent variables with direct, indirect and total bilirubin levels (chi-square test; * indicates $p < 0.05$).

Figure 02 summarizes the significance of association between key neonatal variables (gestational age, age at admission, birth weight, mode of delivery, breastfeeding and place of delivery) and clinical conditions/outcomes. Place of delivery was significantly associated with bruising/cephalohematoma (Sig) and highly significantly associated with meconium aspiration syndrome and neonatal death (HS), while neonate birth weight

was significantly associated with bruising/cephalohematoma (Sig) and highly significantly associated with neonatal death (HS). Age at admission showed a highly significant association with birth asphyxia and a trend-level association ($p < 0.10$) with ABO incompatibility, and gestational age was highly significantly associated with neonatal death. Breastfeeding showed only trend-level associations with ABO incompatibility, Rh incompatibility, birth asphyxia and neonatal death, and mode of delivery showed no significant association with any of the clinical conditions assessed.

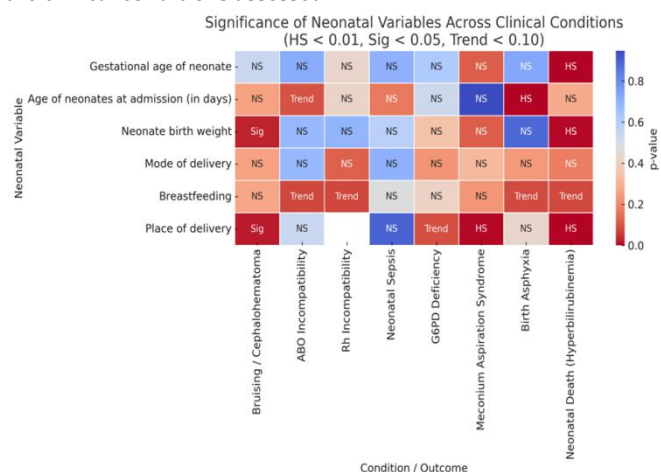


Figure 02: Heatmap of significance levels (NS = not significant, Trend = $p < 0.10$, Sig = $p < 0.05$, HS = highly significant $p < 0.01$) of key neonatal variables across clinical conditions and outcomes associated with neonatal hyperbilirubinemia.

DISCUSSION

In this cohort of 160 jaundiced neonates from Swat, Khyber Pakhtunkhwa, more than three-quarters had elevated total bilirubin (high or severe), and over half had severely raised indirect bilirubin, indicating a predominantly unconjugated hyperbilirubinemia pattern consistent with the expected physiology of haemolytic and breastfeeding-associated jaundice in this age group. This proportion of severe elevation is higher than that reported from some regional cohorts and broadly in line with hospital-based Pakistani data, which have similarly described a substantial burden of significant hyperbilirubinemia among NICU admissions [10,11,12]. Sgro et al reported a mean peak bilirubin of 471 $\mu\text{mol/L}$ (approximately 27.5 mg/dL) among 258 confirmed cases of severe neonatal hyperbilirubinemia in Canada, underscoring that even in well-resourced health systems a subset of neonates present with markedly elevated bilirubin requiring urgent intervention [13].

Late admission (after day 4, and particularly beyond day 7) was significantly associated with higher total and indirect bilirubin in our study, mirroring the pattern described by Fanello et al in the Democratic Republic of Congo, where preterm birth, excessive weight loss, G6PD deficiency and hyperbilirubinemia at birth were identified as high-risk features, and where maternal understanding of jaundice was reasonable but adherence to phototherapy was poor once symptoms subsided [8]. This suggests that delayed recognition and delayed care-seeking, rather than biological predisposition alone, may drive the severity seen in late-presenting neonates

in our setting, an interpretation consistent with reports from other low-resource settings emphasizing gaps in postnatal follow-up and caregiver awareness.

Breastfeeding showed a significant association with both total and indirect bilirubin elevation in our study, in keeping with the well-described entity of breastfeeding-associated jaundice, and echoing findings from Nepal and elsewhere where breastfeeding practices were identified as important, context-dependent risk or protective factors for neonatal jaundice depending on feeding adequacy [14]. Similarly, mode and place of delivery were significant determinants in our cohort: neonates delivered at home and those delivered vaginally had a higher burden of cephalohematoma, meconium aspiration syndrome and mortality than those delivered by caesarean section in a health facility, a pattern consistent with the protective effect of facility-based delivery on jaundice-related complications reported from Bhutan [7].

Rh incompatibility (13.8%), G6PD deficiency (8.1%), ABO incompatibility (18.1%), and newborn sepsis (15.6%) were the principal immunological and enzymatic determinants identified, similar in rank order to studies from Bahrain and Saudi Arabia in which ABO incompatibility and G6PD deficiency were consistently the leading identifiable causes of indirect hyperbilirubinemia [15,16]. Isa et al, in a large Bahraini cohort, likewise reported ABO incompatibility as the most frequent cause of indirect hyperbilirubinemia, followed by G6PD deficiency, with significantly higher bilirubin levels among ABO-incompatible neonates [15]. The male preponderance of G6PD deficiency observed here ($\chi^2=6.36$, $p=0.012$) is consistent with its X-linked inheritance and with findings from Egypt, where G6PD-deficient neonates had significantly higher bilirubin levels than those without the deficiency [17].

Our overall case-fatality of 5.6% attributable to hyperbilirubinemia, concentrated among neonates with the highest total and direct bilirubin levels, reinforces the well-established relationship between severe, particularly delayed-treated, hyperbilirubinemia and adverse neonatal outcomes, and is consistent with global estimates that a meaningful proportion of severe hyperbilirubinemia cases progress to kernicterus or neonatal death, especially in under-resourced settings [16]. Comparable Pakistani hospital-based work by Tikmani et al and Tahir et al has similarly highlighted a substantial population burden of neonatal hyperbilirubinemia and the diagnostic value of early (including cord-blood) bilirubin assessment for identifying at-risk neonates, supporting the case for earlier, more systematic screening in facilities such as ours [12,18].

STUDY LIMITATIONS

This study has several limitations. It was a single-centre, hospital-based studies with a modest sample size, which may limit generalizability to the wider population of Swat and to community-managed jaundice that never reaches hospital care. The cross-sectional design and univariate chi-square analysis preclude causal inference and do not adjust for potential confounding between correlated risk factors (for example, place and mode of delivery). Bilirubin categorization thresholds, rather than continuous bilirubin values, were used

for analysis, which may reduce statistical sensitivity to detect some associations. Prospective, multicentre studies with multivariable analysis are recommended to confirm these determinants and their relative contributions.

CONCLUSION

Neonatal hyperbilirubinemia is a significant clinical burden among neonates admitted to this tertiary care hospital in Swat, with severe elevations of indirect bilirubin predominating. Late admission beyond the first week of life, home and vaginal delivery, and breastfeeding practices were significant, potentially modifiable determinants of bilirubin severity, while ABO/Rh incompatibility, neonatal sepsis and G6PD deficiency remained important identifiable causes. Death attributable to hyperbilirubinemia, though relatively uncommon, was concentrated among neonates with the highest bilirubin levels. Strengthening facility-based deliveries, promoting timely postnatal follow-up and early bilirubin screening particularly for neonates re-presenting after the first week of life and targeted counselling on breastfeeding adequacy could help reduce the burden of severe neonatal jaundice and its complications in this region.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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ETHICAL APPROVAL AND INFORM CONSENT

The study was approved by the Department of Biochemistry, University of Malakand (Ref; No: UOM/BIOCHEM/23), and permission was obtained from the administration of Saidu Group of Teaching Hospitals, Swat, prior to data collection. Written informed consent was obtained from the parent/guardian of each enrolled neonate.

AUTHORS' CONTRIBUTION

Conceptualization and Methodology: [MUHAMMAD ZEESHAN]; Data Collection: [MUHAMMAD HASSAN, IZHAR UDDIN]; Written Original Draft: [ASHFAQ AHMAD]; Writing- Review and Editing: [ASAD KHAN, AKHTAR IQBAL]; Supervision and Co Supervision: [DR. NAUSHEEN NAZIR, DR. MEHREEN GHAYAS]. All authors have read and approved the final version of the manuscript.

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