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Original Research Paper

Neonatology

CORRELATION RED CELL DISTRIBUTION IN NEWBORN SEPSIS.

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KEYWORDS :

INTRODUCTION:

It is estimated that 3.9 million of the 10.8 million deaths in children annually world wide occur in the first 28 days of life. More than 96% of all neonatal deaths occur in developing countries.[1] The South Asian NMR was estimated at 23.2 deaths per 1000 live births, with India's NMR of 21.8 deaths per 1000 live births being the second highest in the region. In India, neonatal mortality accounts for 57% of national under-5 deaths. Thirty-three per cent of neonatal mortality in India is due to infectious disease.[2]

The clinical manifestations of neonatal sepsis are nonspecific and have varied clinical features. These varied features can be seen in other neonatal conditions thus making the diagnosis of neonatal sepsis difficult delay in diagnosis.[3] The gold standard for diagnosing sepsis is a positive result on culture from blood or another sterile body fluid, such as cerebrospinal fluid (CSF) or urine. It has been estimated that cases with a positive blood culture result represent less than 40% of all neonatal sepsis.[4]

Biomarkers for diagnosis of neonatal sepsis have been discovered that help in the early diagnosis of neonatal sepsis, before the onset of clinical manifestation so that early treatment of sepsis can be started and neonate can be properly managed.[5] Nearly 180 markers had been evaluated for neonatal sepsis, but none of these markers was sensitive or specific enough to be adopted as standard of care. Recently several studies showed that high RDW value can predicts diseases severity especially morbidity and mortality in patients admitted to ICU.[6] In this research article we intended to find correlation between RDW and sepsis in newborn population.

METHODOLOGY:

This is a retrospective case control study conducted at Niloufer institute of child health neonatal intensive care unit (NICU) from June 2018 to September 2018. Study population consisted of term and nearterm new-born delivered at niloufer hospital and less than 28 days of life for inclusion in to study. Study subjects were divided in to cases and controls. Cases were new-born hospitalized in the neonatal intensive care unit with symptoms suggesting neonatal sepsis including decrease in sucking activity, vomiting, changes in body temperature (temperature reading of >38.5°C or <36.0°C,), jaundice, sclerema, tachypnea, cyanosis, need for oxygen therapy, need for ventilation, hypotonia, convulsion, bradycardia, hypotension and impaired peripheral perfusion.

New-born who were healthy and no symptoms of clinical sepsis as determined among infants controlled during routine postnatal visits performed before discharge from the hospital were included in the control group. Infants with perinatal asphyxia, meconium aspiration syndrome, congenital malformations, and congenital infections were excluded from the study.

All newborns included in study Peripheral blood samples were drawn during their hospital stay and CBP, CRP and blood typing were determined. CBP and RDW were calculated by the automated haematology analyser. Serum CRP concentrations were measured using nephelometry technique.

Data were analyzed and expressed as mean and standard deviations (SD) for quantitative data.. Demographic and laboratory characteristics of the cases and controls sepsis were compared using t-test. The relation of RDW and CRP with other demographic and laboratory characteristics was evaluated. All analyses were performed using Statistical package for social science (SPSS version 18.0 . SPSS Inc., Chicago, IL, USA) and statistical significance was defined as $p<0.05$.

RESULTS :

In this study total 100 new borns were included during study period, 50 in each group i.e cases and controls. In total 100 subjects 62 were males (30 in cases and 32 in controls) and 38 were female babies (20 in cases and 18 in controls). Near term babies were 47 and term babies were 53 in study subjects. Basic demographic profile of the study populations are described in table 1.

TABLE 1: basic characteristics of study population

	CASES	CONTROL
Males	30	32
Females	20	18
Term	25	27
Preterm	24	23
Birth weight (mean +/- SD)	2245 +/- 523 grams	2374 +/- 652 grams
Maternal age (mean +/- SD)	23.4 +/- 3.6 years	24.3 +/- 4.1 years
Mode of delivery (NVD/LSCS)	32/18	35/15
Gestational age (mean +/- SD)	35. 7 +/- 2.4 weeks	35.3 +/- 3.2 weeks

As for the hematological variables compared in cases and control group hemoglobin, hematocrit, and lymphocytes were not statistically significant between two groups (p value > 0.05). wbc counts, netrophils and platelet counts were statistically significant between cases and control groups (p value < 0.001). red cell distribution levels in sepsis cases group was 19.9 +/- 2.43 and in control was 16.4 +/- 1.59 which was statistically significant with t value of 8.6 and p value of 0.0001 (p value < 0.05).

TABLE 2: hematological variables between cases and control groups

Variable	Cases	Control	T value	P value
Hemoglobin	15.75 +/- 3.52	16.1 +/- 3.49	-0.60	0.27
Hematocrit	49.6 +/- 10.7	49.2 +/- 10.4	0.15	0.43
Wbc count	8803 +/- 4510	13523 +/- 4916	-5.03	0.0001
Neutrophils	4579.8 +/- 3620	7768.8 +/- 5018	-3.64	0.0001
Lymphocytes	2878.8 +/- 1834	4465.2 +/- 2258	-1.45	0.07
Platelet count	115746 +/- 88420	216720 +/- 102560	-5.27	0.0001
RDW	19.9 +/- 2.43	16.4 +/- 1.59	8.6	0.0001

DISCUSSION:

The red cell distribution width (RDW) is a measurement derived from the red blood cell distribution curves generated on automated hematology analyzers and is an indicator of variation in RBC size within a blood sample. The RDW is used along with the indices (MCV, MCH, MCHC) to describe a population of RBCs. The RDW measures the *deviation* of the RBC width, not the actual width or size of individual cells. The more RDW is, the more uneven the RBC size is, and the higher the volume heterogeneity is.[6] Any disease involving red blood cell (RBC) destruction or production can increase variability in RBC size and lead to RDW elevation.[7]

Though the mechanism of increased RDW in sepsis is not known, higher RDW levels demonstrate its association with inflammatory processes.[8] In development of sepsis: First; inflammation may cause an increase of neuro-hormone and endocrine hormone in the body.[6] In studies performed, it has been detected that markers of inflammation including RDW-associated interleukin-6 (IL-6), tumour necrosis factor-alfa (TNF-α) and proinflammatory cytokines suppress maturation process of RBC and increase their half-lives with resultant rise in RDW levels.[8] These neurotransmitters can stimulate RBC proliferation through promoting the generation of erythropoietin (EPO) to result in RDW increase**.** Second inflammatory factors may affect bone marrow hemopoietic system and iron metabolism to cause RDW increase.[6]

In our study red cell distribution levels in sepsis cases group was 19.9 +/- 2.43 and in control was 16.4 +/- 1.59 which was statistically significant p value of < 0.05 indicating that red cell distribution values can be used early identification of sepsis. In study done by Cosar et al term and near-term new-borns with early onset sepsis RDW indices were higher than those of the control group ($p<0.001$).[8]

Recent studies also suggest that RDW is a useful biomarker of disease severity in critically ill patients and an increased RDW is an independent predictor of mortality in sepsis.[7,9] New studies found that RDW increase can be used as an important and independent predictive factor of the incidence of deaths caused by various diseases. Studies have shown RDW in newborns of the death group was significantly higher than that of the survival group, and the incidence of RDW increase was also higher than that of the survival group.[10] Measurement of RDW does not require extra blood samples, and hence it is a very feasible laboratory parameter, causing no unwarranted pricks in this vulnerable population with a very limited blood volume.[11]

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