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Serum Iron, Haemoglobin, & Serum Lipid Peroxidation in Neonates with Respiratory Disorders

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Abstract: Respiratory disorders are the most common cause for higher morbidity and mortality rate in India. Higher oxygen concentration of extrauterine existence causes increased erythrocyte lysis lead to release of iron in neonates. Iron is known to catalyze the formation of Reactive oxygen species. Involvement of hemoglobin and iron in oxygen-mediated reactions stimulate us to study the role of these in neonates.*Objectives* :i)To estimate the concentration of Haemoglobin, serum iron, serum lipid peroxidation in neonates with respiratory disorders; and compare those with that of healthy controls. ii)To study the role of these parameters in neonates with respiratory disorders.*Materials and Methods:* Present study includes 50 neonates suffering from respiratory disorders and 50 healthy neonates as controls. Samples collected from these were used for the estimation of haemoglobin, serum iron, and serum lipid peroxidation.*Observations:* Significantly ($p<0.001$) increased levels of serum iron and lipid peroxidation were observed in neonates with respiratory disorders when compared those with control values. These levels were found significantly ($p<0.001$) higher in preterm than full-term neonates. Concentration of haemoglobin showed no significant difference in both groups.*Conclusion*: Exacerbation of oxidative stress in neonates with respiratory disorders may be due to hypoxia induced free radical generation, higher oxidative tendency of HbF and elevated iron. Premature neonates are probably unprepared for extra uterine life in an oxygen rich environment and due to this they are more prone to oxidative insult. Thus this study reveals the pro-oxidant role of HbF and iron, which enhances the oxidative stress in respiratory disorder.
Key words : Oxidative stress, Iron, Haemoglobin.

Introduction

Birth asphyxia and Respiratory distress syndrome are the two most common causes for the increasing morbidity and mortality in neonates. Birth asphyxia is a consequence of hypoxia, hypercapnea and acidosis. If full recovery does not occur, the cerebral complications resulting due to Birth asphyxia are the most devastating and sometimes it leads to lifelong neurological impairments [1].Respiratory distress syndrome is the commonest cause of respiratory difficulty in the neonatal period and it is responsible for the worldwide deaths of many preterm infants each year [2].

High concentration of Fetal hemoglobin (HbF) in neonates compared to adult haemoglobin (HbA) is useful to carry more oxygen per unit volume than maternal blood [1], due to higher affinity of HbF for oxygen. Transport of oxygen by haemoglobin (Hb) involves iron in electron transport chain. S.M.H.Sadrzadeh during summarization of the present knowledge on the role of iron in human disorders, described toxic potential of free iron. Iron can catalyze formation of reactive oxygen species (ROS) such as hydroxyl and initiation or enhancement of lipid peroxidation (LPO), by reacting with hydrogen peroxide via Fenton reaction. In a same way iron can react with lipid peroxides to produce alkoxy and peroxy radicals, which lead to

propagation of lipid peroxidation. Free Hb, which is an iron compound, can also catalyze peroxidation of purified arachidonic acid and other polyunsaturated fatty acids within normal cell membrane in the presence of hydrogen peroxide and superoxide radical[3]. Most of the molecular oxygen is reduced by mitochondrial cytochrome oxidase in a electron reduction with the formation of H_2O.Partial or incomplete reduction leads to the generation of free radicals (F.R.) or ROS. Free radical denotes species that has one or more unpaired electron in its outer shell. Free radicals cause oxidative destruction of polyunsaturated fatty acid (PUFA), which is known as lipid peroxidation (LPO) particularly damaging because it proceeds as a self-perpetuating reaction. LPO has a significant role in the reduced lung compliance and pulmonary fibrosis [4]. In view of above circumstances following parameters were selected for the study. Concentration of Hb, serum iron and serum LPO [in terms of serum Malondialdehyde (MDA)] were measured in neonates with respiratory disorders and same were compared with those of healthy controls.

Materials and methods

Present work was conducted at the department of Biochemistry Government Medical College Miraj and Padmabhushan Vasantdada Patil General Hospital Sangli. Fifty newborns admitted in NICU with respiratory disorders were included in this study group, out of which 32 were full-term and 18 were preterm. Neonates with congenital anamolies, congenital heart diseases, multiple malformations, and inborn errors were excluded from present study. Birth weight, mode of delivery, sex, gestational age, Apgar score, and postnatal age of the cases were recorded.50 healthy neonates served as controls. Written informed consent was obtained from the parents before withdrawal of the blood. Institutional ethical committee approved the study. Biochemical analysis was carried out with sample of each subject, according to the protocol mentioned below-

Table-1:

Sr.No.	Estimation	Sample used	Method used
1	Haemoglobin	Whole blood	Cyanmethhemogloibin kit method
2	Serum Lipid peroxidation	Serum	K.Satoh [5]
3	Serum Iron	Serum	Randox Kit Method

Results

Concentration of Haemoglobin, Serum lipid peroxidation and iron were shown in Table-2 Serum lipid peroxidation in terms of MDA levels, was increased significantly ($p<0.001$) in neonates with respiratory disorders when compared with those of control values. These levels were found more significant ($p<0.001$) in preterms than those of full-terms.

Table-2: Concentration of Haemoglobin, Serum LPO & Iron in Neonates with Respiratory Disorders-

Group	LPO n moles of MDA per ml	Hb Gm%	Iron µmole/L
1) Control (50)	3.11±0.53	18.96± 2.04	31.44 ± 5.46
2) Whole group of neonates (50)	5.61±0.96	18.54 ± 0.92	45.99 ±6.08
2a)Preterm neonates (18)	6.06 ±0.76	18.97 ± 0.77	50.46 ±0.95
2b)Full term neonates(32)	4.72 ± 0.66	17.78 ± 0.64	38.06 ± 0.93

Serum iron concentration showed significant ($p<0.001$) increase in patient group than healthy controls. Preterm neonates exhibited significant increase ($p<0.001$) in serum iron than full-terms.Patient as well as control group showed no significant difference in the concentration of haemoglobin. Statistical analysis was done by using student 't' test.

Discussion

Present study showed significantly increased ($p<0.001$) concentration of serum LPO in neonates with respiratory disorders than that of control. More elevated levels of serum LPO were observed in premature infants than in full term neonates. Singh S.K. found elevated LPO in respiratory disorders [6] B.Sharda also observed oxidative stress in neonates with respiratory distress syndrome and bronchopulmonary dysplasia[7].Both findings were in line with our findings. In respiratory disorders hypoxia is associated with acidosis and at low pH superoxide radical protonate to form perhydroxyl radical [8], which is more potent reactive oxygen radical. Perhydroxyl radical is less polar than superoxide radical and ought to be able to cross biological membranes as effectively as can H_2O_2. Perhydroxyl radical can directly attack fatty acid and initiate lipid peroxidation to form peroxides. This mechanism may be the cause for increased LPO in neonates with respiratory disorders. Concentration of haemoglobin shows no significant difference between control and study group It is important to maintain normal hemoglobin concentration in infants who have acute pulmonary disease to optimize oxygen delivery [9]. Haemoglobin readily undergoes one electron oxidation and reduction, and it can act as source or sink for free radical. More rapidly oxidizing tendency of HbF generate greater amounts of free radicals like $O_2^{\cdot}$, H_2O_2,OH**.**[7] Serum iron levels were found to be significantly ($p<0.001$) elevated in neonates with respiratory disorders than in control group, at the same time premature infants showed significantly ($p<0.001$)

elevated serum iron levels than those of full term neonates. Low pH during hypoxia assists release of iron from transferrin. Ciccoli L.et al found both iron release and nonprotein bound iron ,were markedly higher in the hypoxic newborns compared to normoxic ones[10].These observations support our findings. Though iron is necessary for normal organ function, it is potentially toxic because it can create strong oxidative stress[9],it is capable of stimulating $OH^{\cdot}$ formation [11] and consequently may result into increased free radical generation i.e. increased oxidative stress.Premature neonates are probably unprepared for extra uterine life in an oxygen rich environment and due to this they are more prone to oxidative insult [12].

Higher oxidative tendency of HbF, enhances release of free iron resulting into increased free radical generation which adds more oxidative stress to already oxidatively stressed neonates in respiratory disorders. Thus this study reveals the pro-oxidant role of HbF and iron, which aggravates the lipid peroxidation and consequently enhances the oxidative stress in neonates with respiratory disorders.

Our work is in line with recent work done by others[7]. With our observations we would like to suggest the use of antioxidants as therapeutic drugs to minimize the increased oxidative stress in neonates, which will help to expand their lives.

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