INTRODUCTION

Respiratory distress syndrome (RDS), also known as hyaline membrane disease (HMD), occurs almost exclusively in premature infants. The incidence and severity of respiratory distress syndrome are related inversely to the gestational age of the newborn infant ***(Pramanik ,2009).***

Respiratory Distress Syndrome (RDS) is one of the most common causes of morbidity in preterm neonates. It occurs worldwide with a slight male predominance ***(Pramanik ,2009).***

The preterm infant who has RDS has low amounts of surfactant that contains a lower percent of disaturated phosphatidylcholine species, less phosphatidylglycerol, and less of all the surfactant proteins than surfactant from a mature lung ***(Nkadi et al., 2009).***

Surfactant is synthesized and secreted by Type II alveolar epithelial cells, also called pneumocytes, which differentiate between 24 and 34 weeks of gestation in the human. It is made up of 70% to 80% phospholipids, approximately 10% protein and 10% neutral lipids, mainly cholesterol. The primary surface-active material found in surfactant is the phospholipid, dipalmitoylphosphatidylcholine (DPPC), while the surfactant proteins are SP-A, SP-B, SP-C and SP-D . Surfactant increases surface pressure while lowering surface tension. High surface pressure resists a decrease in alveolar surface area, while low surface tension stabilizes the lung by decreasing the pressure gradient across the alveolar lining layer ***(Nkadi et al., 2009***) .

Cholesterol is the second most abundant lipid component of pulmonary Surfactant ***(Daniles et al., 2002).***

Cholesterol was found to represent over 50% of the neutral lipid of both the total surfactant and the lamellar body fractions ***(Hass et al., 1979).***

De novo synthesis of cholesterol from [1-^{14}C]acetate accounted for only 1% of the surfactant cholesterol, the remainder being derived from exogenous cholesterol supplied as serum lipoproteins ***(Hass et al., 1979).***

Lipoprotein [1,2-$^{3}H_2$] cholesterol was incorporated into the lamellar body and extracellular surfactant fractions ***(Hass et al., 1979) .***

Lung cholesterol may be subjected to regulation by both low density lipoprotein LDL and high density lipoprotein HDL(***Hass et al., 1980***).

LDL and HDL stimulate alveolar type II cells to secrete surfactant (***Pian et al., 1997).***

Lipid metabolism is important in lung development, with indications that TG from very low-density lipoprotein (VLDL) is essential for surfactant synthesis ***(Yonezawa et al.,2009).***

In late gestation maternal serum lipids increase it is considered a maternal adaptation maintain stable fuel distribution to the fetus. Shortage of lipids in maternal circulation affect fetal growth and mortality ***(Sekhavat et al., 2008) .***

Maternal lipoproteins, such as VLDL, provide the free fatty acid substrate required for fetal surfactant synthesis in vivo ***(Ryan et al., 2002).*** .

Inadequate total fatty acid supplies in utero could interfere with normal fetal growth and maturation, leading to

development of neonatal RDS as one manifestation of risk for postnatal morbidity and mortality (***Lane et al.,2002).***

Lipid metabolism has an important role in fetal development during the late stage of gestation, including growth and fat accretion in utero, increasing amniotic fluid lecithin levels with maturation of pulmonary function and changes in the levels of minor phospholipids in amniotic fluid ***(Gunes et al., 2007).***

Total maternal serum cholesterol <10th population percentile was strongly associated with preterm delivery ***(Edison et al., 2007)***.

Maternal weight gain during pregnancy correlates significantly with newborn weight (***Sekhavat et al., 2008).***

AIM OF THE WORK

The aim of this study was to compare the maternal and cord lipid profiles of preterm infants with respiratory distress syndrome and a control group without respiratory distress syndrome and to assess the effect of maternal nutritional status and lipid profile on the cord blood lipid profile of preterm infants.