Introduction

Neonatal respiratory distress syndrome (RDS) is a condition of increasing respiratory distress, commencing at, or shortly after, birth and increasing in severity until progressive resolution occurs among the survivors. It is due to insufficiency of pulmonary surfactant and is mainly confined to preterm infants ***(Field et al, 1992).***

The incidence and severity of RDS, also known as hyaline membrane disease (HMD), are related inversely to the gestational age of the infant ***(Pramanik, 2006).***

RDS is a developmental disorder rather than a disease process per se, and it is a common cause of morbidity and mortality associated with premature delivery ***(Pryhuber et al., 2005).***

A study by the National Institute of Child Health and Human Development (NICHD) reported that rates of RDS were 71% in those 501-750g, 54 % in those 751-1000g, 36 % in those 1001-1250g, and 22 % in those 1251-1500g ***(Pramanik, 2006).***

RDS is manifested by respiratory distress (cyanosis, tachypnea, grunting, and recession) and respiratory failure is diagnosed by blood gas analysis. Edema is frequently seen on

the second day due to fluid retention and capillary leak. The diagnosis can be confirmed by an x ray film showing ground glass appearance and air bronchograms, although these radiological features are not pathognomonic of RDS ***(Field et al., 1992).***

Cholesterol was found to represent over 50% of the neutral lipid of both the total surfactant and the lamellar body fractions and 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 ***(Gunes et al., 2007)***

The main lipid in plasma are cholesterol and triglyceride, they required to be transported in plasma encapsulated in shell of phospholipids and apolipoproteins forming lipoproteins ***(Dowhan 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. ***Lane et al, (2002)*** suggested that deficiency or reduced transport of essential and/or long-chain polyunsaturated fatty acids could inhibit normal fetal growth and maturation, one effect of which would be delayed development of the fetal lungs.