INTRODUCTION

Neonatal respiratory distress syndrome (RDS) also known as hyaline membrane disease, is a condition of increasing respiratory distress, commencing at, or shortly after, birth and increases in severity until progressive resolution among the survivors, usually between the 2nd and 4th day ***(Fanaroff et al., 2007)***.

Respiratory distress syndrome is the most common cause of respiratory distress in premature infants, correlating with structural and functional lung immaturity ***(Hermansen and Lorah, 2007)***. It is due at least in part, to insufficiency of pulmonary surfactant ***(Bedetti et al., 2006)***. Its incidence is inversely related to gestational age and birth weight. It occurs in 60-80% of infants less than 28 wk of gestational age, in 15-30% of those between 32 and 36 wk, in about 5% beyond 37 wk, and rarely at term ***(Dudell and Stoll, 2007)***.

Enormous efforts have been made to understand the pathophysiology of RDS and to optimize the care of those infants, which has led to improvement in the morbidity and mortality. The mortality rate of RDS decreased by approximately 50% during the last decade with the advancement of surfactant therapy ***(Copetti and Cattarossi, 2007)***.

Carnitine (L-3-hydroxy-4-N-trimethylaminobutyrate) is a small, water soluble molecule that plays a key role in transporting long chain fatty acids across the barrier of the inner mitochondrial membrane for β-oxidation via a carnitine acyltransferase enzyme system ***(Vaz and Wanders, 2002)***. Carnitine is essential for the newborn infant for energy utilization, ketogenesis, thermal regulation and growth ***(Scaglia and longo, 1999)***.

Pulmonary surfactant production is an important process in fetal lung maturation. Antenatal carnitine administration has been shown to be effective in inducing pulmonary surfactant production and lung maturation in both fetal rats and humans (***Lohninger et al., 1996)***.

As carnitine is an integral component of the membrane phospholipid fatty acid turnover in human cells, it is possible that carnitine causes lung maturation via membrane phospholipid repair activity ***(Arenas et al., 1998)***.