INTRODUCTION

Respiratory distress (RD) is the most common neonatal emergency and the main cause of admission to Neonatal Intensive Care Units (NICU). Disorder of respiration in the newborn infant can be categorized as either central nervous system failure or peripheral respiratory difficulty. Hyaline membrane disease (HMD) is by far the most common cause (more than 50% of cases) followed by neonatal pneumonia, transient tachypnea of newborn (TTN) and meconium aspiration syndrome (MAS) **(Stoll and Kliegman, 2004).**

Endothelin is a novel and potent endothelium derived vasoconstrictive peptide present in human plasma ***(Kue et al., 2001).*** It is a peptide of 21 amino acids in chain with two disulfide bonds with three distinct isoforms: ET-1, ET- 2, and ET-3 ***(Benjamin et al., 2005).***

Endothelin causes isolated contraction of puylmonary veins, vascular smooth muscle mitogenesis, myocardial cell hypertrophy, positive inotropic and chronotropic effects, bronchoconstriction, mucous secretion, cellular proliferation, and inflammatory reactions **(Goraca, 2002). (Perreault and Coceani, 2003)**, reported that ET-1 has a potent pulmonary vasoconstrictor effect.

Experimental studies have suggested that ET-1 plays an important role in pulmonary vascular reactivity in neonatal respiratory distress syndrome **(Vroomen et al., 2001).** There is also an elevation of ET-1 in tracheal aspirates from these infants **(Figueras-Aloy *et al.*, 2003).**

In addition to being a potent endogenous constrictor of smooth muscles, ET-1 also acts as a chemoattractant for fibroblasts, stimulates

proliferation of airway epithelial cells and fibroblasts and activates human alveolar macrophages which are capable of releasing ET **(Murlas *et al*., 1995).**

Ambalavanant and Novak, 2003, speculated that peptide growth factors including endothelin-1 may play a role in the development of bronchopumonary dysplasia. Endothelin-1 synthesized by and secreted from tracheal epithelial cells and/or alveolar macrophages has a priming effect on alveolar macrophages to produce super oxide anion, thus possibly contributing to the development of bronchopulmonary dysplasia **(Kojima *et al*., 1996).**

Yigit *et al.,* 2002, reported higher plasma levels of endothelin-1 in infants with meconium stained amniotic fluid versus the control group.

Normally ET-1 level is high in the umbilical cord and a slower decline of endothelin-1 from birth to 40 h of life was observed in newborns with respiratory distress syndrome when compared to controls **(Benijamin *et al*., 2005).**

In vitro, ET-1 has been shown to stimulate the proliferation of airway epithelial cells; it also acts a secretagogue for surfactant from type II alveolar cells, which posses a receptor to ET-1 **(Sen *et al*., 1996).**

The possible role of ET-1 in the early stages of RD has been evaluated. There is evidence that improved pulmonary status may be attributed to the effects of ET-1 on surfactant secretion or development of airway epithelium during the early course of such disease **(Amlersson *et al.,* 1997).**

Hence, assay of plasma ET-1 can be of critical importance in decision-making regarding prognosis and therapy during the early course of respiratory distress.