INTRODUCTION

Respiratory distress syndrome (RDS) is a common sequela of preterm birth ***(Gilbert, 2006).*** Respiratory distress syndrome (RDS) secondary to surfactant deficiency is a common cause of morbidity and mortality in premature infants ***(Tsao et al., 2005).***

The pathogenesis of RDS is unknown, so The association between RDS and many biological markers has been studied; one of these markers is endothelin-1 ***(Benjamin et al., 2005).***

Endothelin-1 (ET-1) is a vasoactive mediator that is synthesized by the pulmonary endothelium and during the perinatal period it plays an important role to reduce pulmonary vascular resistance (PVR) in order to facilitate the clearance of lung fluid ***(Wojciak-Stothard and Haworth, 2006).***

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

Experimental research on newborn lambs has shown that endothelin-1 plasma concentration increases in the early phase of pulmonary hypertension during respiratory distress syndrome ***(de Vroomen et al., 2001).***

AIM OF WORK

This study aims to declare the possible role of endothelin-1 in the pathogenesis of RDS in preterm infants and to detect its clinical and biochemical associations in such situations