SUMMARY AND CONCLUSIONS

Neonatal respiratory distress syndrome (RDS) is a major clinical problem that presents at or shortly after birth as a result of a deficiency of pulmonary surfactant. Neonatal RDS affects approximately 1% of all live births; however, not all infants are at equal risk.

Many mediators were suggested to contribute to the pathogenesis of RDS. Among these, endothelin-1 was proved to play role in pulmonary vascular reactivity in newborns leading to increased pulmonary vascular resistance in preterm newborn infants with respiratory distress syndrome.

The aim of the present study was to evaluate and 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. Twenty preterm neonates with respiratory distress syndrome were prospectively recruited. They included 13 males (65.0 %) and 7 females (35.0 %). They had mean gestational age of 32.35 ± 2.8 weeks and mean weight of 1.61 ± 0.43 kg. In addition, there were 10 preterm infants who were free from RDS and served as controls.

The demographic characteristics of the studied patients showed that they included higher frequency of males than females. The

gestational age of our patients ranged from 26.0 - 36.0 weeks with the majority of patients born at or before 34 weeks (70.0 %). Moreover, patients spent less weeks in utero when compared with controls. In addition, the majority of our patients were delivered by caesarean section (60.0 %); a frequency which is higher than controls despite lacking statistical significance (p=0.60).

Importantly, there was a significantly higher ET-1 level in patients (0.850 ± 0.131 pg/Ml) when compared with controls (0.737 ± 0.143 pg/ml) (p=0.04) and the statistically significant decline in ET-1 levels in both groups from the levels of cord sample in patients and controls (1.133 ± 0.138 and 1.086 ± 0.165 pg/mL respectively) to the levels of the blood sample (0.850 ± 0.131 and 0.737 ± 0.143 respectively). However, there were no statistically significant difference between groups regarding ET-1 levels in the cord sample despite the fact that patients had higher ET-1 levels (1.133 ± 0.138 pg/mL) when compared with controls (1.086 ± 0.165 pg/mL).

The present study also found that patients with RDS generally worse organ functions when compared with controls but the difference was statistically significant only regarding RBCs count (4.18 ± 0.58 versus 4.84 ± 0.86 × 10^6) (p=2.46) and Hb (11.87 ± 3.0 versus 14.81 ± 2.92) (p=0.016).

Comparative analysis of ET-1 levels in respect to demographic, clinical and laboratory parameters didn't reveal significant differences while correlative study had shown inverse correlations between cord ET-1 and WBCs count and blood ET-1 and Hb concentration and RBCs count.

Conclusively, ET-1 is suggested to contribute to the pathogenesis of RDS. Lager studies are recommended to declare the possible mechanisms involved in this process and to detect the other associations between ET-1 and other clinical characteristics and biochemical findings factors.