SUMMARY

Carnitine, a quaternary amine synthesized from the amino acids lysine and methionine, is essential in β-oxidation via transporting long-chain fatty acids across inner mitochondrial membrane. As fatty acid oxidation is an important energy-providing pathway, carnitine plays a crucial role in the energy supply of tissues during fetal life and in the neonatal period.

Beside its role in transporting long-chain fatty acids, carnitine represents an integral component of the membrane phospholipids fatty acid turnover in human cells, a process essential for maturation of tissues in the developing fetus.

Neonatal period in preterm infants represents a great challenge for the developing fetus, with a variety of mortalities and morbidities, where RDS represents the single most important cause of mortality and morbidity.

Hence, the aim of this study was to assess plasma free carnitine levels, both neonatal and maternal, in preterm infants who developed respiratory distress syndrome and those who did not to evaluate its role in the pathogenesis of respiratory distress syndrome (RDS).

This study was conducted on 30 preterm infants and their mothers who were recruited from the delivery room, Obstetrics and Gynecology Department, Benha University and followed up in neonatal intensive care unit, Benha University in the period from July, 2008 to July, 2009.

These preterm infants were ≤ **34** weeks of gestation. They were divided into two groups: ***Group I (control):*** included ten preterm infants who did not develop respiratory distress syndrome. They were **3** males and **7** females with a mean gestational age **31±2.1** weeks and birth weight **1.285±0.311** Kg. ***Group II (cases):*** included twenty preterm infants who developed respiratory distress syndrome within the first 6 hours of life diagnosed on the basis of clinical and radiological findings. They were **16** males and **4** females with a mean gestational age **30.6±1.9** weeks and birth weight **1.246±0.248** Kg. In our study group II was classified into two categories divided by gestational age as into group IIa **(33-34**wks) and group IIb (**28-32**wks), and by birth weight into group IIa **(>1500**gms) and group IIb **(<1500**gms). Mothers of these preterm infants in both groups were also included.

Samples for assessing L-carnitine was withdrawn from mothers either few hours before delivery or on the first day of delivery, while neonatal peripheral or umbilical cord blood samples were drawn in the first 2 hours of life before the full-blown clinical symptoms and signs of RDS developed. Preterm infants who developed RDS in the first 6 hours of life were enrolled in the study group (group II) while preterm infants who did not develop RDS served as the control group (group I). In group II (cases), another umbilical cord or peripheral venous blood samples were obtained after the development of the full picture of RDS. In all samples plasma free carnitine levels were determined by U.V spectrophotometer technique.

The results of this study revealed that the initial neonatal plasma free carnitine levels (1^{st} sample) were significantly lower in preterm infants who developed RDS (group II) than in the control group (group I). Furthermore, the second samples drown after the development of the full-

blown picture of RDS showed a significant decrease in free carnitine levels in the RDS group. Also, there was a highly significant negative correlation between degree of RDS and neonatal plasma free carnitine level where the lowest levels were detected in preterm infants with severe grade of RDS. These findings could be attributed mainly to increased transport of free carnitine to lung tissues for surfactant synthesis.

On the other hand, this study revealed that maternal free carnitine levels were similar in both groups. As maternal deficiency of free carnitine was excluded, it could be possible that maternal-fetal transfer of carnitine was impaired or reduced in preterm infants who later developed RDS.

The effect of gestational age, birth weight, mode of delivery and gender on maternal and neonatal plasma free carnitine level was studied. Maternal and neonatal plasma carnitine levels did not vary in relation to gestational age, birth weight, gender or mode of delivery.

CONCLUSION

In view of this study, the lower plasma free carnitine levels in preterm infants with RDS could be due to:

- Increased carnitine consumption for surfactant synthesis in the immature lung tissue as L-carnitine is involved in the pathway of membrane phospholipids fatty acid turnover, an important tool for remodeling the phosphatidylcholine (PC) fatty acid composition hence dipalmitoylphosphatidylcholine (DPPC) synthesis.

- Presence of a structural or functional placental pathology being responsible for the impaired maternal-fetal carnitine transport. As a result, low plasma carnitine levels may lead to decreased carnitine transport and surfactant synthesis in the immature lung tissue. So, lower neonatal plasma carnitine levels could be a causal factor in the pathogenesis of RDS in preterm infants.