PULMONARY SURFACTANT SYSTEM IN RELATION TO UNDERNUTRITION & RESPIRATORY INFECTIONS : ARTIFICIAL SURFACTANT THERAPY

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Studies of the past few years have shown a significant association among spontaneous abortion, stillbirth, prematurity. perinatal morbidity and mortality in pregnancies in which the fetuses or placentas were infected with Ureaplasma urealyticum, and in few cases with Mycoplasma hominis. Bacterial infection of the amniotic fluid and the placenta may trigger premature labor during pregnancy, leading to premature delivery. One possible mechanism in the initiation of premature labor may be the effect of microbial phospholipase hydrolysis of placental membrane phospholipids to produce an increase in the amount of free arachidonic acid and consequently an increase in the synthesis of prostaglandins. However, no clear picture has emerged regarding the exact mechanism of action of microbial phospholipases in the infectious processes in the premature lung.

The human lung is a very heterogenous organ. It contains about 40 different cell types. Present evidence indicate that type (II) alveolar epthelial cell is the site of surfactant production. Pulmonary Surfactant consist of about 90% Lipid and 10% protein. The lipid is largely phospholipid of which phosphatidylcholine(PC) account for 80-90% by weight. The major surface active component in surfactant is dipalimitoyl- PC. The other major components of surfactant are phosphatidylclycerol,PG(10%),phosphatidylserine phosphatidylethanolamine,cardiolipin,cholesterol and free fatty acids.

Mycoplasmas, especially Ureaplasmas have been shown to colonize the lower respiratory tract of newborn infants and in the absence of other detected organisms does produce pneumonia. It is important to note that early onset neonatal pneumonia is clinically and radiologically not distinguishable from surfactant deficiency syndrome.

Due to these reasons we have hypothesized that pulmonary surfactant could be degraded by mycoplasmal infection resulting in loss of drastic changes in air-tissue interface of the lung, and also bring about major changes in surfactant composition and concentrations of individual phospholipids. Results of in vitro experiments indicate ureaplasmal phospholipases are able to hydrolyse the major phopholipids of surfactant. How undernutrition may be related to this problem as a major contributing factor will be discussed.