Introduction
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Aim of the work

Introduction

Zinc and Iron are trace elements known to be essential for normal growth and development in human. The metabolic function of them are based largely on their presence as an essential component of many metalloenzymes involved in all aspects of metabolism. As regard zinc, it enters in the constituent of several enzymes as carbonic anhydrase enzyme in erythrocyte essential for CO_2 exchange and also in carboxy peptidase in intestine for hydrolysis of protein and also dehydrogenase enzyme of the liver. It is found in liver, muscles, bones, red and white blood cells. Deficiency of zinc produces: dwarfism, iron deficiency anemia, hepato splenomegally, hyperpigmentation, hypogonadism, acrodermatitis enterepathica, depression of immunocompetence and poor wound healing. Zinc in excess prodcues gastrointestinal upset, copper deficiency and decreased high density lipoprotein. **(Hamil, et al., 1979)**.

As regard zinc, manifestations of altered humoral response include distorted serum immunoglobulin profile resulting from prenatal and postnatal zinc deficiency. Dietary zinc deficiency in neonate also impairs antibody production to red blood cells. Repletion with zinc results in normalisation of IgM pool response and an elevation of IgG response (Bach et al., 1975).

Further evidence for role of zinc in immunity comes from the discovery that inborn error of human metabolism, acrodermatitis enteropathica, was caused by defective absorption of zinc. The major causes of death in this disease are all linked to immunodeficiency which is prevented by zinc supplementation **(Moynhan et al., 1973)**.

Zinc is important for neutrophil chemotaxis. The nutrophil function as chemotaxis was impaired in malnourised children and is reversible by zinc

supplementation. Zinc is important for regulation of various functions of macrophages. During acute inflammation or endotoxemia, leucocytes release a mediator that reduce the plasma zinc concentration as well as mediating a number of other changes observed in inflammation **(Chandra, 1984)**.

Zinc is known to play a central role in the immune system, and zinc-deficient persons experience increased susceptibility to a variety of pathogens. The immunologic mechanisms whereby zinc modulates increased susceptibility to infection have been studied for several decades. It is clear that zinc affects multiple aspects of the immune system, from the barrier of the skin to gene regulation within lymphocytes. Zinc is crucial for normal development and function of cells mediating nonspecific immunity such as neutrophils and natural killer cells. Zinc deficiency also affects development of acquired immunity by preventing both the outgrowth and certain functions of T lymphocytes such as activation of Th 1 cytokine production, and B lymphocyte help. Likewise, B lymphocyte, immunoglobulin G is compromised. The macrophage, a pivotal cell in many immunologic functions, is adversely affected by zinc deficiency, which can dysregulate intracelluar killing, cytokine production, and phagocytosis, the effects of zinc on these key immunologic mediators is rooted in the myriad roles for zinc in basic cellular functions such as DNA replication, RNA transcription, cell division, and cell activation. Apoptosis is potentiatied by zinc deficiency. Zinc also functions as an antioxidant and can stabilize membrances. This system and attempts to provide a biological basis for the altered host resistance to infections observed during zinc deficiency and supplementation. **(Shankar – AH; Prasad – AS, 1998)**.

As regard iron, it enters in the structure of hemoglobin and myoglobin for O2 and CO2 transport, oxidative enzymes, cytochrome C and catalase. Deficiency of iron produces hypochromic microcytic anemia, growth failure and behavioral changes. Excess iron produces hemosidrosis. **(Hamil, et al., 1979)**.

The body of newborn infant contain about 0.5 gm iron. Infants breast fed exclusivly should receive iron supplement from 4 months of age. In low birth weight infants or those with perinatal blood loss, stored iron may be depleted earlier, and dietary sources become of paramount importance. Inadequate nutrition of zinc and iron alters immunocompetence in humans and experimental animals. For each of these minerals, deficient status leads to increased susceptibility to infection. Specific components of the immune reponse may be altered in variety of patients and models. However, specific functions for these minerals in immunity have not yet been identified. For zinc and iron, the importance of adequate nutrition in maintaining immunocompetency can not be understated **(Hoffman R, et al, 1990)**.

The earliest research in iron and immunity showed that iron deficiency is associated with increased infectious diseases in infancy. Infants given iron supplements had 50% fewer respiratory and gastroentestinal infections than infants given no iron. Either fortified formula or evaporated milk formula to term infants of lower socioeconomic status during their first 18 months of life, the incidence of both iron deficiency anemia and respiratory infections was highest in this group fed with evaporated milk with lowered iron content. Anatomical and structural changes in immunologically important tissues have been found in experimental iron deficiency. In neonates, splenic development is retarded in iron deficiency. **(Prasad A S, et al, 1963)**.

The effects of zinc and iron, on the immune system are reviewed. Among the essential trace elements in humans, zinc and iron, are essential for the integrity and optimum function of the immunity. Although each element has different function on the immune system, the deficiencies in each of these elements mainly causes the dysfunction of cell-mediated immunity. Deficiencies do not significantly effect the B cell function. These immunological abnormalities can be

improved by their supplementation. Therefore, a proper balance of them is essential for maintenance of immunocompetence. **(Kodama-H, 1996).**

Aim of work :

Study of iron and zinc status and its reflection on some immunologic functions in Septicemic new borns.