Introduction

Bacterial infection continues to be the major cause of morbidity and mortality in the newborns. Because the prognosis for sepsis largely depends on early identification and treatment, these newborns are subjected to extensive diagnostic evaluation and empirical systemic antibiotic treatment. The definitive diagnosis of septicemia is made by a positive blood culture, which requires a minimum of 48 – 72 hours, yields a positive result in only 30 – 70 % of cases, and may not always be available in peripheral health centers (***Manucha et al., 2002***).

The most important neonatal factors predisposing to infection are prematurity and low birth weight. Preterm infants have a 3–10 folds higher incidence of infection than full–term normal birth weight infants. Possible explanations include 1) maternal genital tract infection is considered to be an important cause of preterm labor, with an increased risk of vertical transmission to the newborn. 2) The frequency of intra amniotic infection is inversely related to gestational age. 3) Premature infants have documented immune dysfunction. 4) Premature infants often require prolonged intra venous access, endotracheal intubations, or other invasive procedures that provide a portal of entry of infection or impair body barriers and clearance mechanisms (***Barbara and Stoli, 2008***).

The principal pathogens involved in neonatal sepsis have tended to change with time. Primary sepsis must be contrasted with nosocomial sepsis. The agents associated with primary sepsis are usually the vaginal flora. Most centers report group B streptococci (GBS) as the commonest one, followed by Gram-negative enteric organisms, especially Escherichia coli. Other pathogens include Listeria monocytogenes, Staphylococcus, other streptococci (including the enterococci) anaerobes, and Haemophilus

influenzae. In addition, many unusual organisms are documented in primary neonatal sepsis, especially in premature infants. The flora causing nosocomial sepsis vary in each nursery. Staphylococci (especially Staphylococcus epidermidis) gram-negative rods (including Pseudomonas, Klebsiella, Serratia, and Proteus) and fungal organisms predominate ***(Garcia J, 2000).***

Sepsis and endotoxin activate monocytes, macrophages, lymphocytes, fibroblasts, and endothelial cells that produce and secrete IL-1, TNF-α, interferon, IL-6, IL-8, and other proinflammatory cytokines. IL-6 stimulated by TNF–α, IL-1, and endotoxins of viral and bacterial infections, act as T-cell activation indicators, induce antibody secretion by human B-cells, cause differentiation of cytotoxic T-cells, and have the ability to inhibit TNF-α production (***Kocabas et al., 2007***).

In the past few decades, it has been observed that several mediators of inflammation tend to become elevated during sepsis. The concentrations of some proinflammatory cytokines, especially TNF-α, IL-6 and IL-8, in the systemic circulation were reported to increase in severe infections and septic shock. Serum IL-6, IL-8, and TNF- α levels were all higher in septic than in nonseptic newborns (***Kocabas et al., 2007***).

Interleukin-8 is a cytokine that has a role in the release, activation, and chemotaxis of neutrophils. Serum IL-8 level has been reported to increase in neonatal sepsis and has a sensitivity of about 80-90 % and a specificity of about 76-100 % (***Franz et al., 2001***).

Aim of the Work

The aim of this work is to determine the possible role and value of serum interleukin-8 (IL-8) & tumor necrosis factor-α (TNF – α) in the diagnosis of neonatal sepsis.