Introduction

Neonatal sepsis is a term that has been used to describe the systemic response to infection in newborn infants. It is estimated that about 5 million neonates die every year in low-income countries. Infections contributes to approximately 30 to 40% of neonatal deaths in these countries ***(Escobar, 2003).*** Neonatal septicemia constitutes an important cause of morbidity and mortality among neonates. Early diagnosis and management of neonatal septicemia can bring down the morbidity and mortality but in fact, there is no obvious predictor of mortality rates in neonatal septicemia ***(Aronis et al., 1999).***

Protein C is a vitamin K-dependent protein which exists in bovine plasma as a precursor of serine protease. Activated protein C (APC) is an endogenous anticoagulant which is important for regulation of blood coagulation ***(Levi et al., 2000).***

APC effectively down-regulates the coagulation processes by selectively degrading the activated coagulation cofactors: factor Va and factor VIIIa. By inhibiting these 2 rate-limiting steps of coagulation cascade, APC limits thrombin formation and thus, reduces the risk of multiogram dysfunction ***(Hancock et al., 1995).*** Being intrinsically deficient, neonates are likely to be especially vulnerable to the effects of low APC values. The role of APC deficiency in predicting mortality among severely septicemic newborns is not clearly understood. Previous studies have defined severe septicemia in neonates as sepsis with systemic inflammatory response syndrome (SIRS) and organ dysfunction ***(Barton et al., 2004).***

Aim of the work:

We aimed in our study to detect the level of activated protein C in septicemic neonates and to evaluate the role of activated protein C in predicting mortality in neonatal septicemia.