Introduction

Neonatal jaundice is the most common condition that requires medical attention in newborns. Jaundice is yellowish discoloration of the skin, mucous membrane and sclera which is the result of accumulation of unconjugated bilirubin in blood. In most infants, unconjugated hyperbilirubinemia reflects a normal transitional phenomenon. However, in some infants, serum bilirubin levels may excessively rise to degree that may cause neurotoxicity to brain and can cause death in newborns and lifelong neurologic sequelae in infants who survive as kernicterus ***(Hansen, 2007)*** .

Therefore, an early identification of newborn infants at risk of developing severe hyperbilirubinemia, along with a possible bilirubin induced neurological dysfunction, and the prediction of a possible need of phototherapy continue to be a problem in neonatology ***(Knüpfer et al., 2005)***.

Increased heme catabolism is an important mechanism responsible for hyperbilirubinemia in the first days after birth ***(Maisels and Kring, 2006)***.

Hemoglobin released from erythrocytes into the circulation by intravascular hemolysis binds immediately with haptoglobin, a serum glycoprotein, and forms a stable hemoglobin-haptoglobin (Hb-Hp) complex ***(Delanghe and Langlois, 2001)***.

Hb binding by haptoglobin is thought to be important in the rapid hepatic clearance of hemoglobin from the plasma and in the inhibition of glomerular filtration of hemoglobin. The presence of specific receptors on the liver parenchymal cells that recognize and endocytose the Hp-Hb complex has led to a widely held belief that the major function of Hb binding by Hp is to target plasma Hb for rapid clearance and degradation in the liver ***(Okuda and Oshiro et al.,1992).***

Haptoglobin has been proposed to be more useful than the other protein markers to assess severity of hemolysis ***(Delanghe and Langlois., 2001).***