Introduction

Neonatal jaundice affects 60% of full-term infants and 80% of preterm infants in the first 3 days after birth. Although transient, the condition accounts for up to 75% of hospital readmissions in the first week after birth **(Melton,1999) .**

Jaundice usually becomes apparent when total bilirubin levels exceed 5 mg/dL. Neonatal jaundice progresses in a cephalopedal direction. However, estimates of total serum bilirubin levels based on visual evaluation are often inaccurate because of interobserver variability, especially in dark-skinned infants **(Melton,1999) .**

Hyperbilirubinemia in newborns is primarily due to immaturity of the liver enzyme system. Jaundice can be classified as physiologic or nonphysiologic according to post delivery timing of onset, clinical course, resolution, rate of bilirubin increases, and total serum bilirubin levels**(Melton,1999) .**

The probability of "subtle" or minor neurologic abnormalities in relation to Hyperbilirubinemia is of prime clinical importance **(Vinod K .2001).**

Neonatal indirect Hyperbilirubinemia is a common problem in the neonatal period. Neurological disturbances such as athetoid-dystonic cerebral palsy, hearing loss, gaze palsy, developmental delay, and impairment of intelligence due to kernicterus are serious problems . Although neonatal indirect Hyperbilirubinemia (NIH) has long been a known entity, its management and outcome continue to be problematic .**(Yilmaz et al.,2001)**

Bilirubin is a heme catabolite known for its potential toxicity to the neonatal central nervous system (CNS) Although extreme hyperbilirubinemia (>30 mg/dL) has been associated with adverse neurodevelopmental consequences in term newborns without hemolytic disease, lower bilirubin concentrations (<8 mg/dL) have not been associated with significant alterations of CNS function. (**Roberto et al., 2002).**

Moderate Hyperbilirubinemia (13-25mg/dL), is associated with an increase in minor neurologic dysfunction throughout the first year of life. The dose-response relationship between the degree of Hyperbilirubinemia and the severity of the minor neurologic dysfunction condition indicates that total serum bilirubin levels above 18.6 mg/dL should be avoided **(Soorani et al ., 2001).**

newborns with serum bilirubin levels between 20-24 mg/dl are at a greater risk of neurological abnormalities **(Yilmaz et al.,2001).**

Since kernicterus is a serious entity with severe neurological sequelae in survivors, criteria for treatment requires a high level of sensitivity. The critical level for intervention should be at or below the threshold level for bilirubin neurotoxicity. Since the earlier reports, the "20 mg/dl level" of serum bilirubin has been generally accepted as the limit value of exchange transfusions in full-term newborns with hemolytic disease . Despite traditional treatment based on vigintophobia (fear of 20), recent studies have proposed a new criterion for exchange transfusion; that is, bilirubin levels of 25 mg/dl in nonhemolytic and otherwise well babies **(Yilmaz et al.,2001).**

The most well-established toxic effects of bilirubin on the central nervous system involve the basal ganglia and auditory nuclei. Cognitive

function is generally relatively spared, even among children with kernicterus. To our knowledge, deficits in social interaction among children with kernicterus, beyond those attributable to their motor and auditory disabilities, have not been reported. There has been concern that shorter postpartum stays and less aggressive jaundice treatment in the past several years might have resulted in increased incidences of extreme hyperbilirubinemia and its sequelae. **(Lisa A et al .,2005) .**

Kernicterus, a preventable brain injury resulting from severe neonatal jaundice. Newborn jaundice, a usually benign condition that typically resolves with supervision and appropriate nutritional intake, can progress to severe hyperbilirubinemia in 8–10% of healthy newborn infants. Severe hyperbilirubinemia may need treatment with phototherapy. Some newborns discharged as healthy have developed severe hyperbilirubinemia after discharge and succumbed to serious and often irreversible posticteric sequelae (**Vinod K , 2004).**

The common insult in all cases of bilirubin-induced neurologic dysfunction (BIND) results from a total serum bilirubin (TSB) concentration that exceeds the infant's neuroprotective defenses and leads to neuronal injury, primarily in the basal ganglia, central and peripheral auditory pathways, hippocampus, diencephalon, subthalamic nuclei, midbrain, cerebellum and pontine and brain-stem nuclei for oculomotor function and for respiratory, neurohumoral, and electrolyte control. The manifestations of acute bilirubin encephalopathy and chronic kernicteric sequelae may be minimal to severe and occur as various combinations (or possibly, isolated findings) of extrapyramidal disorders, neuromotor abnormalities, sensorineural hearing loss, and visual disability. Although not yet demonstrated, some experts believe that milder and subtler neurologic manifestations of BIND exist (**Vinod K , 2004).**