SUMMARY AND CONCLUSION

Jaundice is the most common condition requiring medical attention in newborns. The yellow coloration of the skin and sclera in newborns with jaundice is the result of accumulation of unconjugated bilirubin. In most infants, unconjugated hyperbilirubinemia reflects a normal transitional phenomenon. However, in some infants, serum bilirubin levels may raise exclusively, which can be a cause for concern because unconjugated bilirubin is neurotoxic and can cause death in newborns and lifelong neurologic sequelae in infants who survive (kernicterus). For these reasons, the presence of neonatal jaundice frequently results in diagnostic evaluation.

A common complication of G-6-PD deficiency is that of severe neonatal hyperbilirubinemia with the potential of bilirubin encephalopathy or kernicterus.

Because of the association of G-6-PD deficiency with severe, acute hemolysis, this hyperbilirubinemia has traditionally been related to as hemolytic in origin. In many cases, this may indeed be the cause, and substances which has been applied to the umbilicus for antisepsis, have been implicated as triggers of hemolysis. Additional triggers may include metabolites of Fava, transmitted via breast milk of mothers who had ingested the bean, and henna, which is frequently applied to the newborn’s skin in some Middle Eastern societies. G-6-PD-deficient neonates with obvious, acute hemolysis, in whom no trigger can be identified, are sometimes encountered. Possible agents in such circumstances may include viral or bacterial infections, or newly introduced chemical cleaning substances.

This study was conducted on 100 neonates with unconjugated hyperbilirubinemia that had been recruited from the NICU in Benha children hospital (Bench) during the study period from January 2010 to October 2010.

Neonates included were those with indirect hyperbilirubinemia Newborns who were excluded were those with cholestasis as the cause of hyperbilirubinemia.

Investigations done were serum bilirubin (with direct fraction), complete blood picture (including reticulocytic count), Coombs' test, maternal & neonatal blood group & RH, serum C - reactive protein (CRP) and G-6-PD enzyme assay.

The age of neonates ranged from after birth to day 10. Candidates were 68 males (68%) and 32 were females (32%). 57% of the studied cases (57 neonates) were delivered by N.V.D., while 43% (43 neonates) were delivered by C.S.

There are different classifications of the causes of neonatal hyperbilirubinemia. We categorized exaggerated physiological jaundice, prematurity, breastfeeding or breast milk jaundice, cephalhematoma, sepsis, ABO and RH incompatibility and.

As regarding G-6-PD-deficient neonates as a cause of indirect hyperbilirubinemia, its percentage was 8 % only.

Hyperbilirubinemia in G-6-PD - deficient neonates is thought to be secondary to reduced hepatic conjugation and excretion of bilirubin, rather than increased bilirubin production resulting from hemolysis. Thus no difference in reticulocyte count and hematocrit level between G-6-PD-deficient and normal groups. While there was a strong relation between TsB and G-6-PD level, where peak bilirubin level range was high and duration of phototherapy was long in neonates who had G-6-PD deficiency. In addition, there was no statistically significant difference between G-6-PD normal group and G-6-PD deficient group as regarding the Anthropometric measurements, vital Signs, blood group of the mother and her neonate and mode of delivery.

In **conclusion,** neonatal hyperbilirubinemia is one of the most common problems and requires hospital admission for investigation and treatment. Despite a low prevalence of G-6-PD deficiency in our

study, we recommend that we suspect G-6-PD deficiency in full-term male neonate when we find prolonged indirect hyperbilirubinemia with high TsB level and longer duration of phototherapy than usual and not associated with laboratory evidence of hemolyses specially if there is history of G-6-PD deficiency in the family also we recommend that G-6-PD deficiency tests be performed in all Iranian and Mediterranean icteric newborns, unless other investigators ascertain and document that G-6-PD deficiency tests are not necessary to be done routinely. In addition, we recommend that measurement of the enzyme UGT be made available for the clinical use in the evaluation of neonatal hyperbilirubinemia.