Introduction

Birth asphyxia continues to be the leading cause of neonatal brain injury ***(Freeman, 1988)***. In infants who develop moderate to severe hypoxic ischemic encephalopathy more than 800,000 deaths every year and at least an equal number of neonates suffer from long term neurological morbidities **(WHO, 1991).**

In addition to the irreversible lesion that occurs during ischemia a significant portion of cellar death occurs after an ischemic insult to the brain (**Horn and Schlote, 1992).**

Vascular endothelial growth factor (VEGF) is a polypeptide growth factor that's activated by tissue hypoxia (**semenza, 2000).**

It promotes cell survival by inhibiting apoptosis pathway (**Chenug, 1997).** In addition, it increases vascular density through its potent permeablizing property (**Dvorak, 2002).**

The up-regulation of (VEGF) that occurs in the brain trauma, tumor, growth and stroke might be responsible for the observed increase in vascular permeability and subsequent brain edema formation (**Marti et al., 2000).**

In animal models; antagonism of VEGF decreases the occurrence of brain edema after ischemia (**Van Bruggen et al., 1999),** however the role of (VEGF) in perinatal asphyxia in human neonates is yet to be clarified.

Aim of the work

To assess vascular endothelial growth factor levels in cord blood of infants suffering from perinatal asphyxia and to determine whether there is association exists between the cord blood VEGF and the risk for development of encephalopathy and neurodevelopmental outcomes at age of 3 and 6 months.