

1.1.Introduction:

Neonatal Hypoxic-Ischemic Encephalopathy (HIE), is one of the most common causes of the cerebral palsy and other severe neurologic deficits.

Perinatal asphyxia refers to a condition of impaired gas exchange that leads, if persistent, to fetal hypoxia and hypercapnia. It is one of the most devastating complications associated with the process of birth and occurs during the first and second stages of labor. Perinatal asphyxia occurs in between 2–10% of deliveries and is a major cause of acute mortality and chronic neurologic disability amongst survivors. (Kanik et al, 2009)(Ong et al, 2009).

Neonatal hypoxic ischemic encephalopathy (HIE); the term used to designate the clinical and neuropathological findings of encephalopathy that occurs in a neonate with objective data to support a hypoxic /ischemic mechanism. Hypoxic-ischemic encephalopathy is an important cause of mortality and morbidity in newborns. It occurs in 6 per 1000 live term births and accounts for about 20% of occurrences of cerebral palsy in childhood. Up to 40% of infants with moderate HIE and 100% of those with severe HIE either die or develop neurosensory impairments, including cerebral palsy, mental retardation, and deafness. (Zupan, 2010).

Cerebral palsy is one of the most costly neurologic disabilities because of its frequency (2/1000 births) and persistence over the life span. Central gray matter damage following perinatal hypoxia-ischaemia frequently leads to death or motor abnormality often with deficits in other developmental domains. Predicting these different outcomes of HIE is difficult yet very important for early management, planning and providing for needs on discharge and later and not

least for parents to know how their children will be affected. (Christine et al, 2006).

It is lack of sufficient blood flow in conjunction with decreased oxygen content in the blood leads to loss of normal cerebral auto regulation and to diffuse brain injury. The exact nature of the injury depends on the severity of the hypotension and the degree of the brain maturation. (Donald and Di Salvo, 2001).

Although intervention is limited and mostly supportive at this time, it is still important to promptly and accurately identify neonates who have sustained a hypoxic-ischemic brain injury to facilitate optimal management and make decisions about the provision of active life support. (Pottumarthi , 2006) and (Babcock et al, 1996).

HIE may occur in both term and preterm babies during the transition from in-utero status towards extra uterine adaptation. (6)The pattern of injury in neonates who were delivered before 36 weeks is distinct from the pattern in those delivered at, or after, 36 weeks gestational age. (Donald and Di Salvo, 2001).

In preterm HIE with gestational age less than 36 weeks and birth weight less than 1000g, there is a greater risk for peri ventricular leuckomalacia (PVL). It may occur due to antenatal factors (placenta abruption, pre-eclampsia, premature rupture of membranes, chorioamnionitis and group B streptococcal infection) or peri/ postnatal factors (respiratory distress, sepsis, anemia and bradycardia). (Pottumarthi , 2006).

Manifestations of preterm HIE could be spastic diplegia, quadriplegia, blindness, mental retardation or learning disability with poor outcome if both IVH and PVL with volume loss. (Pottumarthi , 2006).

In term HIE, maternal causes include infection, pre-eclampsia, diabetes mellitus and cocaine abuse while fetal causes include growth retardation, hypocalcaemia, hypoglycemia, sepsis, hyperthermia and congenital heart disease.

The prognosis of term HIE depends on the degree of severity leading to 50% mortality and significant morbidity in 80% of survivors. (Babcock et al, 1996).

Proper diagnosis of HIE lesions and their extensions helps in prediction of outcome and planning of proper management. (Cai et al, 2010).

Outcome of full-term and preterm infants with neonatal encephalopathy of hypoxic-ischemic origin is often assessed in infancy or early childhood and data on outcome in childhood and adolescence is limited.

Accurate identification and characterization of the severity, extension and location of the brain injuries rely on the selection of the appropriate neuroimaging modalities, including ultrasonography (US), computed tomography (CT) and magnetic resonance imaging (MRI). (Cai et al, 2010).

Ultrasound is widely used for the examination of neonatal/ infant brain. It is portable, convenient, very reliable, safe, non invasive and widely available diagnostic tool that allows evaluation of even severely- ill patients in intensive care units. (Vries and Jongmans, 2010).

All of the brain lesions could be effectively and simply monitored with transfontanellar ultrasound scanning via the anterior fontanelle, through the

squamosal portion of the temporal bone and the posterior fontanelle. 5 MHz, sector transducer is needed.

The anterior fontanelle can be used as an acoustic window to get coronal and sagittal images of the brain. Six coronal and six sagittal images are obtained. The base of skull must be perfectly symmetrical on coronal scans. For the sagittal images, the reference plane should be in midline. So, US study of the brain is the most valuable modality during the first 6 months of life. (Vries and Jongmans, 2010).

The cranial U/S shows The earliest ultrasonographic abnormality in PVL consists of bilateral, coarse, globular or broad bands of echogenicity in the periventricular white matter. (Vries and Jongmans, 2010).

MRI performed in the neonatal period has made a huge contribution to recognition of different patterns of injury. These different patterns of injury are related to the severity of later motor and cognitive disabilities. Long-term follow-up shows that cognitive and memory difficulties may follow even in children without motor deficits. It is therefore recommended to perform follow-up assessment into childhood in children with and without adverse neurological outcome in early infancy. (Vries and Jongmans, 2010) and (Shroff et al, 2010).

Magnetic resonance imaging of the brain is invaluable in assessing the neonate who presents with encephalopathy, but successful imaging requires adaptations to both the hardware and sequences used for adults.

Knowledge of the perinatal and postnatal details are essential for the correct interpretation of the imaging findings. Diffusion-weighted imaging (DWI) is

clinically useful for the early identification of ischemic tissue in the neonatal brain, the pattern of which can predict outcome, but may underestimate the final extent of injury, particularly basal ganglia and thalamic lesions. Serial imaging with quantification of both tissue damage and structure size provides invaluable insights into the effects of perinatal injury on the developing brain. (Rutherford et al, 2010).

1.2.Objectives of This Work:

The objectives of this study are:

General:

To describe the imaging presentation of neonate brain.

Special:

Introduction

1-To describe the imaging presentation of HIE.

2-To find the accuracy of MRI (T1WI, T2W1, FLAIR and DWI) and U/S and to correlate with lab findings of HIE problems.

3-To find the points of strength and weakness of US and MRI imaging modalities for evaluating various patterns of HIE.