

Frequency of Hypoxic-Ischemic Encephalopathy among Hospitalized Neonates in West Iran

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Hypoxic-ischemic encephalopathy (HIE) is brain damage from a shortage of oxygen or blood flow to the tissues^[1,2] and is characterized by clinical and laboratory evidence of acute or subacute brain injury due to asphyxia^[1-6]. It is a major contributor to neonatal death and morbidity^[4-6]. 15%-20% of HIE cases die during the neonatal period and 30% of those who survive suffer from neurodevelopmental disorders^[1,3,6].

An estimated 23% of the 4 million neonatal deaths and 8% of all deaths at <5 years of age throughout the world each year are associated with signs of asphyxia at birth^[1,4]. Even at referral centers in developed countries, death or moderate to severe disability occurs for 53% to 61% of infants diagnosed as having moderate to severe HIE^[1,4,6]. Children with moderate/severe neonatal encephalopathy are at risk for reduced school performance, whereas those with mild encephalopathy have school performance scores similar to those of their peers^[1,6]. HIE is one of the most common causes of cerebral palsy and other severe neurologic deficits in children occurring in two to nine of every 1000 live births^[1-6]. The incidence of HIE reported in different studies varies widely^[2-6], which may be explained by the selection criteria for studies of HIE during the neonatal period^[3,4].

The aim of the present study was to evaluate the frequency of hypoxic-ischemic encephalopathy in hospitalized neonates with seizure in Hamedan (west Iran) in a two year period. This is a retrospective cross sectional study on 34 neonates from 2004 to 2006.

Inclusion criteria were: all neonates with seizures due to HIE asphyxia having pH below 7, 5th minute Apgar score between 0 and 3,

decreased muscle tone and consciousness, cortical atrophy in brain CT scan and multiple organ involvement (eg, kidney, lungs, liver, heart, intestines). Neonates with jitteriness were excluded from the study.

The study was based on the recorded files of the patients. CT scan findings, blood gas findings, Apgar score of 5th minute, decreased muscle tone and consciousness, seizure, age, sex and birth weight were recorded and analyzed using SPSS 13. Management plan for evaluation of hypoxic-ischemic encephalopathy included: Profound metabolic or mixed acidemia (pH<7), persistence of Apgar score of 0-3 for longer than 5 minutes, neonatal neurologic sequelae (eg, seizures, coma, hypotonia), multiple organ involvement (eg, kidney, lungs, liver, heart, intestines) and cortical atrophy in brain CT scan.

From 34 neonates with seizure, 11 (32.4%) had HIE. The infants who developed HIE had significantly 5th minute Apgar score between 0 and 3, decreased muscle tone and consciousness, pH below 7 in blood gas, cortical atrophy in brain CT scan and multiple organ involvement. The mean age of the neonates was 14.03±10.05 days (range 1 to 29 days). 25 (73.5%) neonates were boys and 9 (26.5%) girls. 23 (67.6%) neonates had normal weight (2500 to 4000 gr), 6 (17.6%) low birth weight (1500 to 2500 gr) and 5 (14.7%) very low birth weight (less than 1500 gr).

In 1980, the term hypoxic ischemic encephalopathy (HIE) came into use for all phases of ischemic changes^[1]. HIE is a potential cause of brain injury that can produce some alterations of the neurologic development of the newborn^[4-6]. The incidence of HIE reported in different studies varies widely. The variability in the reported incidence of HIE may be explained by the selection criteria for studies of HIE during the neonatal period^[2-6]. In our study the incidence rate of Hypoxic-ischemic encephalopathy was 32.4%, which is higher than rates reported from other countries^[1,2,4,5]. This difference may be due to evaluation of the incidence of HIE in newborns *with* seizure in our study. HIE occurs in two to nine per 1000 live births in developing countries^[1-6]. Thomberg et al from Sweden reported an incidence rate of five and seven per 1000 live births^[4]. The

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