Association between serum interleukin-1β levels and perinatal asphyxia

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Abstract

Objective:
Asphyxia is a major cause of acute mortality and chronic neurologic disability in neonates. We sought to define the predictive values of serum concentrations of interleukin-1β in newborns with perinatal asphyxia to see if there is a relation between interleukin-1β (IL-1β) levels to the short term neurological deficit.

Methods:
This was a prospective (case-control) study conducted between June 2007 and July 2008, at the Neonatal Intensive Care Unit, Ghaem Hospital, Mashhad, Iran. Serum IL-1β levels were measured at birth, 24 and 48 h post-partum in 38 consecutive uninfected neonates with perinatal asphyxia(blood pH< 7.2, low Apgar score , signs of fetal distress) and 41 randomly selected healthy newborns (normal infants free of a postnatal clinical event during the first weeks of life). Receiver-operating characteristic (ROC) curves were used for the determination of thresholds for the asphyxiated group versus healthy neonate group.

Results:
A total of 79 infants were studied. Serum interleukin-1β concentrations in the infants who developed hypoxic-ischemic encephalopathy was 6 folds higher as compared to values in the normal infants (p< 0.006) and 5-folds higher compared to infants with asphyxia who did not subsequently develop hypoxic-ischemic encephalopathy (p< 0.006). There was also a significant relationship between serum IL-1B and outcome at the time of discharge.

Conclusions:
Serum levels of IL-1B are increased substantially in neonates with asphyxia, and this is most pronounced in neonates with poorer prognosis.

Keywords:
Hypoxic- ischemic encephalopathy, Interleukin-1β, Newborn, Perinatal asphyxia

Introduction

Four million children are born annually with severe perinatal asphyxia worldwide (1). Estimates suggest that between 2 and 4/1000 full-term newborn infants suffer From asphyxia at or shortly before birth. Approximately 15% to 33% of such asphyxiated infants who exhibit hypoxic-ischemic encephalopathy (HIE) actually die during the neonatal period. Of the survivors, 25% will exhibit permanent neuropsychological deficits(2, 3). There is evidence supporting the involvement of the inflammatory cascade in the pathogenesis of ischemic brain injury. IL-1β is an important cytokine released mainly by mononuclear cells and macrophages in response to infection