

<https://doi.org/10.46344/JBINO.2023.v12i02.20>

NEONATAL ASPHYXIA IN CHILDREN

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ABSTRACT

Perinatal asphyxia is one of the main causes of death in term newborns. Prevalence and mortality of asphyxia are still high. The determinant factors for birth asphyxia include Primi-parity, prolonged labour, foetal malpresentation, presence of meconium, preeclampsia, antepartum hemorrhage, gestational diabetes mellitus, premature rupture of membrane, post-term pregnancy, fetal distress, non-vertex presentation, cord entangled among many others. Therefore, the existing efforts of emergency obstetric and newborn care should be strengthened to prevent birth asphyxia from the complications of fetal mal-presentation, premature rupture of fetal membranes, meconium stained amniotic fluid and vacuum delivery. Early detection and intervention of high risk mothers should also be carried out by health care providers, and mothers should be monitored with partograph during labour.

Keywords: neonates, asphyxia, children

Introduction

Perinatal asphyxia is defined as the inability of the newborn to initiate and sustain enough respiration after delivery and is characterized by a marked impairment of gas exchange [1-5]. According to World Health Organization (WHO) birth asphyxia is a failure to initiate and sustain breathing at birth. Lack of effective respiration results in hypoxemia (lack of oxygen) and hypercapnia (accumulation of carbon dioxide), both of these depress cardiac function. In asphyxiated newborns, severe hypoxic damage occurs in many organs, including the brain, heart, kidney, liver, lungs, and gut but brain damage is of most concern and perhaps the least likely to recover.

Every year approximately 4 million babies are born asphyxiated; this results in 1 million deaths and an equal number of serious neurological consequences including hypoxic-ischemic encephalopathy, cerebral palsy, mental retardation, and epilepsy (J. E. Lawn et al., 2007). According to the World Health Organization, perinatal asphyxia is one of the three common causes of under-five child mortality (11%) following preterm birth (17%) and pneumonia (15%) [1].

Neonatal asphyxia

Neonatal asphyxia assumed to be related to intrapartum related hypoxia-ischemia, accounts for one million neonatal deaths annually. In the low resource setting BA is usually defined as a failure to initiate or sustain spontaneous breathing at birth. In the resource replete setting BA is a biochemical definition related to impaired gas exchange, due to interruption of placental blood flow (PBF). An umbilical arterial pH <7.00 referred to as severe fetal acidemia, reflects a degree of acidosis, where potential risk of adverse neurologic sequelae is increased.

Epidemiology

Systematic and meta-analysis review in East and Central Africa showed

prevalence of perinatal asphyxia with 15.9% and regional subgroup analysis indicates prevalence of perinatal asphyxia was 18.0% in East and Central African countries respectively. The pooled prevalence of perinatal asphyxia was high therefore, there is a call to reduce the high burden of this problem in the region [6].

In a retrospective descriptive study of neonates born at term or near term in Chris Hani Baragwanath Academic Hospital, Johannesburg, South Africa, the occurrence of asphyxia ranged from 8.7 to 15.2/1 000 live births, (Bruckmann et al., 2015). Another descriptive retrospective study conducted in a tertiary academic hospital in Johannesburg, South Africa, found a rate of perinatal asphyxia of 4.7 per 1000 live births; 76.5% of all asphyxiated newborns had evidence of hypoxic-ischemic encephalopathy [7].

Clinical features of neonatal asphyxia

Perinatal asphyxia can result in systemic effects, including neurologic insult, respiratory distress and pulmonary hypertension, and liver, myocardial, and renal dysfunction. Depending on the severity and timing of the hypoxic insult, a neonate with hypoxic-ischemic encephalopathy due to perinatal asphyxia can demonstrate a variety of neurologic findings. Using the Sarnat staging for encephalopathy can be useful. In Sarnat Stage I, the least severe stage, there is generalized sympathetic tone and the neonate may be hyper-alert with prolonged periods of wakefulness, mydriasis and increased deep tendon reflexes. In Sarnat Stage II, the neonate may be lethargic or obtunded, with decreased tone, strong distal flexion, and generalized parasympathetic tone with miosis, bradycardia and increased secretions. Seizures are common in Sarnat Stage II. Sarnat Stage III, the most severe, is characterized by a profoundly decreased level of consciousness, flaccid tone, decreased deep tendon reflexes

and very abnormal EEG. Clinical seizures are less common in Sarnat Stage III due to the profound injury in the brain preventing the propagation of clinical seizures [1].

Conclusion

The overall pooled prevalence of perinatal asphyxia is remarkably high. Prolong labor, meconium-stained amniotic fluid, instrumental deliveries, and low birth weight are the determinants factors of perinatal asphyxia. Efforts might be made to improve the quality of intrapartum care services to prevent labor, delivery and fetal complications and to identify and make a strict follow up of mothers with meconium-stained amniotic fluid.

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