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A term neonate with hypoxic ischemic encephalopathy: A case report

Abigail D'Souza; Ashane Lynch*

***Corresponding Author: Ashane Lynch**
Trinity School of Medicine, St. Vincent and the Grenadines, USA.
Email: ashane.lynch@gmail.com

Abstract

Hypoxic-ischemic Encephalopathy (HIE), also known as intrapartum asphyxia, is a paramount cause of brain injury in the newborn. It can result in devastating consequences including mental retardation, cerebral palsy, and epilepsy [1]. The majority of sequelae in HIE are a result of impaired cerebral blood flow and oxygen delivery to the brain. The newborn can briefly compensate for short periods of depleted oxygen but if the asphyxia is prolonged, the brain tissue deteriorates. The severity and duration of oxygen deprivation determines whether HIE occurs and how detrimental it is. Neuroimaging techniques, with cranial ultrasound, CT scans and MRIs are performed to aid in the diagnosis of HIE [2]. This review presents a case of a term female neonate who was stuck in the birthing canal for 7 minutes raising concern for hypoxic ischemic encephalopathy. This case report discusses the underlying pathophysiologic effects of HIE as well as the variable outcomes and management strategies.

Keywords

hypoxic ischemic encephalopathy; brain injury; intrapartum asphyxia; neonatal encephalopathy; Oxidative stress.

Introduction

Hypoxic-ischemic encephalopathy occurs in 1.5 per 1000 live full term births [3]. Approximately 15-25% of these individuals die in the postnatal period and 25% develop permanent neuropsychological sequelae [4]. By the age of 2 years, 60% of infants with HIE will die or have severe disabilities that includes cerebral palsy, epilepsy and mental retardation [1]. There has been no decline in the incidence rate of HIE despite advances in techniques aimed at preventing hypoxic ischemic events such as fetal monitoring [5]. The main treatment of HIE focuses on reducing the severity of subsequent brain injury [6].

A frequently occurring cause of hypoxic injury in a term infant is due to maternal circulatory problems leading to intrauterine asphyxia. These include cloting of placental arteries, inflammatory mediators as well as placental abruption [7]. There is then a diminished exchange of oxygen and carbon dioxide in the neonatal circulation and a severe elevation of lactic acidosis [7]. Furthermore, there is evidence indicating that 25% of term infants born with a cord pH <7.0 have an increased risk of neonatal neurologic morbidity and mortality [8].

Inflammation after hypoxic ischemia can lead to further neuronal injury. This process involves the activation of microglia and the release of proinflammatory cytokines resulting in the phagocytosis of injured neurons. Preventing this inflammatory reaction aids in neuroprotection and can be used in the clinical treatment of ischemic brain injury [9]. This report presents a case of postnatal hypoxic ischemic encephalopathy due to problems during delivery.

Case Presentation

A newborn baby girl was born by spontaneous vaginal delivery at 39.6 weeks gestation to a 29-year-old gravida 2, para 2 mother. Delivery history was significant for the baby being stuck within the birthing canal for 7 minutes. During this time, the mother was only 7 cm dilated, and the baby's heart rate dropped to 50 bpm. The cervix then had to be manually reduced to 10 cm due to the fetal bradycardia.

Prenatal history was significant for the mother being positive for Group B strep, which was treated prophylactically. Her toxicology screen was positive for marijuana in which her last known usage was a week prior to giving birth. Upon delivery, the baby weighed 7 lbs and 4.2 oz. APGAR scores at 1, 5 and 10 minutes were 2, 3, and 6 with a final discharge APGAR score of 8. Cord pH was 7.12, cord CO_2 was 53.9 and cord base excess was -12.4. Following delivery, it was noted that the baby was posturing. She was taken to the NICU and a complete blood count, c-reactive protein, blood culture and chest x-ray were ordered. CPAP 5 L at 21% was given. Vitals signs were noted as 100.8 F temperature, HR of 187 bpm, RR of 60 breaths/min and pulse oxygen of 100%. Labs showed MCH of 35.4 and MCHC of 33.3. Blood gas had a bicarbonate of 14, total CO_2 of 15, arterial pH of 7.18 and arterial O_2 of 101. Lactic acid was noted at 6.3 mmol/l and the Alan test was positive indicating that there was inadequate blood flow. Chest x-ray with AP and lateral viewing showed bilateral mild prominent lung markings and mild ground glass densities on the right lung, which was concerning for neonatal respiratory distress syndrome.

Within 1 hour after delivery she was given dextrose via IV 250 ml at 11 ml/hr, ampicillin sodium 300 mg IV, and gentamicin sulfate 13 mg IV. She had another episode of abnormal posturing and was in acute respiratory distress. She was then given phenobarbital 65 mg IVP and 10 ml of ampicillin sodium 300 mg in sterile water at 20 ml/hr. She was still posturing 30 minutes later and was given 10 ml of phenobarbital 65 mg in dextrose at 60 ml/hr. Within the next three hours she was given 6.5 mg of gentamicin sulfate 13 mg in sterile water at 13 ml/hr via IV twice.

The newborn was transferred to a nearby hospital due to concerns of hypoxic ischemic encephalopathy the next day.

Discussion

Hypoxic ischemic Encephalopathy (HIE) is induced by factors that disrupt cerebral blood flow and lead to a lack of oxygen in the affected area. It is typically traced to antepartum or intrapartum anomalies such as cephalopelvic disproportion or abruption [10]. This makes HIE one of the leading causes of increased risk of death and lifelong disabilities in individuals. These disabilities can present as visual or learning impairments, epilepsy, blindness or even cerebral palsy [11]. One of the most widely accepted pathophysiological mechanisms for HIE is this generation of oxidative free radicals seen in hypoxic events, that subsequently lead to damage. Due to an infant's brain having high concentrations of sensitive and immature cells with a low concentration of antioxidant enzymes, their brains require high levels of oxygen supply, leaving them extremely susceptible to hypoxia [12]. Following an hypoxic-ischemic event, such as being 'Stuck' within the birthing canal like our patient, oxidative stress plays an important role in the pathogenesis of HIE. Oxidative stress can later trigger the release of oxygen and nitrogen species, generate free radicals, cause inflammation, apoptosis, autophagy and even necrosis [13].

Chest x-rays for our patient indicated that our patient was in neonatal respiratory distress, showing bilateral mildly prominent lung markings and mild ground glass densities in the right lung. Neonatal respiratory distress syndrome, which was a concern during our case, is associated with an elevated risk of neonatal hypoxic ischemia [14]. Due to the presence of respiratory distress in our patient, it could be said that this, along with being 'Stuck' within the birthing canal for 7 minutes, attributed to our patients presentation of HIE, due to increasing levels of oxidative stress.

Term neonates who are subjected to significant hypoxic events are frequently diagnosed with the usage of MRIs. Initial findings are subtle and therefore require the usage of diffusion weighted imaging to show the progression of the lesions. After initial injury, MRIs will show an increase in lactic acid and reduction of N-acetyl-aspartate, revealing a patern of progression of the hypoxic event [15]. The asphyxia patern on MRI scans shows selective injury to the putamen, thalamus and cerebral cortex while often sparing the white mater of the brain [16]. Infants who show an increase in the T2-weighted MRI signal in the posterior internal capsule often require resuscitation to survive and have a high metabolic acidosis in the umbilical cord blood [17].

Acidosis is associated with neonatal morbidity and is often defined as low umbilical pH below or a high umbilical base deficit which is usually indicated as a negative base excess [18]. The patient in this case had a low cord pH of 7.12 and a base excess of -12.4 which were predictive values for a poor neonatal outcome. Furthermore, it has been shown that umbilical cord lactate levels >3.21 mmol/l has a sensitivity of 69.7% and a specificity of 93% to predict the occurrence of poor neurological outcomes including hypoxic ischemic encephalopathy [19], which was also seen in our patient's elevated lactate level of 6.3 mmol/l.

One clinical strategy used to mediate the effects of HIE as the standard treatment is therapeutic hypothermia. This is used as a protective tool in moderate to severe HIE in clinical setings. The two methods clinically used are selective head cooling to 34.5 C or to do total body cooling to 33.5 C [12]. This decrease in temperature redistributes blood blow and affects microcirculation disturbances, leading to

improved levels of antioxidants [20]. This treatment must be given within the first six hours of birth if HIE is suspected and must be maintained for three days, as studies have shown that hypothermia reduces cerebral injury and improves brain outcome secondary to hypoxic ischemic attacks [21]. Recent studies have also suggested, in adjunct with therapeutic hypothermia, allopurinol, a xanthine-oxidase inhibitor, can be used as a direct scavenger of free radicals, leading to a decrease in neuronal damage in HIE infants [22-25]. Another recent study, using animal models, indicated that the used of glycine in HE patients could decrease the mitochondrial autophagy that occurs during hypoxic events, leading to atenuation in hypoxic ischemic injury in the neurons of these patients [26].

Conclusion

Hypoxic ischemic injury in a developing brain often occurs as a result from asphyxiation at birth. There are varied outcomes and these are often determined by the severity and the duration of the hypoxia. If the hypoxic event is severe enough to damage the brain, it takes 12 to 36 hours to lead to neonatal hypoxic ischemic encephalopathy [27]. HIE is an evolving process though it is caused by a single initial injury. The inflammatory response triggered by the ischemia leads to neuronal damage and results in conditions such as cerebral palsy and epilepsy. Diagnostic imaging as well as biochemical indicators such as umbilical cord pH, lactic acid levels, base excess are useful to predict the occurrence of poor neonatal outcomes including HIE. Furthermore, there is some evidence that anti-inflammatory mediators and hypothermia can have promising neuroprotective effects for the newborn.

References

1. Pierrat V, Haouari N, Liska A, et al. Prevalence, causes, and outcome at 2 years of age of newborn encephalopathy: population based study. Archives of Disease in Childhood: Fetal Neonatal Edition. 2005; 90: F257–261.

2. Bano S, Chaudhary V, Garga UC. Neonatal Hypoxic-ischemic Encephalopathy: A Radiological Review. J Pediatr Neurosci. 2017; 12: 1-6.

3. Kurinczuk JJ, White-Koning M, Badawi N. Epidemiology of neonatal encephalopathy and hypoxic-ischaemic encephalopathy. Early Hum Dev. 2010; 86: 329-338.

4. Lai MC, Yang SN. Perinatal hypoxic-ischemic encephalopathy. J Biomed Biotechnol 2011; 2011: 609813.

5. Kumar S, Paterson-Brown S. Obstetric aspects of hypoxic ischemic encephalopathy. Early Hum Dev. 2010; 86: 339–344.

6. Shankaran S. Neonatal encephalopathy: Treatment with hypothermia. J Neurotrauma. 2009; 26: 437–443.

7. Locatelli A, Incerti M, Ghidini A, Greco M, Villa E, Paterlini G. Factors associated with umbilical artery acidemia in term infants with low Apgar scores at 5 min. Eur J Obstet Gynecol Reprod Biol. 2008; 139: 146-50.

8. Graham EM, Ruis KA, Hartman AL, Northington FJ, Fox HE. A systematic review of the role of intrapartum hypoxia-ischemia in the causation of neonatal encephalopathy. Am J Obstet Gynecol. 2008; 199: 587-95.

9. Chew LJ, Takanohashi A, Bell M. Microglia and inflammation: impact on developmental brain injuries. Ment Retard Dev Disabil Res Rev. 2006; 12: 105–12.

10. Martinez-Biarge M, Diez-Sebastian J, Wusthoff CJ, Mercuri E, Cowan FM. Antepartum and intrapartum factors preceding neonatal hypoxic-ischemic encephalopathy. Pediatrics. 2013; 132: e952-e959.

11. Allen CL, Bayraktutan U. Oxidative stress and its role in the pathogenesis of ischaemic stroke. Int J Stroke. 2009; 4: 461-470.

12. Zhao M, Zhu P, Fujino M, et al. Oxidative Stress in Hypoxic-Ischemic Encephalopathy: Molecular Mechanisms and Therapeutic Strategies. Int J Mol Sci. 2016; 17: 2078.

13. Doyle KP, Simon RP, Stenzel-Poore MP. Mechanisms of ischemic brain damage. Neuropharmacology. 2008; 55: 310-318.

14. Lauterbach MD, Raz S, Sander CJ. Neonatal hypoxic risk in preterm birth infants: the influence of sex and severity of respiratory distress on cognitive recovery. Neuropsychology. 2001; 15: 411-420.

15. Takeoka M, Soman TB, Yoshii A, et al. Diffusion-weighted images in neonatal cerebral hypoxic-ischemic injury. Pediatr Neurol. 2002; 26: 274-281.

16. Barkovich AJ, Westmark K, Partridge C, Sola A, Ferriero DM. Perinatal asphyxia: MR findings in the first 10 days. AJNR Am J Neuroradiol. 1995; 16: 427-38

17. Okereafor A, Allsop J, Counsell SJ, et al. Paterns of brain injury in neonates exposed to perinatal sentinel events. Pediatrics. 2008; 121: 906-914.

18. Malin GL, Morris RK, Khan KS. Strength of association between umbilical cord pH and perinatal and long term outcomes: systematic review and meta-analysis. BMJ. 2010; 340: c1471.

19. Allanson E, Waqar T, White C, Tuncalp O. Dickinson J. Umbilical lactate as a measure of acidosis and predictor of NEONATAL risk: A systematic review. Obstetric Anesthesia Digest. 2017; 37: 177-177.

20. Bayir H, Adelson PD, Wisniewski SR, et al. Therapeutic hypothermia preserves antioxidant defenses after severe traumatic brain injury in infants and children [published correction appears in Crit Care Med. 2009; 37: 1536. Crit Care Med. 2009; 37: 689-695.

21. Silveira RC, Procianoy RS. Hypothermia therapy for newborns with hypoxic ischemic encephalopathy. J Pediatr (Rio J). 2015; 91: S78-S83.

22. Chaudhari T, McGuire W. Allopurinol for preventing mortality and morbidity in newborn infants with suspected hypoxic-ischaemic encephalopathy. Cochrane Database Syst Rev. 2008; (2): CD006817. Published 2008.

23. Kaandorp JJ, Benders MJ, Rademaker CM, et al. Antenatal allopurinol for reduction of birth asphyxia induced brain damage (ALLO-Trial); A randomized double blind placebo controlled multicenter study. BMC Pregnancy Childbirth. 2010; 10: 8.

24. Buonocore G, Perrone S, Turrisi G, Kramer BW, Balduini W. New pharmacological approaches in infants with hypoxic-ischemic encephalopathy. Curr Pharm Des. 2012; 18: 3086-3100.

25. Legido A, Valencia I, Katsetos CD, Delivoria-Papadopoulos M. Neuroprotección en la encefalopatía hipóxico isquémica perinatal. Tratamientos con eficacia clinica demostrada y perspectivas futuras [Neuroprotection in perinatal hypoxic-ischemic encephalopathy. Effective treatment and future perspectives]. Medicina (B Aires). 2007; 67: 543-555.

26. Cai CC, Zhu JH, Ye LX, et al. Glycine Protects against Hypoxic-Ischemic Brain Injury by Regulating Mitochondria-Mediated Autophagy via the AMPK Pathway. Oxid Med Cell Longev. 2019; 2019: 4248529. Published 2019.

27. Volpe JJ. Perinatal brain injury: from pathogenesis to neuroprotection. Ment Retard Dev Disabil Res Rev. 2001; 7: 56-64.

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Authors Information: Abigail D'Souza; Ashane Lynch*
Trinity School of Medicine, St. Vincent and the Grenadines, USA.

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