ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Death Due to Birth Asphyxia: A Case Report.

Haneil Dsouza[1], Prashantha Bhagavath[2]*, Navin Patil[3], Francis NP Monteiro[4], Karthik Rao N[5], and Raghavendra Rao[5].

[1]Assistant Professor, Department of forensic medicine, KMC Manipal, Manipal University, Karnataka, India.
[2]Associate Professor, Department of forensic medicine, KMC Manipal, Manipal University, Karnataka, India.
[3]Assistant Professor, Department of Pharmacology, KMC Manipal, Manipal University, Karnataka, India.
[4]Professor, Department of Forensic medicine, AJ institute of medical sciences and research centre, Mangalore, Karnataka, India.
[5]Assistant Professor, Department of Medicine, KMC Manipal,Manipal University, Manipal, Karnataka, India.

ABSTRACT

All deaths are unfortunate but deaths of vulnerable populations have a greater emotional impact. Among these populations the foremost is women and children. Developing countries face the dual hardship of trying to both decrease maternal mortality and as well as child mortality. Birth asphyxia is the fifth largest cause of under-five child deaths (8.5%), after pneumonia, diarrhoea, neonatal infections and complications of preterm birth. Birth asphyxia accounts for an estimated 0.92 million neonatal deaths annually and is associated with another 1.1 million intrapartum stillbirths, as well as an unknown burden of long-term neurological disability and impairment. In this case of birth asphyxia, all events and circumstances of the pregnancy were favourable until the actual delivery. Unfortunately here, despite all measures taken to save a child that should have been a low risk candidate for birth asphyxia; the baby could not be saved. The treatment and management of birth asphyxia still has a long way to go so as to not only reduce morbidity but most importantly reduce mortality.

Keywords: Birth asphyxia, APGAR, death, hypoxic ischaemic encephalopathy

Corresponding author

ISSN: 0975-8585

INTRODUCTION

All deaths are unfortunate but deaths of vulnerable populations have a greater emotional impact. Among these populations the foremost is women and children. Developing countries face the dual hardship of trying to both decrease maternal mortality and as well as child mortality. Unfortunately by decreasing the mortality in one population, it does not necessarily mean that the other population's health needs will automatically take care of themselves. Hence health service delivery has to be aimed at both the mother and child. In the most unfortunate of instances, in spite of all measures and precautions, the outcome is still negative. Even in certain pockets in developing countries that have a reasonably high standard of medical care, comparable in fact to the developed world, things can still go wrong. This might reflect not just on the idiosyncrasies of the human body both also on the fact that science and medicine has a long and rocky road ahead of it still, even for issues at the start of life.

Birth asphyxia is the fifth largest cause of under-five child deaths (8.5%), after pneumonia, diarrhoea, neonatal infections and complications of preterm birth [1]. Birth asphyxia accounts for an estimated 0.92 million neonatal deaths annually and is associated with another 1.1 million intrapartum stillbirths [2], as well as an unknown burden of long-term neurological disability and impairment [3]. If 10 million child deaths [1] are combined with 3.2 million stillbirths [4], then birth asphyxia plus intrapartum stillbirths constitute the number-one cause of child and late foetal deaths.

In this case of birth asphyxia, all events and circumstances of the pregnancy were favourable until the actual delivery. Unfortunately here, despite all measures taken to save a child that should have been a low risk candidate for birth asphyxia; the baby could not be saved. Here the various complications that birth asphyxia that are possible even in a short duration of time.is amply demonstrated.

CASE HISTORY

The baby was the result of a spontaneous conception and the pregnancy was a supervised one. The mother had undergone pre-natal check-ups, had been immunized against tetanus and was on iron and calcium prophylaxis as well. Ante-natal scans were said to be normal. There was no history of high blood pressure or gestational diabetes mellitus. There was no history of leaking or bleeding per vaginum and foetal movements were well appreciated. The pregnancy proceeded till 37 weeks.

However the mother experienced seizures just before delivery. The delivery was a forceps assisted VD. The baby birth weight and length were appropriate for the gestational age. The baby did not cry immediately after birth. The APGAR score at one minute was 2 out of 10 but the 5 minute score was not known. At birth the baby's heart rate was under a 100 beats per minute with no spontaneous breaths. It received one cycle of bag and mask ventilation but spontaneous breathing was established only after 10 minutes. There was also no improvement in tone. The baby was thus referred to a higher care centre for further management.

On initial examination in the higher care centre, the baby was lethargic, no spontaneous cry or activity was present. The baby had decreased tone suggestive of hypoxic ischaemic encephalopathy stage 2. Hypothermia protocol was thus started for 72 hours after which gradual rewarming was done. Preliminary investigations showed decreased renal function tests and liver function tests and elevated Troponin T levels, suggestive of birth asphyxia. Hypoglycaemic episodes were seen. Serum insulin levels were high while cortisol levels were normal. The hypoglycaemic episodes were corrected with intra-venous dextrose after which no further episodes of hypoglycaemia were noted.

The baby was then observed to have nystagmus and lip smacking movements suggestive of mild seizures. This was managed with loading dose of phenobarbitone. On day 6 of admission there started poor respiratory efforts with jerky breathing. Hence the baby was intubated and assisted ventilation started. Blood was sent for culture but empirical treatment with antibiotics was started. The sepsis screen done showed growth of Enterobacter cloacae and appropriate antibiotics were given. On day 8 of admission there was decreased urinary output due to which the possibility of acute renal failure was entertained with subsequent switching of antibiotics. However the renal function tests were normal.

The baby also had severe thrombocytopenia and anaemia requiring transfusions of platelets and packed red cells. The baby also required fresh frozen plasma transfusions during hospitalization. Subsequent hypotension was noted which required ionotropic support. Further attacks of seizures were seen which was treated with phenytoin. On day 13 of admission, episodes of saturation were noticed which required high ventilatory parameters. By day 15 of admission, the episodes of desaturation were more persistent and in spite of all resuscitative measures, the baby died.

DISCUSSION

In less developed countries perinatal asphyxia remains a major cause of death and disability. The pattern of risk factors, the nature of sequelae, and the options and priorities for intervention (both preventive and therapeutic) are areas that require proper study and research. Criteria for the assessment of asphyxia in many studies have been non-specific, for example in the largest follow up study in the USA the correlation between Apgar scores and long term outcome was poor.[5] Early onset neonatal encephalopathy (generally regarded to be due to intrapartum hypoxic/ischaemic injury) is probably the most specific method for assessing asphyxia in the new-born period.[6] Hypoxic-ischaemic encephalopathy (HIE) is known to be associated with marked derangement of cerebral energy metabolism and is also more predictive of outcome. [7]

If the incidence of HIE is significantly higher in developing countries this may present heavy social and economic costs. It may present a particular burden for women both in terms of caring for handicapped children and, if the affected infant dies, exposure to the risk of another pregnancy with a short birth interval.[8] The traditional spacing mechanisms of breast feeding and abstinence are halted by the death of a new-born infant.

Prenatal risk factors such as multiparity, heavy work and poor maternal nutrition increase the risk of asphyxia and poor pregnancy outcome largely by increasing the risk of low birthweight infants. Intrapartum risk factors for asphyxia are often unpredictable, for example cord prolapse and maternal haemorrhage, and can only be addressed if there is access to trained birth attendants and secondary level referral facilities which can deal with obstetric complications. Postnatal complications may convert a mild-moderate asphyxial insult into a severe one. An interesting research question still to be answered is whether the potentially damaging effects of hypothermia - hypoglycaemia, metabolic stress, and apnoea especially in low birthweight infants - outweigh the suggested benefits of controlled hypothermia as a useful therapy in reducing the sequelae of asphyxia.[9] Perinatal asphyxia also contributes, or predisposes, to a much wider range of problems in the neonatal period: feeding intolerance,[10] septicaemia,[11] hepatic damage,[12] hypoglycaemia,[13] transient hyperinsulinism,[14] acute renal failure,[15] conjunctivitis,[16] myocardial dysfunction leading to changes in cerebral blood flow,[17] diminished splenic function,[18] consumption of coagulation factors,[19] thrombocytopenia,[20] necrotising enterocolitis,[21] and changes in cortisol and dihydoxyepiandrosterone concentrations which may have secondary effects on immune function.[22] Later additional sequelae may include growth hormone deficiency,[23] complex partial seizures,[24] cortical visual impairment,[25] and blindness.[26] Unfortunately as this case reflects, despite all measures in some cases, the most dreaded complication of death still arises. The treatment and management of birth asphyxia still has a long way to go so as to not only reduce morbidity but most importantly reduce mortality.

REFERENCES

[1] Bryce J, Boschi-Pinto C, Shibuya K, Black RE: WHO estimates of the causes of death in children. Lancet 2005, 365:1147-1152.

[2] Lawn J, Shibuya K, Stein C: No cry at birth: global estimates of intrapartum stillbirths and intrapartum-related neonatal deaths. Bull WHO 2005, 83:409-417.

[3] Shibuya K, Murray C: Birth Asphyxia. In The Global Burden of Disease: a comprehensive assessment of mortality and disability from diseases, injuries and risk factors in 1990 and projected to 2020 Edited by: Murray C and Lopez A. Cambridge, MA, Harvard University Press;1996:429-453.

[4] Stanton C, Lawn JE, Rahman H, Wilczynska-Ketende K, Hill K: Stillbirth rates: delivering estimates in 190 countries. Lancet 2006, 367:1487-1494

[5] Nelson KB, Ellenberg JK. Apgar scores as predictors of chronic neurological disability. Pediatrics 1981; 68: 36-44.

[6] Volpe J. Neurology of the newborn. 2nd Ed. Philadelphia: Saunders, 1987.
[7] Roth SC, Edwards AD, Cady EB, et al. Relationship between cerebral oxidative metabolism following birth asphyxia, and neurodevelopmental outcome and brain growth at one year. Dev Med Child Neurol 1992; 34:285-95
[8] Acsadi GTF, Johnson-Acsadi G. Childbearing patterns affecting infant and early childhood mortality. Optimum conditions for childbearing. London: International Planned Parenthood Federation, 1986: 13-37
[9] Palmer C, Vannucci RC. Potential new therapies for perinatal cerebral hypoxia ischemia. Clin Perinatol 1993; 20: 411-32.
[10] Berseth CL, McCoy HH. Birth asphyxia alters neonatal intestinal motility in term neonates. Pediatrics 1992; 90: 669-73.
[11] Antia-Obong OE, Utsalo SJ, Udo JJ, Udo KT. Neonatal septicaemia in Calabar, Nigeria. Cent AfrtMed 1992; 38: 161-5.
[12] Saili A, Sarna MS, Gathwala G, Kumari S, Dutta AK. Liver dysfunction in severe birth asphyxia. Indian Pediatr 1990; 27: 1291-4.
[13] Singhal PK, Singh M, Paul VK, Deorari AK, Ghorpade MG, Malhotra A. Neonatal hypoglycemia - clinical profile and glucose requirements. Indian Pediatr 1992; 29: 167-7 1.
[14] Schultz K, Soltesz G. Transient hyperinsulinism in asphyxiated newborn infants. Acta Paediatr Hung 1991; 31: 47-52.
[15] Jayashree G, Dutta AK, Sama MS, Saili A. Acute renal failure in asphyxiated newborns. Indian Pediatr 1991; 28: 19-23.
[16] Pandey KK, Bhat BV, Kanungo R, Srinivasan S, Rao RS. Clinicobacteriological study of neonatal conjunctivitis. Indian Jf Pediatr 1990;57:527-31.
[17] Van-Bel F, Walther FJ. Myocardial dysfunction and cerebral blood flow velocity following birth asphyxia. Acta Paediatr Scand 1990; 79:756-62.
[18] McKay JG, Hermansen MC, Maley BE. Diminished splenic function in asphyxiated term infants. Jf Perinatol 1990; 10: 12-5.
[19] Andrew M, O'Brodovich H, Mitchell L. Fetal lamb coagulation system during birth asphyxia. Am Jf Hematol 1988; 28: 201-3.
[20] Castle V, Andrew M, Kelton J, Giron D, Johnston M, Carter C. Frequency and mechanism of neonatal thrombocytopenia. J Pediatr 1986; 108:749-55.
[21] Boo NY, Goon HK. Epidemiology of necrotising enterocolitis in Malaysian neonates. Singapore MedJ7 1989; 30: 444-8.
[22] Procianoy RS, Giacomini CB, Oliveira ML. Fetal and neonatal cortical adrenal function in birth asphyxia. Acta Paediatr Scand 1988; 77:671-4.
[23] Gao TS, Shi YF, Gao SM. Evaluation of adult idiopathic growth hormone deficiency with other pituitary hormones deficiency. Chung Hua Nei Ko Tsa Chih 1990; 29: 205-9.
[24] Pratap RC, Gururaj AK. Clinical and electroencephalographic features of complex partial seizures in infants. Acta Neurol Scand 1989; 79:123-7.
[25] Roland EH, Jan JE, Hill A, Wong PK. Cortical visual impairment following birth asphyxia. Pediatr Neurol 1986; 2: 133-7.
[26] Goggin M, O'Keefe M. Childhood blindness in the Republic of Ireland: a national survey. BrJ Ophthalmol 1991; 75: 425-9