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Case Report

A Severe Asphyxiated New Born

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Abstract

Perinatal asphyxia is still a major cause of mortality and morbidity despite significant improvements in neonatal intensive care. The incidence of perinatal asphyxia is 6/1000 in term infants, and it is the third leading cause of death after prematurity and sepsis. Perinatal asphyxia is an important cause of permanent damage to the central nervous system which may result in cerebral palsy and developmental disability later on. It can also affect renal, gastrointestinal, and hepatic systems and may cause severe non-reversible sequelae. Therefore, management of asphyxic new-borns, appropriate resuscitation, and planning of follow-up and treatment by an experienced team are helpful to reduce mortality and morbidity. Herein, we report a severe asphyxiated newborn in the light of literature review.

Keywords: Perinatal asphyxia; newborn

Introduction

Perinatal asphyxia is a clinical process in which hypoxia and ischemia coexist. It can be defined as the clinical depression table associated with hypoxia, hypercapnia and acidosis in the newborn and/or foetus depending on the impairment of the functions of the biological unit consisting of mother, foetus and placenta and the impairment of postpartum pulmonary gas exchange. According to the clinical findings, it is the clinical presentation in the newborn which develops due to low Apgar score, acidosis in the cord blood and hypoxic ischemic encephalopathy [1,2]. Although the reported values for the incidence of perinatal asphyxia are variable due to differences in diagnostic criteria, the incidence of perinatal asphyxia is reported to be 6/1000 in the literature [3,4]. Also, asphyxia (23%) is the third most common cause of newborn deaths after premature birth (28%) and sepsis (26%) [5]. Herein, we report a severe asphyxiated newborn in the light of literature review.

Case Report

A 42-year-old mother, in her third pregnancy, gave birth to a male term baby weighing 2615 g with caesarean section due to foetal distress. His height was 46cm and the head circumference was 33cm. After birth, his overall condition was very poor, his heart was not beating, and he was not breathing. There was ecchymosis around the umbilicus and the chest area. He had respiratory depression and the Apgar score was 0 at the first min and 2 at the fifth min. Cornea was dull and pupillary light reflex was weak. The blood pressure was 70/30 mmHg. There was no other abnormal finding. Following resuscitation and intubation, he was referred to the intensive care unit with the diagnosis of severe asphyxia. There were two costal fractures on the left side on posterioanterior chest X-ray. The echogenicity of the bilateral kidney parenchyma found to be increased in abdominal ultrasound (USG) and cranial sulci were present on cranial USG (Figure-1 A chest X-ray image of the patient). His personal and familial history were unremarkable. In the first arterial blood gas analysis; pH was 6.56 mm/Hg, PCO2 was 113 mm/Hg, PO2 was103 mm/Hg, HCO3 was 9.5 mmol/L, BE was -25 mmol/L. In blood count analysis, leukocyte count was 69.140mm3; platelet count was 65.000 mm3, Htc59%, and Hgb 18g/dL. In biochemical analysis of blood; Na was 129 mmol/L, blood glucose was 37mg/dL, and CRP was 3.52mg/Dl(reference <0.5). Total urine analysis revealed protein positivity. The metabolic screening tests were normal. The mortality score was 32.6% according to the Score for Neonatal Acute Physiology and Perinatal Extension II(SNAPPE-II). The newborn was internalized with the diagnosis of asphyxia and was connected to the ventilator in the SIMV mode. For hypoglycaemia, 2cc of 10% dextrose was administered as bolus and 10% maintenance fluid was initiated. When glucose failed to recover,

the fluid was uptitratedto 12.5%. NaHCO3 was also administered for severe metabolic acidosis. Prophylactic phenobarbital was applied due to asphyxia. Cultures were obtained due to poor overall status of the patient and low platelet and high leukocyte counts were detected. Meropenem + vancomycin + intravenous immunoglobulin was administered. Posterioanterior chest X-ray also revealed an intensive infiltration. Echocardiography(ECHO) showed pulmonary hypertension+patent foramen ovule. Sildenafil was planned for pulmonary hypertension;however, it was unable to be administered, as the gastric content was dirty and contained bile. Ranitidine and metronidazole were added to the treatment. Hypoglycaemia, hyponatremia, and metabolic acidosis were stabilized after 24 hours. In the biochemical blood analysis on Day 3, 25OHvitamin D3 was <3ng/mL, Ca was 7.6 mg/dL, total protein was 3.9 mg/dL, albumin was 2 mg/dL, AST was1999 U/L, ALT was 614 U/L, LDH was 3422 U/L, creatine kinase was 7070 U/L (reference 24-170), PT time was 45 sec (reference 11-15 s), PT activity was 16% (reference 70-100), INR was 4.97, urea was 77 mg/dL, and creatinine was 2.73 mg/dL. Diffuse oedema was also detected in physical examination.

Fresh frozen plasma was administered at 10 cc/kg, human albumin was administered for two days. One-fourth of vitamin D vial was applied intramuscularly. As the levels of the hepatic enzymes were elevated, phenobarbital was discontinued. Vancomycin was switched to teicoplanin due to high urea level. In the ECHO performed on Day 6 postnatally, pulmonary hypertension was recovered. The levels of AST was110 U/L, ALT was 93 U/L, LDH was 1506 U/L. Since platelet count was detected as 31.000mm3 in the blood count, platelet suspension was administered at 10cc/kg for two days.On postnatal Day 7, repeated cranial USG showed a milimetric calcification in left ventricle and abdominal USG revealed ascites and Grade1 ectasia. Urea was 109 mg/dL and creatinine was 2.4 mg/dL.Acute renal failure secondary to severe asphyxia was considered. The urine output was good. The patient was consulted with paediatric nephrology. Since the gastric content was clear on the postnatal Day 8, enteral feeding was started minimally. On the postnatal Day 14, upon severe increase in the distention of the abdomen, the erect abdominal plain film was obtained and bowel perforation was detected. The patient was operated urgently by the paediatric surgeons. On the postoperative Day 2, his overall status deteriorated again and he was taken to operation twice. Meanwhile, gastric perforation developed (Figures 2-3 The erect abdominal plain graph of the patient, the appearance of abdominal distention). The stomach was repaired by the paediatric surgeon (Figure 4. Postoperative overall status of the patient). The patient, who started to receive enteral feeding on the postoperative Day 4, was discharged with cure on the postnatal Day 34. The hepatic enzymes and creatinekinase were normal on Day 7, the blood count returned to normal on Day 14and renal functions returned to normal on Day 21. He was followed under mechanical ventilation for 20 days.

Discussion

Perinatal asphyxia is one of the major causes of mortality and morbidity in newborns, even in developed countries, despite recent developments in neonatal care, diagnosis and treatment. According to the estimates of the World Health Organization, 3% of all infants in developing countries are suffering from asphyxia, 23% of them die due to newborn asphyxia, and in the same number of infants, serious sequelae remain [6].

It is reported that 20% of perinatal asphyxia develops in the antepartum period, 35% in the intrapartum period, 35% in the intrapartum-antepartum period and 10% in the postnatal period [7]. Although 90% of perinatal asphyxia occurs due to intrauterine and intrapartum events, asphyxia time may not be determined in many cases [6,7].In our case, we attributed the perinatal asphyxia to the advanced maternal age, emergent caesarean birth due to foetal distress, and the antepartum causes. It has been reported in the literature that cerebral palsy due to perinatal asphyxia is encountered more in males [10].Similarly, the Turkish Society of Neonatology reported that three fourth of the babies who were diagnosed with asphyxia were male [10], as in our case.

The Apgar score is often used to determine the clinical condition of the new-born at birth. The prolonged duration of the low Apgar score is associated with mortality and the increased likelihood of neurological morbidity in the surviving new-borns [8]. In term infants, if the Apgar score is between 0 and 3 in the 1st minute and it does not improve at 20 minutes, then the mortality increases from 5.6 to 59% [11,12]. The Apgar score of our patient was 0 at the first min and 2 at the fifth min.

In perinatal asphyxia, blood flow is rearranged so as to provide more oxygen support to vital organs such as the brain and heart. In this case, there may be damage to the organs such as the kidney, liver and intestine which are already affected by hypoxia. In the literature, the additional organ involvement is reported as central nervous system (72%), pulmonary (71-86%), cardiac (43 to 78%), renal (46 to 72%), liver (80 to 85%), hematological (32 to 54%), and gastrointestinal tract (29%) following the asphyxia [13,14,15].

In a study conducted by Star et al. [16] with 205 cases, the kidney was identified as the mostly affected organ with 40.5%. In the study of Shah et al. [14], they reported that the hepatic involvement was 85%, pulmonary involvement was 86% and renal involvement was 70%. In our case, the central nervous system being in the first place, pulmonary, cardiac, renal and gastrointestinal systems were affected. The most severely affected system was the gastrointestinal system in which postnatal both intestinal and gastric perforation developed secondary to mesenteric ischemia on Day 14 of asphyxia. The intestinal and gastric perforations were repaired by the paediatric surgeon.

The effect of perinatal asphyxia on bone marrow can be seen as thrombocytopenia. Thrombocytopenia may continue up to 12 hours to three days, particularly after brain damage. It may cause intracranial haemorrhage[8]. In our case, thrombocyte count was 65.000 mm3 on the first day and thrombocyte count was 31.000 mm3 on the third day. Therefore, platelet suspension was administered at a dose of 10cc/kg for two days. Since the PT time was long, fresh frozen plasma was administered for two days to prevent possible bleeding complications.

As a result of renal system involvement secondary to asphyxia, the levels of creatinine and urea may increase, oliguria-anuria may occur, fluid retention and hyponatremia due to inappropriate release of anti-diuretic hormone may develop. It is reported in the literature that as a result of liver dysfunction, the liver enzymes such as ALT, AST, LDH, particularly in the first 3-4 days may elevate, hypoglycaemia may occur and there may be prolongation of coagulation studies [8,17].

Adequate ventilation, heat, perfusion, supply of glucose, calcium and acid-base balance are the gold standard in treatment. One of the major causes of neurological damage in asphyxiated neonates is the timing of postnatal ventilation and perfusion. For this reason, it is very important to monitor the oxygen and carbon dioxide levels and keep them within the normal limits [11].

In our case, right after the resuscitation and intubation, cardiac and respiratory support was commenced. Hypoglycaemia, hyponatremia and metabolic acidosis improved after 24 hours. The infection was taken under control with the appropriate antibiotic treatment. Hepatic enzymes and creatine kinase levels were normal on Day 7 and renal functions returned to normal on Day 20.

In conclusion, perinatal asphyxia may develop in intrauterine period, during birth or in the postnatal period. Recognition of risky cases, their referral to perinatal centres, adequate antenatal care in pregnancy, foresight approach of the experienced health personnel to the new-born with asphyxia, appropriate and adequate resuscitation are crucial in terms of reducing mortality and morbidity.

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Figure-1 A chest X-ray image of the patient

(Figures 2-3 The erect abdominal plain graphy of the patient, the appearance of abdominal