Ventricular Tachycardia due to Perinatal Asphyxia

NILAY NIRUPAM, SUSHMA NANGIA AND ARVIND SAILI

From Department of Pediatrics, Division of Neonatology, LHMC and Kalawati Saran Children's Hospital, New Delhi, India.

Correspondence to:
Dr Nilay Nirupam, Senior Resident, Department of Pediatrics, Lady Hardinge Medical College, New Delhi 110001, India.
dr.nilay@yahoo.com
Received: November 17, 2013;
Initial review: December 18, 2013;
Accepted: January 21, 2014.

Background: Perinatal asphyxia is known to precipitate myocardial dysfunction, rhythm abnormalities and congestive cardiac failure. **Case characteristics**: A 2-day old neonate with perinatal asphyxia. **Observation**: He developed shock secondary to ventricular tachycardia, and required synchronized cardioversion for reversion of abnormal rhythm. **Outcome**: Reversal of arrhythmia leading to recovery. **Message**: Early identification and management of ventricular tachycardia in neonate with perinatal asphyxia can be life-saving.

Keywords: *Arrythmia, Cardioversion, Neonate.*

Symptomatic neonatal arrhythmias are rare in neonates; most are secondary to underlying congenital heart defects, metabolic disturbances, septicemia and primary rhythm abnormalities. We report ventricular tachycardia and cardiogenic shock secondary to perinatal asphyxia in a neonate.

CASE REPORT

A male neonate (birth weight 2.7 kg) was born to a primigravida mother at term gestation, and required positive pressure ventilation in delivery room for 15 minutes. The Apgar scores were 1, 3 and 5 at 1, 5 and 10 minutes of life, respectively. The cord pH was 6.64 with evidence of mixed acidemia. Subsequently baby received mechanical ventilation. He developed multiple episodes of clonic seizures at 7 hours of life. Phenobarbitone (40 mg/kg) and phenytoin (20 mg/kg) were administered for control of seizures.

At 26 hours of life, the baby developed tachycardia (220/min) and irregular heart rate. The electrocardiograph showed broad QRS complexes (duration 0.12) suggestive of monomorphic ventricular tachycardia. His capillary refill time (CRT) was around 2 seconds with good volume pulses and mean blood pressure of 39 mmHg with oxygen saturation (SpO_2) of 92%. The central venous pressure was 8 cm of water.

His hemoglobin was 19.2 g/dL, total leucocyte count 17,300/mm^3 and platelet count was 1.0×10^9 L. The arterial blood gas revealed a PaO_2 of 59 mmHg and pH of 7.33: dextrose was 73 mg/dL, serum sodium 142 meq/L, serum potassium 5.3 meq/L, ionized calcium 4.6 mg/dL, serum magnesium 2.3 mg/dL, blood urea 47 mg/dL, and serum creatinine of 1.2 mg/dL. The serum bilirubin was 2.7 mg/dL with alanine transaminase of 213 U/L, aspartate transaminase of 247 U/L and alkaline phosphatase of 312 U/L. The CPK-MB was 479 U/L (Normal 20-80 U/L) and troponin–T by card test was positive. Serum phenytoin levels were 3.2 mcg/mL (Normal 10-20 mcg/mL). The chest roentgenogram was normal.

Child did not improve to a loading dose of lidocaine (1 mg/kg) followed by infusion (25 g/kg/min), and signs of poor peripheral perfusion with shock developed. Synchronized cardioversion was given with 1 joule/kg and the heart rate reverted to 170 min. The peripheral perfusion and capillary refill time improved with return of normal sinus rhytm. Subsequently vasopressor support was tapered off. An echocardiography on day 2 of life was suggestive of right ventricular and left ventricular hypokinesia with mild tricuspid regurgitation. A repeat electrocardigram obtained on day 4 did not reveal evidence of Brugada syndrome or WPW syndrome. There was T wave inversion in leads V4 and V5, indicating ischemia.

DISCUSSION

Perinatal asphyxia is a common problem, with the incidence varying from 0.5-2% of live births [1]. The hypoxic-ischemic insult leads to organ dysfunction affecting kidney, central nervous system, heart and lungs. The incidence of clinical cardiac dysfunction in perinatal asphyxia varies from 24-31% [2]. The cardiac manifestations described include myocardial dysfunction with shock, valvular dysfunction, rhythm abnormalities and congestive cardiac failure.

Symptomatic neonatal arrhythmias are rare [3], and supraventricular tachycardia (SVT) is the commonest. Ventricular arrhythmias are often secondary to metabolic disturbances and hence potentially treatable. In a case series, nine neonates reporting to a tertiary care centre in India were identified to have arrhythmias over a 3 year period [4]. Four had primary rhythm disorder and five had secondary arrhythmias of ventricular origin, 3

attributable to metabolic disturbances, 1 to septicemia and 1 to left atrial mass lesion. Another case report highlighted the occurrence of hyperkalemia and ventricular tachycardia in an extremely low-birth-weight neonate [5]. Neonates with congenital adrenal hyperplasia/hypoglycemia with ventricular tachycardia have been described in literature [6,7]. In one case report, neonatal tachycardia associated with maternal bupivacane block was reverted with cardioversion [8]. Studies evaluating ECG and echocardiographic changes in perinatal asphyxia have reported the occurrence of ECG abnormalities in around 40% of neonates [9,10].

We attribute the occurrence of monomorphic ventricular tachycardia in present child to perinatal asphyxia. The secondary causes like hypoglycemia, hypothermia, hyperkalemia, acidosis and hypovolemia were ruled out with relevant tests. His PaO_2 was low. There was no evidence of structural heart disease and abnormal conduction pathway. Phenytoin levels were within therapeutic range. The elevated CPK-MB and troponin–T levels suggested presence of myocardial ischemia. The severe hypoxic-ischemic injury to myocardium could have precipitated a fatal arrhythmia in our neonate.

We report this case to highlight the need to screen neonates with perinatal asphyxia for rhythm irregularities, especially when they develop shock. Early identification and prompt management of any precipitating cause can be life-saving.

Contributors: All the authors were involved in management of the patient. NN: reviewed the literature and drafted the manuscript. SN: and AS: critically reviewed the manuscript. All authors approved the final version of the manuscript.
Funding: none; *Competing interests*: None stated.

REFERENCES

1. Dubin. Arrhythmias in the newborn. Neoreviews. 2000;1:146-51.
2. Perlman JM, Tack ED, Martin T, Shackelford G, Amon E. Acute system organ injury in term infants after asphyxia. Am J Dis Child. 1989;143:617-20.
3. Tanel RE, Rhodes LA. Fetal and neonatal arrythmias. Clin Perinatology. 2001;28:187-207.
4. Poddar B, Basu S, Parmar V. Neonatal arrhythmias. Indian J Pediatr. 2006;73:131-4.
5. Singh D, Dutta S, Narang A. Hyperkalemia and ventricular tachycardia in an ELBW neonate. Indian Pediatr. 2003;40:64-6.
6. Virdi VS, Bharti B, Poddar B, Basu S. Ventricular tachycardia in congenital adrenal hyperplasia. Anesth Int Care. 2002;30:380-1.
7. Chelliah YR. Ventricular tachycardia associated with hypoglycaemia. Anesth Int Care. 2000;28:698-700.
8. Heinonen KM. Rare procedures during delivery room resuscitation- cardioversion of ventricular tachycardia in an asphyxiated neonate. Intensive Care Med. 1992;18:491-2.
9. TapiaRombo CA, AlvarezVazquez E, Mendoza Zanella RM, PerezOlea V, RosasFernandez C. Detection of transitory myocardial ischemia secondary to perinatal asphyxia. Arch Med Res. 2000;31:377-83.
10. Rajakumar PS, Vishnu Bhat B, Sridhar MG, Balachander J, Konar BC, Narayanan P, *et al*. Electrocardiographic and echocardiographic changes in perinatal asphyxia. Indian J Pediatr. 2009;76:261-4.