The Story of Racing Hearts

Shabih Manzar, MD

Summary:

A case of neonatal hypoxic-ischemic encephalopathy (HIE) is presented. The findings of HIE in association with chorioamnionitis and fetal acidemia is preceded by a prolonged maternal and fetal tachycardia.

Keywords: Chorioamnionitis, Fetal tachycardia, Hypoxic Ischemic Encephalopathy (HIE)

Case:

A male infant was delivered vaginally at 393/7 weeks of gestation. Mother was an eighteen-year-old gravida1, para 1-0-0-0. She had a history of elevated blood pressures. Pregnancy medication included only prenatal vitamins. All her prenatal labs, including RPR, HIV, hepatitis B, chlamydia, and gonorrhea were negative. Significant history revealed rupture of membrane nineteen hours prior to delivery, foul-smelling amniotic fluid, and fever with a highest temperature of 102oF (38.9 °C). Fetal heart monitoring showed maternal and fetal tachycardia (Figure).

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At delivery, the infant had no cry, poor tone, and poor respiratory effort. He was taken to warmer, dried, stimulated, and bulb suctioned. He was then placed on continuous positive airway pressure (CPAP), oxygen saturations improved, and the infant was transported to the neonatal intensive care unit (NICU). Apgar scores were 5 and 8 at 1 and 5 minutes, respectively. In the NICU, while on CPAP, he developed seizures and was placed on hypothermia therapy per unit protocol. The cord blood gas showed severe acidemia (Table). The infant's physical examination was significant for tachycardia and abnormal muscle tone.

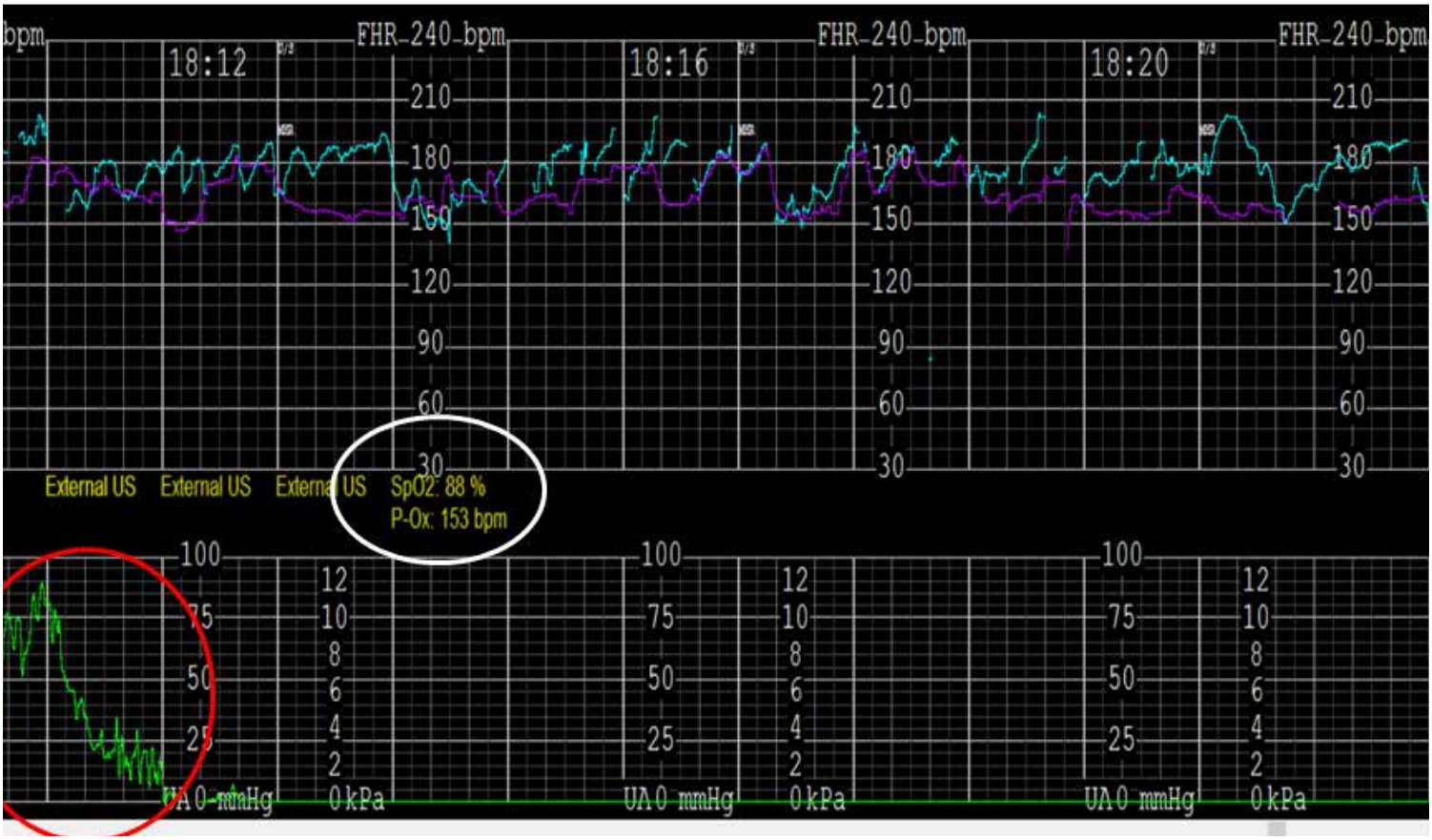

Figure 1: Title: Category II Fetal Heart Rate Strip X-axis: Time (12 minutes strip) Fetal heart rate (Turquoise color)- range 150-200 (Normal 120-160) Maternal heat rate (Purple color)-range 149-190 (Normal 70-100) Red circle: Uterine contraction White circle: Maternal heart rate and oxygen saturations

Table: Cord blood gas results

pH	7.00
pCO_2	50
pO_2	26
HCO_3	9.5
Base Access	−17.3

Vital signs showed a temperature of 100.9 °F (38.3 °C) and a heart rate of 191 beats per minute. The rest of the exam was normal.

The infant transitioned well post warming and started on PO feeds, which he tolerated well. The neurological exam at discharge was normal. He passed a pre-discharge hearing screen test. The infant was assessed by the pediatric neurologist and was sent home on phenobarbitone with follow up with his primary physician, neurology clinic, developmental clinic, and early steps intervention.

Discussion:

Fetal tachycardia was secondary to maternal tachycardia, which was secondary to high maternal temperature. With the history of prolonged rupture of membranes and foul-smelling amniotic fluid, the cause of maternal fever was suspected to be chorioamnionitis. Later, placental pathology showed stage 2, grade 2 chorioamnionitis.

The exact mechanism of fetal tachycardia resulting in acidemia is unknown; however, it could be postulated that tachycardia increases the oxygen demand of the fetal heart leading to hypoxia. Persistent hypoxemia then generates lactic acid and causes a shift in the buffer system resulting in acidemia. Tachycardia and cardiogenic shock that resulted in acidosis have been reported earlier (1).

Recently Toomey and Oppenheimer (2) showed an association between fetal tachycardia and acidemia. By using a logistic regression model, they found a tachycardia point estimate of 3.4 (95% CI 1.14-10.14). On careful observation of fetal heart rate (Figure), we noted a 12- minutes epoch of maternal and fetal tachycardia. Maternal oxygen desaturation down to 88% was also noted that could have lead to poor oxygen delivery to the fetus resulting in severe acidosis as noted in the cord blood gas.

The mechanism of fetal tachycardia secondary to maternal fever and chorioamnionitis, could be explained by the cytokine-mediated fetal inflammatory response, as described by Romero et al. (3).

In conclusion, simultaneous maternal and fetal tachycardia, when seen on antenatal cardiotocography (CTG), is an ominous sign and a potential risk factor for fetal distress and acidosis.

References:

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Disclosure: The author does not identify any relevant disclosures.

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Corresponding Author

Shabih Manzar, MD
Attending
Department of Pediatrics
Division of Neonatology
College of Medicine
Louisiana State University of Health Sciences
1501 Kings Highway
Shreveport, LA 71130
Telephone: 318-626- 4374
Fax: 318-698-4305
Email: smanza@lsuhsc.edu