Transient Prolongation of QT Interval in a Neonate

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There is limited information in the literature about sudden infant death syndrome (SIDS) and apparent life-threatening events (ALTEs) in mature neonates during the first 2 weeks of life. Lethal arrhythmias may occur in infants with QT interval prolongation due to temporary cardiac sympathetic innervation imbalance.

We report on a male neonate with a prolonged QT interval (the corrected QT interval was 0.53 seconds) who presented with ALTEs. He had frequent episodes of apnea and cyanosis with continuous bradycardia at the age of 3 days and was resuscitated. Three days later, the QTc was 0.48 seconds and the heart beat had increased spontaneously. At the age of three months, the baby was well with a normal QTc (the QTc was 0.41 seconds). The baby's father had an apparent life-threatening event at the age of three days without any significant sequelae later. No more detail information could contribute to our etiological consideration.

It is wise to be suspicious of any infant or child with a QTc (corrected QT interval) greater than 0.44 seconds, but this finding should not be used in isolation. When discovered in a newborn, a long QT interval may be the marker of a true long QT syndrome with its attendant risk of sudden death, or simply a transient electrocardiographic abnormality.

Key words: prolonged QT interval, sudden infant death syndrome (SIDS), apparent life-threatening events (ALTEs)

Sudden infant death syndrome (SIDS) and apparent life-threatening events (ALTEs) in mature neonates during the first 2 weeks of life are uncommon with an incidence of 1% to 3% [1]. ALTEs do occur in the early newborn period in a presumably low-risk term group [2]. Attempts to explain SIDS have taken into account two possible pathogenetic mechanisms. The first hypothesis suggests that newborns die of apnea which occurs during sleep. Both central and obstructive apnea have been implicated [3,4]. The second hypothesis suggests that babies die suddenly of cardiac arrhythmia. Lethal arrhythmias may occur in infants with QT interval prolongation, due to temporary cardiac arrhythmia [5-7]. We report on a neonate with a prolonged QT interval who presented with ALTEs in the first two weeks of life.

Case Report

A male neonate with a birthweight of 3,500g was born at full gestation to a 31-year-old primigravida via vaginal delivery in a community hospital. He had frequent apnea and cyanosis in the nursery at the age of 3 days. He was resuscitated and intubated. Due to persistent apnea and bradycardia (around 60 to 70 beats per minute) and failure to wean from the ventilator, he was transferred to our neonatal intensive care unit. After admission, he had one episode of apnea, hypoventilation and continuous bradycardia around 70 to 85 beats per minute with a prolonged QT interval (the corrected QT interval was 0.53 seconds; Fig. 1). There was no family history of arrhythmia. Three days later, the QTc was 0.48 seconds (Fig. 2). At that time, the heart beat had increased to 110 beats per minute. The hemogram, brain echography, echocardiography, EEG and auditory brain stem response were within normal limits. Arterial blood gases were PH 7.447, PaCO2 20.0 mmHg, PaO2 75.7 mmHg, base excess *7.0 mmol/L and bicarbonate 13.9 mmol/L. Electrolytes levels were normal (sodium 138 mEq/L, potassium 5.0 mEq/L, chloride 110 mEq/L and ionized calcium 5.05 mg/dl). Creatine kinase was 220 u/L with CK-MB 23u/L. The parent’s EKG studies were normal with 0.41 seconds and 0.42 seconds of corrected QT interval. According to the baby’s grandmother, the father had an apparent life-threatening event at the age of three days. After the resuscitations of intubation and oxygen supplement, he was extubated one week later without significant sequelae. His heart beat was 130 beats per minute and his corrected
The QT interval was 0.44 seconds. The baby was discharged in good health without any medications two weeks later. At the age of three months, he had a normal QTc (QTc was 0.41 seconds).

Discussion

According to Bazett's formula, the QTc should not exceed 0.425 seconds except in infants. A QTc up to 0.49 seconds may be normal in the first 6 months of life. It is wise to be suspicious of any infant or child with a QTc greater than 0.44 seconds, but this finding should not be used in isolation. QT prolongation could be due to hypocalcemia, hypothermia, hypothyroidism, cerebral vascular accident, antiarrhythmia drugs, myocardial damage and native long QT syndrome. A QTc over 0.60 seconds is frequently associated with severe ventricular arrhythmias and second-degree AV block. Special attention should be given to infants who have a very long QT interval and major ST segment change; the risk of sudden death in these infants is high, especially in the first week of life [7]. In our case, the QTc interval was 0.53 seconds with persistent bradycardia and hypoventilation. Cases of temporary prolongation of the QT interval have been reported, sometimes with documented life-threatening arrhythmias [7]. They can be transient, benign or have a high correlation with lethal arrhythmias. The QT interval might return to normal within the first month. Prolongation of the QT interval in neonates may be transient or may present as an early form of the long QT syndrome. The length of the QT interval in neonates may be transient or may present as an early form of the long QT syndrome. The QT prolongation may be secondary to a variety of medications, metabolic disorders, and other medical conditions [8]. The ischemic changes of myocarditis and cardiomyopathy can cause a similar clinical picture of long QT syndrome. Our patient had a normal heart structure and cardiac function. Symptoms include sudden death, seizures and syncope [9]. Schwartz hypothesized that one or more of the following is true: (1) the long QT syndrome is hereditary; (2) birth defects occur in the brain, spinal cord, thoracic sympathetic ganglia, or adrenergic terminals in the myocardium; (3) right-sided sympathetic activity is decreased; or (4) there are developmental delays in right-sided sympathetic activity [5]. Schwartz favors the latter hypothesis. Prolongation of the QT interval, at some time of life, may be a marker of sympathetic imbalance and would allow early identification of some babies at risk. The genesis of SIDS may be considered multifactorial. Different causes and mechanisms are involved. It is likely that the majority of the proposed hypotheses account for a few cases. Respiratory death is slow and allows a few minutes to observe apnea, cyanosis, and struggle, whereas arrhythmic death is instantaneous and silent [10]. Prolongation of the QT interval in the first week of life is strongly associated with SIDS. Neonatal electrocardiographic screening may permit the early identification of a substantial percentage of infants at risk for SIDS, and preventive measures can be taken [11]. In considering the cause and effect of SIDS and prolonged QT interval, it was difficult to differentiate what was the primary cause in our case. We couldn’t exclude the possibility of postresuscitation effect. The father had an ALTE as a newborn and we could not find any significant cause of the ALTE. We more favor the prolongation of QT interval as primary pathogenesis.

Figure 1. Electrocardiogram at 6 days of age. QT prolongation was noted with a QTc of 0.53 seconds.

Figure 2. Electrocardiogram at 10 days of age. QTc was 0.48 seconds.
In one study, a neonate who had presented with a sustained irregular heart rate during labor was found to have QT prolongation and repetitive polymorphic ventricular arrhythmias [12]. Sinus bradycardia in an otherwise normal fetus may be a symptom of long QT syndrome. With prolonged QT syndrome, early diagnosis and therapy are crucial. The β-blocking agents (e.g., propranolol) alone or in combination with left cardiac sympathetic denervation or permanent cardiac pacing are known to diminish the risk for sudden death [13]. As neonates are particularly at risk of sudden death, early introduction of permanent cardiac pacing should be considered when propranolol failure is suspected [8,10].

We feel there is a high correlation between prolonged QT interval and ALTEs. It’s uncommon to observe that the prolonged QT interval on the electrocardiogram in certain individuals is associated with an increased incidence of life-threatening arrhythmias and sudden death. When discovered in a newborn, a long QT interval may be the marker of a true long QT syndrome with the risk of sudden death, or simply a transient, benign electrocardiographic abnormality. The relationship between long QT syndrome, various conduction disturbances and late potentials will require further scrutiny.

References