OLGU SUNUMU / CASE REPORT

Prematüre yenidoğanda 2:1 atriyoventriküler blok ve konjestif kalp yetersizliğinin nadir bir nedeni: Hipokalsemi

A rare cause of 2:1 atrioventricular block and congestive heart failure in preterm infants: Hypocalcemia

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Özet– Atriyoventriküler blok yenidoğan döneminde nadir görülür ve genellikle doğumsal kalp hastalığına ve annenin otoimmün hastalığına ikincil olarak gelişir. Bu yazıda, hipoklasemiye bağlı 2: 1 atriyoventriküler blok ve konjestif kalp yetersizliği gelişen bir prematüre bebek olgusu sunuldu. Olguda atriyoventriküler blok ve konjestif kalp yetersizliği bulguları intravenöz olarak verilen %10'luk kalsiyum glukonat infüzyonunu takiben hızlı bir şekilde düzeldi. Bu nedenle atriyoventriküler blok ve kalp yetersizliği gelişen yenidoğanlarda serum kalsiyum düzeyinin ölçülmesini önermekteyiz.

Summary– Atrioventricular (AV) block in the neonatal period is a rare disorder. It is frequently associated with underlying structural congenital heart disease and maternal lupus. Presently a premature baby who developed 2:1 AV block and congestive heart failure due to hypocalcemia. was presented. Dramatic clinical improvement was observed following treatment with intravenous 10% calcium gluconate. Therefore, it is suggested that serum calcium level of newborns with AV block and congestive heart failure be measured.

Atrioventricular (AV) 2:1 block in infants which causes bradycardia requires emergency intervention. This type of arrhythmia may present with severe congestive heart failure or may be totally asymptomatic in newborns and infants.

Abbreviations

AV	*Atrioventricular*
Ca	*Calcium*
EF	*Ejection fraction*
EKG	*Electrocardiography*
EKO	*Ekokardiyograf*
FS	*Fractional shortening*
PHR	*Peak heart rate*

During neonatal period bradyarrhytmia due to electrolyte disorders is not frequently seen. During neonatal period hypocalcemia is a rarely seen cause of cardiac arrhytmia. In the literature very limited number of case reports with 2:1 AV block have been cited. In hypocalcemia, ventricular arrhytmias associated with prolongation of QT interval, alterations in QRS, and ST can be observed on electrocardiograms (ECG).

In this paper, a 2-day-old infant who developed 2:1 block and congestive heart failure due to hypocalcemia and treated successfully with calcium (Ca) gluconate infusion was presented.

CASE PRESENTATION

A 2-day-old infant monitored in a neonatal intensive care unit with diagnoses of premature birth, and transient tachypnea of newborn was consulted to our clinic because of bradycardia. On physical examination of the infant whose mother had not any known disease, revealed moderately well general health state. Infants blood pressure, and peak heart rate were 60/40 mm Hg and 75 bpm, respectively. During monitorization a 1/6 systolic murmur was heard over mesocardiac focus. On electrocardiography of the patient with 2:1 AV block prolongation of QT (QTc 530 msec), increased atrial (166/min) and ventricular rates (88/min) were detected (Figure 1).

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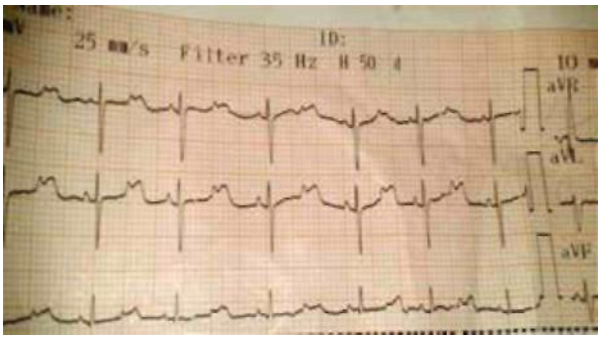

Figure 1: Electrocardiogram before calcium gluconate therapy. 2:1 atrioventricular block

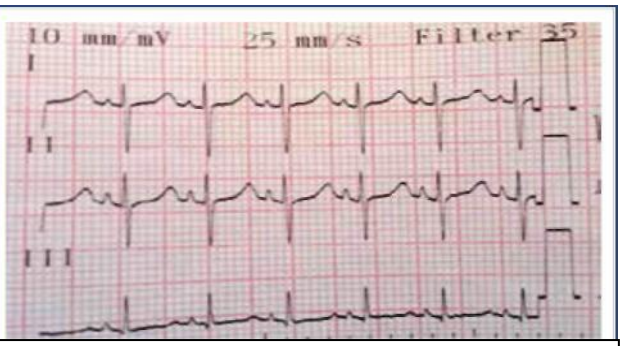

Figure 2: Electrocardiogram following calcium gluconate therapy; peak heart rate 166 beat/min, PR: 0.08 msec; QTC: 450 msec

Echocardiographic evaluation of the patient without structural heart disease revealed left ventricular ejection fraction (EF) of 52%, and fractional shortening of (FS) of 27 percent. Laboratory test results were as follows: hemoglobin 12.9 g/dL, fasting blood sugar (FBG) 74 mg/dL, calcium 4.8 mg/dL, magnesium 2 mg/dL, albumin 3.7 g/dL, phosphorus 9.9 mg/dL and alkaline phosphatase 707 U/L (n: 48–406 U/L). Renal and hepatic function test results were within normal limits. Parathormon 102 pg/mL (n: 12–65 pg/mL), 25-OH D vitamin level 29.1 ng/mL (n: 7.6-75 ng/mL) were also measured. For the treatment of hypocalcemia Ca gluconate (10%) at a dose of 1 ml/kg was diluted at a rate of 1/1 and infused within 20 minutes. Treatment of congestive heart failure was initiated with dopamine infusion at a dose of 8 mcg/kg/min. At 12. hour of the treatment blood pressure and heart rate of the case were 85/60 mmHg and 165 bpm, respectively and clinical symptoms of congestive heart failure improved. During monitorization ECG findings of the case returned to normal and on control echocardiograms left ventricular EF, and FS were measured as 75 and 36 %, respectively (Figure 2). On the 5. day of his hospitalization serum Ca level was 10 mg/dL which necessitated cessation of infusion therapy.

DISCUSSION

Cardiac arrhytmias are rarely seen during neonatal period. Infants monitorized in the neonatal intensive care units, generally hypoxia and serum electrolyte disorders are responsible from bradycardia. During this period supraventricular tachycardia is more frequently seen than bradycardia. Mostly potassium-related electrolyte disorders induce arrhytmias in newborns. Hypocalcemia is among very rarely seen causes of arrhytmia.[1]

Congenital heart block is a rarely seen condition. It generally develops secondary to cardiac anomalies, and maternal autoimmune disease. In our case cardiac anomaly did not exist and findings of autoimmune disease of both mother and her child were not found. AV 2:1 block with very high mortality rates may develop secondary to hypocalcemia and prolonged QT interval. Since cases with AV blocks related to hypocalcemia can be treated, it is important to make a differential diagnosis among other causes of AV block.[2]

In infants followed up in the intensive care unit, disorders of calcium metabolism are frequently observed. Transient physiologic hypoparathyroidism is frequently seen in newborns and it can explain hypocalcemia in some cases. Early-onset hypocalcemia generally develops within the first 7 days of the life of premature infants. In premature infants, incidence of hypocalcemia is increased related to respiratory distress syndrome (RDS). Deficient oral feeding in premature infants, being an infant of a diabetic mother and perinatal asphyxia are among etiologicd factors which increase risk of hypocalcemia. In a study by Oppe and Redstone higher incidence of early-onset hypocalcemia has been demonstrated.[1] As was seen in our case especially in infants with respiratory distress syndrome, increased incidence of hypocalcemia has been reported related to increased endogenous phosphate release due to both deficient intake and catabolism. In premature infants exposed to hypoxia and anoxia thyrocalcitonin release increases which contributes to prolongation of hypocalcemia .[1]

Hypocalcemia is a very rarely seen etiology of reversible heart failure and dilated cardiomyopathy. Although it is most frequently reported in adults, in children cases with dilated cardiomyopathy related to hypocalcemia secondary to rachitism have been also reported. Only a few cases of dilated cardiomyopathy have been reported.

Generally these patients were previously refractory to conventional treatment for heart failure, and detailed examinations revealed the presence of hypocalcemia.[3–8] In hypocalcemia since contractibility of myocardium decreases, congestive heart failure may develop without any underlyingetiological factor. In chronic hypocalcemia, myocardial contractibility is severely impaired leading to aggravation of congestive heart failure. This condition is very rare and it is completely resolved with replacement of calcium deficiency. In our case symptoms of heart failure rapidly resolved following Ca gluconate therapy.

The correlation between hypocalcemia, and prolonged QT interval was firstly described by Carter and Andrus in 1922 and Fishbein et all. published a case which demonstrated the correlation between hypocalcemia and heart block.[1,10] Also other case reports demonstrating prolongod QT and QTc intervals and changes in T wave associated with hypocalcemia have been published.[1,9]

In our case sisnce calcium infusion dramatically resolved symptoms of AV block and congestive heart failure, we thought that this problem was related to hypocalcemia.

Only a few cases that developed hypocalcemia-related AV block have been reported so far. Only 10 newborns with 2:1 AV block and hypocalcemia have been cited in the literature. In the literature 3 separate case presentations on six, two, and one patient have been published. As was the case with our patient, 2:1 AV block, prolongation of QT interval and hypocalcemia were detected in a premature baby on 2. or 3. day of his life.[11] In a case without cardiac defect Ca gluconate administration resolved bradycardia and ECG tracings returned to normal .[12]

Transient transcutaneous pacemaker has been used to manage bradycardia in children and adults. [13] However in our case since AV block was rapidly returned to normal sinus rhythm following calcium infusion, any invasive intervention was not required.

Conclusion

If symptoms of bradycardia, and heart failure are found in premature infants, cardiac rhythm should be controlled with ECG and serum calcium level should be checked. If these findings are associated with prolongation of QT interval in infants with atrioventricular block and heart failure, hypocalcemia should be absolutely thought as an etiologic factor.

AV block and heart failure secondary to hypocalcemia can be treated rapidly with intravenous calcium infusion Treatment of the patient can be possible without resorting to many examinations and interventions.

Conflict of interest: none declared.

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Anahtar sözcükler: Atriyoventriküler blok; hipokalsemi; prematüre yenidoğan.

Keywords: Atrioventricular block; hypocalcemia; premature baby.