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Cardiac Hypertrophy Due to Hydrocortisone Treatment in a Preterm Infant: A Case Report

CESE REPORT
OLGU SUNUMU

Preterm İnfantta Hidrokortizon Tedavisine Bağlı Gelişen Kardiak Hipertrofi: Olgu Sunumu

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ABSTRACT
ÖZET

Infants with chronic lung disease are at greater risk for pulmonary compromise in childhood. Hydrocortisone has been studied in the treatment of chronic lung disease. A preterm infant, with a gestational age of 26 weeks, was admitted to the neonatal intensive care unit. On the 28th postnatal day, the patient still had oxygen requirement (FiO_2: 0.40) and hydrocortisone treatment was given for seven days. After the treatment the echocardiography revealed a remarkable thickening of the left ventricle and interventricular septum. The case reported here is the first preterm infant with cardiac hypertrophy due to hydrocortisone administration for treatment of chronic lung disease.

Key words: Hydrocortisone, side effect, cardiac hypertrophy

Kronik akciğer hastalığı olan infantlar çocukluk çağında solunum fonksiyonlarının bozulması açısından büyük risk taşırlar. Hidrokortizon kronik akciğer hastalığının tedavisinde de kullanılmaktadır. Gestasyon haftası 26 olan preterm bir yenidoğana yenidoğan yoğun bakım ünitesindeki takibinde postnatal 28. günde oksijen ihtiyacı (FiO_2: 0,40) olması nedeniyle hidrokortizon tedavisi yedi gün uygulandı. Tedavi sonrası kontrol ekokardiyografisinde dikkate değer sol ventrikül ve interventriküler septum hipertrofisi saptandı. Rapor edilen bu vaka hidrokortizon kullanımına bağlı kardiyak hipertrofi gelişen ilk preterm infanttır.

Anahtar kelimeler: Hidrokortizon, yan etkiler, kardiyak hipertrofi

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Introduction

Infants with chronic lung disease (CLD) are at risk for pulmonary compromise during childhood, rehospitalization, neurodevelopmental delay, and late mortality (1). Corticosteroids are widely used to treat CLD (2). Dexamethasone treatment is known to be effective in the treatment of CLD; however, it is associated with many side effects. Due to the serious side effects of dexamethasone there is an ongoing search for other corticosteroids with similar effectiveness, but fewer side effects. Hydrocortisone (HC) has been used in randomized trials for the treatment of CLD since the 1970s; however, the long-term effects are still being evaluated (3). Herein we report a possible cardiac side effect of HC used to treat a CLD patient, which has not been previously reported in preterm infants.

Case Report

A female preterm infant (gestational age: 26 week; birth weight; 760 g) was born to a 28-year-old mother who was pregnant for the first time. Prenatal history was unremarkable, except for preterm premature rupture of the membranes (PPROM) at 26 weeks of gestation. History of consanguinity, gestational diabetes mellitus, hypertrophic cardiomyopathy, and inherited metabolic disease was negative. Vigorous resuscitation and intubation were required during the delivery, and Apgar scores were 4 and 7, at 1 and 5 min, respectively. The patient was admitted to the neonatal intensive care unit (NICU) and received surfactant treatment for respiratory distress syndrome (RDS). Echocardiography screening for patent ductus arteriosus on the third postnatal day was normal. On the 28th postnatal day, the patient was still on continuous nasal positive airway pressure and required 40% O_2 and we therefore administered HC as rescue treatment.

Baseline evaluations before HC treatment, including echocardiography and laboratory findings (cell blood counts, blood gases, electrolytes, and renal and hepatic function tests), were normal. Hydrocortisone (1 mg $kg^{-1} \cdot d^{-1}$) was administered in 2 doses. As the patient's O_2 requirement decreased, HC treatment was gradually decreased over 7 days. When the patient was reevaluated for the possible side effects of corticosteroid treatment, echocardiography showed remarkable thickening of the left ventricle and interventricular septum (7.3 mm in an 800-g infant) with no left ventricle outflow obstruction (Figure 1). The only pathologic parameter associated with cardiac hypertrophy on physical examination was hypertension (84/42 mmHg). No other side effects of corticosteroids, such as hyperglycemia, fluid retention, or electrolyte imbalance, were observed.

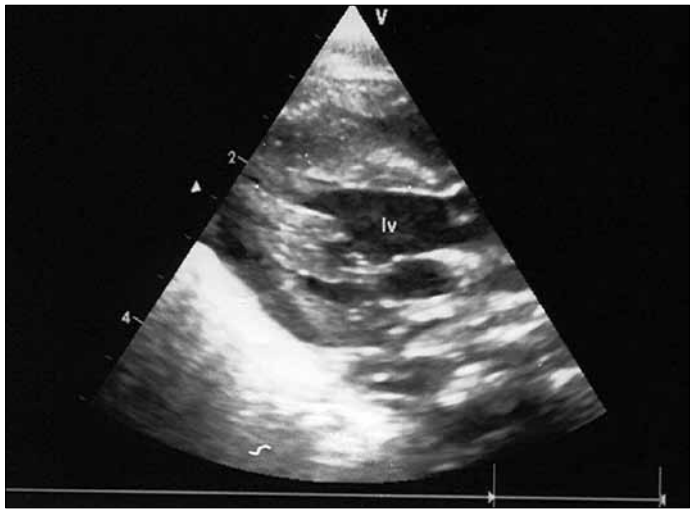

Figure 1. Echocardiography after Hydrocortisone treatment shows remarkable thickening of the left ventricle and interventricular septum
IV: Interventricular septum

The patient was monitored regularly based on echocardiographic evaluations and a gradual reduction in cardiac hypertrophy was recorded during the following 4 weeks. She was discharged on postnatal day 65 without the need for oxygen, following normal cardiac evaluation results.

Discussion

Hypertrophic cardiomyopathy is a well-known complication of steroid therapy in premature infants, which develops in response to steroid courses of ≥2-3 weeks duration. Steroid-induced hypertrophic cardiomyopathy is characterized by concentric thickening of the interventricular septum and free walls of the ventricles, as well as a reduction in intracavity dimensions. Prolonged steroid therapy in infants and premature neonates induces increased protein synthesis in myocytes, leading to hypertrophy. Such changes are transient in premature infants and resolve within 12 weeks after discontinuation of steroids (4).

It is estimated that <50% of extremely low birth weight infants receive dexamethasone in the NICU (5). The side effects of dexamethasone in newborns are well documented, as are its beneficial effects on respiration in at-risk infants (6). Cardiac hypertrophy is a known side effect of dexamethasone and maximal left ventricle hypertrophy usually develops by the end of the second week, with resolution often beginning during weaning from the drug. Although there are many reports of cardiac hypertrophy, only 2 pediatric cases of cardiac hypertrophy caused by HC have been reported. Scirè et al. (7) reported a 1-month-old infant treated with high-dose HC therapy for congenital adrenal hyperplasia and showed marked left ventricular hypertrophy after 10 months of the treatment. Secondly, Conwell et al. (8) reported a 13-year-old girl with clinical features of Addison's disease who developed acute cardiac failure after initiation of HC treatment. The patient's cardiomyopathy improved over 1 week and her condition then remained stable with oral steroid replacement therapy. Both patients had cardiac hypertrophy after the newborn period and Scirè et al.'s (7) patient received long-term HC treatment.

Another common cause of neonatal hypertrophic cardiomyopathy is commonly described in infants of diabetic mothers in whom hyperinsulinemia may contribute to the development of transient asymmetrical septal hypertrophy (9). There was no maternal history of gestational diabetes mellitus. Other associations with neonatal HCM include several inborn errors of metabolism, such as glycogen storage disease type II, GM1 gangliosidosis and mucolipidosis type II, rhabdomyomatous tumor infiltration, Noonan syndrome and Beckwith-Wiedemann syndrome. A complete metabolic screen was not performed in this infant since there was no evidence of hypoglycaemia or acidosis, and cardiac hypertrophy resolved spontaneously. In addition, there was no family history of primary HCM and this would be an unusual presentation of this condition in the early newborn period (10).

To the best of our knowledge, the present case is the first preterm infant with cardiac hypertrophy due to HC for treatment of CLD. We observed hypertension in addition to cardiac hypertrophy in our patient. Marked left ventricular hypertrophy mimicking hypertrophic cardiomyopathy may be related to the hypertensive state induced by steroid therapy or to the direct anabolic effect of corticosteroids on myocardial cells (7). Both hypertrophy and hypertension in the presented case resolved over time; therefore we consider that our patient's clinical picture strongly suggests that there was a close relation between the 2 pathologies.

Conclusion

Based on our observations in the presented case, we propose that preterm infants receiving HC treatment should be echocardiographically monitored for the reported side effect.

Conflict of Interest
No conflict of interest was declared by the authors.

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Authors' contributions: Conceived and designed the experiments or case: YUS, OE. Performed the experiments or case: NA. Analysed the data: YUS, SSO. Wrote the paper: YUS, OE, SSO, NA, UD. All authors have read and approved the final manuscript.

Çıkar Çatışması
Yazarlar herhangi bir çıkar çatışması bildirmemişlerdir.

Hakem değerlendirmesi: Bağımsız hakemlerce değerlendirilmiştir.

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