

Betamethasone Induced Hypertrophic Obstructive Cardiomyopathy in Infant

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ABSTRACT

Hypertrophic obstructive cardiomyopathy (HOCM) is a major potential complication of dexamethasone therapy in preterm infants. HOCM may also be caused by Betamethasone in infants if given for longer time. We present a case of a 3 month old male baby who was prescribed betamethasone by quack for 1 month for treatment of cough and cold. The baby developed HOCM and systolic anterior motion of mitral valve which completely resolved after stoppage of betamethasone in 12 weeks.

KEY WORDS: betamethasone, hypertrophic cardiomyopathy (HOCM), infant

INTRODUCTION:

Hypertrophic cardiomyopathy (HCM) is a heterogeneous, relatively common, and potentially life-threatening form of cardiomyopathy. The causes of HCM are heterogeneous and include inborn errors of metabolism, neuromuscular disorders, syndromic conditions, and genetic abnormalities of the structural components of the cardiomyocytes^[1]. Unfortunately, use (and misuse) of several drugs and medications are well known causes of injury to cardiac muscles^[2].

Steroid like dexamethasone, is widely used in cases of chronic lung disease (CLD) for both prevention as well as management in infants. Case reports are published mentioning the use of steroids in decreasing the duration of ventilator dependence. Prolonged use of these steroids leads to development of HCM^[3]. When these steroids are stopped, there occurs the reversal of echocardiographic changes over 2-3 weeks^[4]. Most of the published reports on drug induced HCM mention dexamethasone as a cause. We present a case report of betamethasone induced HCM which was totally reversed over 3 months.

CASE REPORTS:

A 3 months old male baby presented with

tachycardia, tachypnea and fever since 3 days. There was history of 2 episode of upper respiratory tract infection in last 2 months. Patient visited local practitioners where he was prescribed oral medications. On examination child was having cushingoid facies and respiratory distress. BP 98/52, HC 39.5 cm, Wt 7 kg (>90th centile), bilateral breath sounds equally heard with coarse crepitations, systolic murmur, soft abdomen with palpable liver of 4 cm below costal margins, spleen not palpable. History revealed that the child was prescribed symptomatic treatment, oral antibiotics and oral betamethasone drops by local quacks. Parents also revealed that as the symptoms were frequent therefore they were advised to take oral betamethasone drops daily for 1 month. Baby started gaining weight which parents considered it to be normal. There was no history of previous hospitalisation.

On investigation complete blood picture, liver and kidney functions and serum electrolytes, urine routine microscopy were all within normal limits. Serum cortisol 17.07µg/dL, serum cholesterol 295 mg/dL, ultrasound abdomen and electrocardiograph were within normal limit. Echocardiographic findings were showing concentric left ventricle (LV) hypertrophy (symmetrical), inter ventricular septum (IVS), posterior wall (PW) 10 mm, peak left ventricular outflow tract gradient 24 mmHg, systolic anterior motion of mitral valve, moderate mitral regurgitation with normal biventricular functions. During course of stay patient was managed symptomatically and betamethasone was stopped.

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Patient was discharged after 5 days and kept under follow up. A repeat echocardiography after 12 weeks showed reversal of HCM and normal study (right ventricle 13, aorta 11, left atria 15, PW/IVS 04/03, Left ventricle internal diameter end diastole and end systole were 17 and 14 respectively, ejection fraction 60%).

DISCUSSION:

Steroid-induced hypertrophic cardiomyopathy is a distinct clinical and echocardiographic entity for infants on steroids. Some insights into the underlying mechanisms have been provided by animal experiments^[5]. For premature infants hypertrophic cardiomyopathy is a known complication of steroid therapy, developing with steroid courses of 2–3 weeks duration or longer^[7]. However, hypertrophic cardiomyopathy has not been reported in the context of betamethasone, which is frequently used by local quacks as a part of cough remedies. Myocytes respond by increase in protein synthesis for prolonged use of steroid therapy, leading to hypertrophy of myocardium in premature neonates and young infants. Such changes are transient in premature infants, as they resolve within 1–2 weeks after discontinuation of the steroids^[7]. In older children it may take longer for ventricular hypertrophy to resolve, therefore there is need for longer follow-up^[8]. Alpert reported HCM in a 14-month-old baby on high-dose steroids for hypsarrhythmia and infantile spasms, with the changes regressing in 1 year on withdrawal of steroids^[9]. Septal hypertrophy causing venturi effect which draws the anterior mitral leaflet against the ventricular septum, resulting in outflow obstruction and a pressure gradient develops. The same phenomenon, systolic anterior motion (SAM) of mitral valve was present in our case. SAM can be seen in steroid-induced hypertrophic cardiomyopathy^[10].

Balys et al reported a 4-month-old baby who developed HOCM following dexamethasone treatment for subglottic stenosis. The baby had clinical worsening hemodynamics which improved on stoppage of the medicine^[11]. Scire et al reported marked LVH mimicking HCM in an child treated with steroids for congenital adrenal hyperplasia (CAH), in which the LVH regressed on reducing the drug dosage^[12].

CONCLUSION:

Infants receiving glucocorticoids should undergo regular echocardiographic monitoring, with high index of suspicion for hypertrophic

cardiomyopathy. Stopping of steroids after diagnosing HCM would be life saving.

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Cite this article as: Patil R, Khan A. Betamethasone Induced Hypertrophic Obstructive Cardiomyopathy in Infant . *PJSR* ;2019;12(1):59-61.
Source of Support : Nil, Conflict of Interest: None declared.