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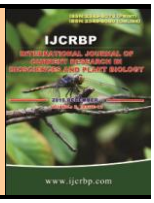

Case Report

Effect of Sildenafil on Pulmonary Artery Hypertension with Mild Triscuspid Regurgitation - A Case Report

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Abstract

The present work reports the case report of an 8 months old male child admitted during April 2014 with complaints of fever since 10 days and difficulty in breathing since 4 days. The hypothesis of this case study was that the administration of Sildenafil would improve haemodynamics in children with mean pulmonary arterial pressure. The administration of 0.5mg/kg/day of Sildenafil showed improvement in pulmonary conus. Long term effects of the drug are required to assess the safety and efficacy of Sildenafil in this treatment.

Keywords

Haemodynamics

Pulmonary hypertension

Sildenafil

Introduction

Pulmonary hypertension (PH) is a haemodynamic and pathophysiological condition defined as an increase in mean pulmonary arterial pressure (PAP) 25 mmHg at rest as assessedby right heart catheterization (Hatanos, 1975; D'Alonzo et al., 1991). The worse prognosis in children with a median survival estimated at 10 months.[2] Pulmonary artery hypertension (PAH) is a progressive and fatal disease (Humpl et al., 2005).

The exact incidence and prevalence of PH in children is not known. Drugs for treatment of Pulmonary Hypertension includes Calcium channel blockers, endothelin receptor antagonists, phosphodiesterase 5 inhibitors, prostanoids etc. (Galie et al., 2009). Since the pulmonary vasculature contains substantial amounts of phosphodiesterase type-5 the potential clinical benefit of phosphodiesterase type-5 inhibitors has been investigated in PAH. In addition, phosphodiesterase type-5 inhibitors exert antiproliferative effects (Wharton et al., 2005; Tantini et al., 2005). Sildenafil is a type 5 phosphodiesterase inhibitor and pulmonary vasodilator (Humpl et al., 2005). Therefore, we hypothesized that sildenafil would improve haemodynamics in children with PAH.

Case report

An 8 months old male child admitted on 1st April 2014 with complaints of fever since 10 days and difficulty in breathingsince 4 days. He was taking

treatment from some local practioner in the form of antibiotics for lower respiratory tract infection and was oxygen dependent since then. Rest of history was normal.

On admission vitals were, HR-146/min, RR-56/min on 6 l/min of oxygen, TEMP- 99.4% and SPO_2- 94% on O_2. On systemic examination R/S- chest was symmetrical with nasal flaring and visible intercostal and subcostal recessions and added sounds in the form of wheeze and crepitations.

Rest of systemic examination was normal. Treatment was started in the form of antibiotics and oxygen and nebulisation with bronchodilators. Reports – Hb-7.1 g%, TLC- 7000 cells/cumm, DLC- $P_{42},L_{55}E_2M_1$ and PBF- no toxic granules and band cells seen. CRP was negative and electrolytes were normal. X-ray done on 2nd April which showed prominent pulmonary conus. ECG showed right ventricular hypertrophy and ECHO revealed Mild TR with Pulmonary Artery Hypertension.

On 2nd April 2014 child severe respiratory distress as child was on breast feeds. In view of aspiration pneumonia child was kept on ventilator for 5 days. Tab Sildenafil was started on 2nd April 2014 @ 0.5mg/kg/day. Later on X-ray chest after starting Sildenafil was done which showed improvement in pulmonary conus. Soon child became cheerful, playful and regained his appetite and permitted discharge.

Discussion

Pulmonary hypertension is characterized by elevated PAP and secondary RV failure (Tsapenko et al., 2008). the exact underlying mechanism of pulmonary hypertension are still poorly understood, however, it is hypothesised that pulmonary medial hypertrophy and endothelial dysfunction leads to impaired production of vasodilators such as nitric oxide and prostacyclin and an increased expression of vasoconstrictors such as endothelin-1(Leiboviten et al., 2007). The majority of patients present with PH associated with CHD or idiopathic/heritable forms. Dyspnoea, fatigue, and failure to thrive are common symptoms (Konduri and Kim, 2009).

Sildenafil citrate is highly selective inhibitor of phosphodiesterase type5 (PDE5). It induces pulmonary vasodilation by increasing intracellular cyclic guanosine monophosphate (cCMP) concentrations (Leiboviten et al., 2007). We choose to treat our patient with Sildenafil Citrate because it stabilizes second messenger of endogenous nitric oxide cGMP. It is a potent, specific, orally available PDE_5 inhibitor and minimal side effects. It has shown efficacy causing pulmonary vasodilatation within 60mins and an RCT is underway to define dose and efficacy (D'Alonzo et al., 1991). No failure of drug use or side effects were seen in our patient. Its use is limited in children. Long term efficacy and safety of Sildenafil therapy in PAH in children requires further study based on large and well designed randomised controlled trialsas it is an experiment drug which requires more study to validate the safety, efficacy and dosage in paediatric population (Wang et al., 2014).

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