USE OF IVABRADINE IN SUBACUTE EPISODE OF HEART FAILURE: IS IT FEASIBLE IN CHILDREN?

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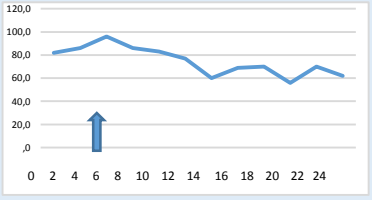
Recently, ***ivabradine*** has been shown to be useful in decreasing heart rate (HR) and improving left-ventricular (LV) function in pediatric population with chronic heart failure (HF). No reports are available about safety and efficacy of Ivabradine in acute phase of HF in children. We report the cases of 2 patients.

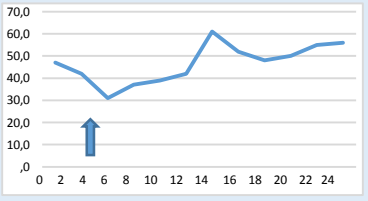
CASE 1

A girl of 12 years old with dilated/LV non-compaction CMP and Wolf-Parkinson-White syndrome was admitted to our Pediatric Intensive Care Unit (PICU) because of acute HF. At admission, she presented clinical signs of hypoperfusion and pulmonary congestion (cold and wet HF phenotype). NT-proBNP was 10650 pg/ml. Echocardiography showed a markedly dilated LV (end-diastolic, end-systolic LV diameter Z scores respectively +7SD and +10SD) with severe systolic dysfunction (LVEF 17%), and moderate-severe mitral valve regurgitation. Right ventricle function and dimension, and estimated pulmonary pressure were normal. She required a combined inotrope support and iv high dose of diuretics. After inotrope weaning, ultrasounds confirmed a LVEF of 14%. Ivabradine was started at 2,5 mg twice a day in PICU setting. After Ivabradine start, HR decreased from 97 to 70 bpm in 24 hours. The hourly hemodynamic monitoring by MOST care® showed a significant increase in cardiac index (from 2.0 to 3.0 l/min/m2), and stroke volume (from 35 to 60 ml). No changes in blood pressure have been observed.

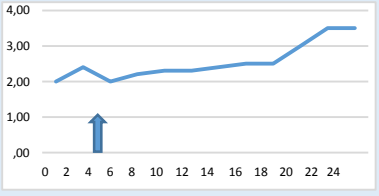
Heart rate



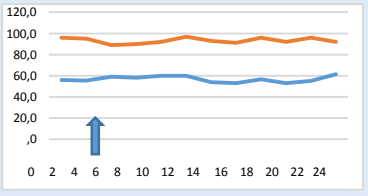
Stroke volume



Cardiac Index



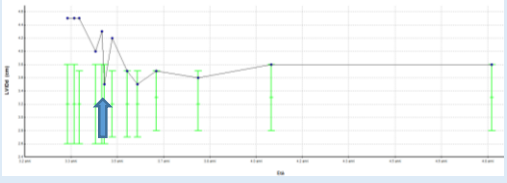
Systolic and diastolic blood pressure



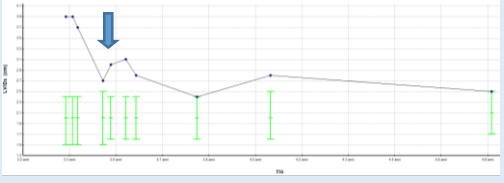
CASE 2

A 3.4y girl presented with cardiogenic shock in severe DCM (LVEF 15%). She required triple inotrope support and invasive respiratory assistance. Serum virology revealed the presence of HHV6 and Parvovirus B19 genome. Genetic searches excluded genomic component. Immunoglobulin infusion and Ganciclovir were started. Inotrope weaning failed, and she was placed on urgent heart transplantation list. Extubation was reached after 2 months in PICU. After 6 months reoccurring HF symptoms and severe systolic dysfunction persisted defining a stage D HF: inotrope dependence. Cardiac MR showed no fibrotic replacement. A HR reduction strategy with **ivabradine** (started at 0.05 mg/kg/dose, up titrated to 0.3 mg/kg/dose), in addition to standard anti HF therapy (beta blocking, ACE inhibitor and anti aldosteronic) was adopted. HR was slowly reduced without hypotensive episodes. After 8 months, the baby was stabilized and discharged. After 1 year, a complete LV recovery was obtained: LV end-diastolic and end-systolic diameter Z scores are normal (respectively +0.64SD and +1.7SD vs +5SD and +8SD at admission), LVEF is normal (58% vs 28%), and mitral valve insufficiency is assent (severe at admission). Patient is now in Ross class I, and she has been delisted for heart transplantation.

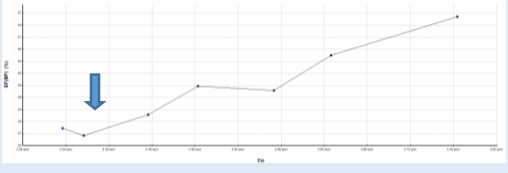
LV end-diastolic diameter /BSA (SD)



LV end-systolic diameter /BSA (SD)



LVEF (BP) %



Conclusion: *Ivabradine use in the setting of acute HF is well tolerated, effective in HR reduction, and able to increase cardiac output, permitting to achieve a clinical and ventricular recovery.*