**Abstract: P1180**

**Magnesium orotate for the treatment of heart failure in pregnant patients with unoperated atrial or ventricular septal defect**

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Background: Women with not closed atrial or ventricular septal defect (ASD/VSD) have small increased risk of maternal mortality or moderate increase in morbidity. Nevertheless, maternal cardiac complications occur in 12% of completed pregnancies. Offspring complications, including offspring mortality (4%), are more frequent than in the general population. Magnesium orotate (MO) is a non-steroidal anabolic orotic acid plus Mg\(^{2+}\) approved for pregnant patients.

Purpose: To evaluate the safety and efficacy of MO in pregnant women with unoperated ASD/VSD.

Methods: We studied 64 consecutive women with unoperated ASD (n=42) or VSD (n=22), aged 26±6 years. Patients were randomized to control group with conventional follow-up (n=32) and MO-group (n=32); in addition to standard therapy, they received MO from 2nd trimester 1000 mg t.i.d. for 1 week, followed by 500 mg t.i.d. The primary endpoints were a major adverse cardiovascular event (MACE), which included death, heart failure (HF), thrombo-embolic event, pulmonary arterial hypertension (PAH), and arrhythmias and pregnancy outcomes. Baseline and outcome data were analysed and compared for control patients vs. MO-group.

Results: At baseline, there were no significant differences between control and MO-group. NYHA functional class I had 21 (65.6%) and 20 (62.5%) patients, NYHA II had 4 (12.5%) and 5 (15.6%) patients, respectively (p>0.05). Atrial and/or ventricular ectopic beats had 28 (87.5%) and 29 (90.6%) patients (p>0.05).

No maternal mortality and no thrombo-embolic event occurred in both groups. In 17 control patients, at least one MACE occurred (53.1%): 7 developed HF (21.9%), 10 worsened HF (31.3%), 2 had atrial flutter (6.3%), 1 had a ventricular tachyarrhythmia (3.1%), and 1 patient developed PAH (3.1%).

In MO-group, no patient developed a MACE (p=0.005). MO reduced the HF occurrence (p=0.028) and HF worsening during pregnancy (p=0.015). Improvements were noted in control-adjusted changes in HF signs (−46.9%; p=0.005), cardiac hospital admissions (−40.6%; p=0.009) and in frequency of ectopic beats (−37.5%; p=0.022).

Perinatal mortality rate was 0 in the cohort, premature birth occurred in 8 controls (25%) followed by being small for gestational age (21.9%) vs. 0 in MO-group (p=0.028).

MO had no maternal and offspring adverse effects.

Conclusions: Long-term MO-therapy for pregnant patients with unoperated ASD/VSD prevents MACEs, improves HF status, and contributes to successful obstetric and foetal outcome. This study provides the evidence that metabolically acting MO is a promising therapy for pregnant patients with congenital heart disease.