PO-0501 GLIBENCLAMIDE CLOSES NEONATAL DUCTUS ARTERIOSUS IN RATS

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Background Treatment for patent ductus arteriosus (PDA) in premature infants may fail following perinatal indomethacin exposure. Glibenclamide, a sulfonylurea, constricts the fetal rabbit ductus in vitro, and fetal rat ductus in vivo. Clinical doses for diabetes include 0.1 mg/kg (adults) and 1 mg/kg (children with neonatal diabetes).

Aims To show ductus constriction acceleration with Glibenclamide in newborn rats as a model of treatment for premature PDA.

Methods Glibenclamide (1 mg/Kg) was injected intraperitoneally (IP) to newborn Wistar rats immediately after cesarian section, and the ductus diameter was studied at 60 min with rapid whole-body freezing, by cutting on a freezing microtome, and measurements performed on a microscope using a micrometre. Two near-term rat models were studied on the 21st day. In a chronic fetal indomethacin-exposure model, mother rats were treated with indomethacin (10 mg/kg, gavage) for two days before birth. In a hypoxia model, neonates were incubated in 8% oxygen. In a premature model, rats were delivered on the 19th day (two days before term) and incubated in 80% oxygen.

Results In the three models, neonatal ductus constricted slowly. Glibenclamide 1 mg/kg, caused accelerated constriction and the effects were dose-dependent. Glibenclamide (1mg/kg IP) was associated with hypoglycemia, which was controlled with 50% glucose via gavage.

Conclusions Glibenclamide (1 mg/kg IP) constricts the neonatal ductus in 60 min in three rat models. Hypoglycemia was controlled with glucose, indicating its usefulness in the treatment of PDA in premature infants.

PO-0502 CONGENITAL HEART DISEASE AT MATERNIDADE JÚLIO DINIS 2012–2013

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Background/Aim Congenital heart disease (CHD) is the most common congenital disorder in newborns (prevalence ranges from 6 to 13 per 1000 live births). Transversal study of the newborns diagnosed with CHD at Maternidade Júlio Dinis between 2012–2013.

Methods Patients were identified by searching the hospital’s electronic discharge records of the ICD-9 for each CHD (745.0–747.11). The following data were analysed: gender; gestational age; birth weight; pregnancy and delivery type; need of resuscitation; family history and maternal conditions that increase the risk for CHD; echocardiography reason and source of referral; clinical manifestations; paediatric cardiology agreement on the diagnosis; treatment and follow-up.

Results A total of 161 patients were documented, corresponding 24.8% to preterm newborns. The prevalence of echocardiographic findings was 26.8 per 1000 live births. At birth, resuscitation was needed in 20.5% patients. Cardiovascular findings suggestive of CHD were the reason to request echocardiogram in 75.8% cases and prenatal suspicion was responsible for 19.9%. Ventricular septal defect was the most prevalent (53.4%) CHD. Complex heart defects were found in 6.8% patients. A total of 101 patients were referred to paediatric cardiology and the concordance in diagnosis was around 99%. Surgical repair was performed in 5.6% patients. During this 2 years period, mortality related to CHD was 0.67 per 1000 infants (< 1 year age).

Conclusion This Portuguese CHD study shows a high prevalence of these disorders. Congenital heart defects are common conditions that have significant impact on morbidity, mortality and healthcare costs. A multidisciplinary team able to detect most of them in the neonatal period is crucial to minimise it.

PO-0503 TEN YEAR STUDY OF PREVALENCE AND DIAGNOSIS OF CONGENITAL HEART DISEASE (CHD) IN AN ASIAN COUNTRY: IMPLICATIONS ON ANTENATAL DIAGNOSIS AND NEWBORN SCREENING

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Rationale Congenital heart disease (CHD) constitutes a significant proportion of birth defects and is a leading cause of mortality. Current literature regarding the local prevalence of CHD and timeliness of diagnosis is not available.

Objective This study aims to determine the live birth prevalence of CHDs in Singapore General Hospital (SGH), the antenatal diagnosis rate, and the proportion of CHD patients diagnosed after discharge from the SGH neonatal unit.

Methods This was a retrospective observational study. All livebirths at SGH from January 2003 to December 2012 diagnosed with CHDs according to the hospital’s birth defect register were