

Cardiac arrhythmias affected five antenatally-diagnosed fetuses (56%), with one requiring emergency delivery at 28 weeks and ongoing neonatal management.

The majority of cardiac rhabdomyomas in both groups were located in the ventricles. Tumour growth continued up to 28 weeks of age amongst all surviving children, followed by spontaneous regression, with no need for resective surgery. There was a high prevalence of neurological morbidity in both groups.

Conclusion Antenatal cardiac rhabdomyomas, occurring as part of the TSC, can cause significant morbidity, which is rarely fatal, but warrants careful monitoring until the point of tumour regression. The burden of neurological disease is high in children, compared with the largely favourable cardiac outcome.

PF.57 FETAL MACROSOMIA: A RETROSPECTIVE OBSERVATIONAL STUDY

doi:10.1136/archdischild-2013-303966.066

G Argentesi, KF Walker, S Raouf. *Royal Derby Hospital, Derby, UK*

Objectives Maternal obesity is one of the biggest challenges facing modern obstetrics. The focus of this study was to investigate whether speculation that fetal macrosomia may be on the rise as a consequence of rising levels of maternal obesity and to observe if there was an increase in complications as a result of fetal macrosomia, which is defined as a birth weight of 4.0 kg and above.

Method A retrospective observational study of all babies weighing 4.0 kg or more born in 2011 at Royal Derby Hospital. Data was collected on maternal parameters such as BMI, fasting glucose and glucose tolerance test, gestation at delivery, delivery outcomes, neonatal birth weight, Apgar scores and their overall outcome. The data was then compared to data from both 2001 and 1991 recovered from the hospital archives.

Results In 2011, 11.1% of the total babies born that year had a birthweight of ≥ 4.0 kg. In 2001, 10.3% and in 1991, 10.7%. The average BMI of women who gave birth to a baby weighing ≥ 4.0 kg in 2011 was 28.

Conclusion Although there is speculation that fetal macrosomia is on the rise, in association with gestational diabetes and a rise in maternal BMI, we found that over the last 20 years the number of macrosomic babies has not increased at the Royal Derby Hospital. The overall maternal BMI was only slightly higher than average and deliveries involving macrosomic babies were not complicated by a higher rate of caesareans sections or instrumental deliveries or obstetric complications.

PF.58 MANAGEMENT AND OUTCOME OF VASA PRAEVI: A TEN YEAR REVIEW

doi:10.1136/archdischild-2013-303966.067

T Kubba, F Ushakov, S Cicero, G Attilakos. *Department of Fetal Medicine, UCL and UCLH Institute for Women's Health, London, UK*

Introduction Vasa Praevia (VP) describes fetal vessels coursing through the membranes over the internal os, unprotected by placental tissue or umbilical cord. VP is associated with significant fetal risk when membrane rupture occurs. The RCOG guideline on VP recommends antenatal admission from 28–32 weeks until delivery in a unit with appropriate neonatal facilities to facilitate quicker intervention in the event of bleeding or labour.

Aim To review the management and outcome of VP cases at a tertiary teaching hospital.

Methods We undertook a ten year retrospective review (2002 to 2012) of all cases of confirmed VP. Cases were identified using the discharge codes of all inpatient episodes and the fetal medicine unit database. We reviewed the ultrasound scans and notes of all cases.

Results We identified 15 confirmed cases of VP. 14 cases were diagnosed antenatally. The median GA at diagnosis was 25+3 weeks. 9 cases were admitted antenatally (duration: 2 days to 5 weeks). None of the admitted cases went into labour.

11/15 cases had elective LSCS and 4/15 had emergency LSCS (2/4 had category 1 LSCS). The median GA at delivery was 37 + 3 weeks. The single undiagnosed case resulted in neonatal death secondary to VP.

Conclusions

1. VP is a rare condition.
2. A high proportion of cases were diagnosed antenatally, however there may be cases which were never diagnosed and did not cause adverse events.
3. Further evidence is needed on the necessity and timing of antenatal admission.

PF.59 WHAT IS THE OPTIMAL DOSE OF LOW MOLECULAR WEIGHT HEPARIN IN PREGNANT WOMEN WITH RAISED BMI?

doi:10.1136/archdischild-2013-303966.068

¹S Rahman, ²D Stewart, ¹L Byrd, ¹C Tower. ¹St. Mary's hospital, Manchester, UK; ²Newcastle university medical school, Newcastle, UK

Introduction Obesity is a risk factor for thromboembolism in pregnancy. Recent RCOG guidelines suggested a non-evidence based weight-dependent protocol in which monitoring of anti-Xa levels was not required. This contrasted with local guidelines which used BMI based dosing, using anti-Xa levels to determine correct dose with BMI > 35. We sought to investigate the impact of the different strategies in a cohort of women treated with antenatal thromboprophylaxis.

Methods We retrospectively audited the thromboprophylaxis practise amongst 42 women between September 2009 and September 2011. We observed tinzaparin dosing, frequency of anti Xa levels, dose changes, and pregnancy outcomes.

Results 39/42 (93%) had a BMI over 35 and had anti-Xa measurements. Using the local protocol 15/39 (38%) required dose increases and all patients received a higher dose than suggested by RCOG guidelines (median 3000 IU, interquartile range (IQR) 3000–5000 IU). There were no thrombotic events and 25/38 (66%) achieved a vaginal delivery. The median estimated blood loss at delivery was 350 ml (IQR 200–725 mls) and 3 women suffered a major PPH > 1500 mls.

Discussion All our patients received a higher tinzaparin dose than suggested by RCOG guidelines, but shown by anti-Xa monitoring to be therapeutic. In these small numbers, there was a high vaginal delivery rate (66%) and although 3 women suffered a major PPH, the median blood loss was within an acceptable range for this high risk population. Without appropriate monitoring, RCOG guidelines may be resulting in suboptimal anticoagulation in women with raised BMI, whilst exposing them to the risks and side effects of LMWH.

PF.60 NEONATAL HYPOPHOSPHATASIA: A RARE DISORDER AND NEW TREATMENT

doi:10.1136/archdischild-2013-303966.069

¹S Hiremath, ¹VK Devendra Kumar, ²R Padidela, ²Z Mughal. ¹Lancashire Women & Newborn Centre, Burnley General Hospital, Burnley, UK; ²Central Manchester University Hospitals NHS Foundation Trust, Manchester, UK

Hypophosphatasia is a rare inborn error of metabolism resulting from mutations in the gene for the tissue-nonspecific isozyme of alkaline phosphatase (TNSALP). There is deficiency of alkaline phosphatase activity leading to severe rickets/osteomalacia. Severely affected babies die from respiratory insufficiency. There is no licenced medical treatment available. We report a case diagnosed