Women with fetal abnormality or obstetric indication for immediate delivery were excluded.

Interventions Participants were randomly allocated to either serial weekly trans-abdominal amnioinfusions when the deepest pool of amniotic fluid was less <2 cms or expectant management.

Results 58 pregnancies recruited: 28 in the amnioinfusion group (AI); 28 in the expectant management group (exp); two post-randomisation exclusions. Overall perinatal survival in both groups was 17/56.

Mean gestational age for AI group was 28.4 weeks vs. 29.8 weeks for exp (mean SD-1.4, 95% CI –0.2–1.5). One case of severe maternal sepsis requiring admission to HDU in the expectant management arm.

Overall chance of surviving without long-term respiratory or neurodevelopmental disability is 7.1%; 4/28 (14%) in the AI group and 0/28 in the exp group (RR 9.0; 95% CI 0.51, 159.70).

Conclusions The pilot findings do not suggest that clinicians should alter the current practise of expectantly managing rupture of membranes between 16 + 0 and 24 + 0 weeks of pregnancy. A larger definitive study to evaluate whether amnioinfusion has a cost-effective and acceptable role in improving healthy survival in very early rupture of membranes indicated.

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4.2 VARIATION IN BETA DEFENSIN 1 GENOTYPE IS ASSOCIATED WITH PRETERM BIRTH

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Introduction Human beta defensin 1 (HBD1) is an antimicrobial and immunomodulatory peptide present in cervical mucus. Variation in cervical antimicrobial expression is associated with preterm labour. We hypothesised that SNPs in the HBD1 gene may be associated with preterm birth.

Methods This is a retrospective case control study using blood collected at 11–13 weeks from women attending King's College Hospital March 2006-September 2010. 50 women with PPROM and 50 with spontaneous preterm labour were matched with 300 who delivered >37/40. SNPs rs1799946 (5'UTR) and rs1047031 (3'UTR) were genotyped by KASP assay (Kompetitive Allele Specific PCR, KBioscience). Data were analysed using multiplicative (rs1047031) and recessive (rs1799946) models and Chi Square Test.

Results There was no difference in BMI, smoking status or ethnicity between groups. Genotyping was successful in 98% (n = 390) and 97% (n = 386) samples for rs1047031 and rs1799946 respectively. Allele distribution demonstrated Hardy-Weinberg equilibrium. AA-homozygotes (rs1799946) had increased risk of PPROM; OR 2.24 (95%CI 1.11–4.49), p = 0.0.0257. 117 women had at least one prior delivery <37/40, 36 had a history of delivery <28/40. AA-homozygotes (rs1799946) had increased risk of delivery <37/40; OR 2.14 (95%CI 1.24–3.68), p = 0.005 and <28/40; OR 4.08 (95%CI 1.93–8.63), p < 0.0001. A allele carriers (rs1047031) were less likely to deliver <28/40; OR 0.288 (95%CI 0.121–0.683), p = 0.003.

Conclusion rs1799946 is associated with PPROM, a doubled risk of delivery <37/40 and a four-fold increase in the risk of delivery <28/40. rs1047031 may be protective. Variation in immune genotype may contribute to the clinical phenotype of women who deliver preterm.

4.3 THE RELATIONSHIP BETWEEN CAUSE AND TIMING OF PREVIOUS STILLBIRTH AND THE RISK OF STILLBIRTH IN SECOND PREGNANCIES

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Background Women with a previous stillbirth are at increased risk of stillbirth in their second pregnancy. However, there is little information on the relationship between the cause and timing of stillbirth in the first pregnancy and the risk in the second.

Methods and Results We identified 244,122 records in nationally collected Scottish data with complete information on their first and second births. The risk of stillbirth in the second pregnancy was 2.7 per 1,000 among 242,800 women with previous live birth and 15.9 per 1,000 among 1,323 women with previous stillbirth (odds ratio [OR] = 5.95 [95% CI 3.84–9.22] p < 0.001). Adjustment for maternal characteristics had no material effect. The risk was similarly elevated for different causes of stillbirth in the first pregnancy. It was also similarly elevated whether the previous stillbirth was extreme preterm (24–32 weeks) or late preterm/term (33–43 weeks). However, the association in the second pregnancy significantly varied across the range 24–43 weeks (test of proportional hazards assumption p = 0.01). Previous stillbirth was strongly associated with the risk at 24–28 weeks (15.66 [8.44–29.05], P < 0.001), at 29–32 weeks (5.16 [1.64–6.27], P = 0.005) and 33–36 weeks (6.03 [2.47–14.72], P < 0.001) but there was no significant association at term (1.7 [0.42–6.82]), probably due to routine elective delivery at 37–38 weeks.

Conclusion Previous stillbirth is a strong risk factor for stillbirth in second pregnancies irrespective of the cause of the first stillbirth. The recurrence risk is much higher at extreme preterm gestational ages, but is still present at 33–36 weeks.

Fetal Medicine Posters

PF.01 PRENATAL CHROMOSOMAL MICROARRAY USE: A PROSPECTIVE COHORT OF FETUSES AND A SYSTEMATIC REVIEW AND META-ANALYSIS

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Background Chromosomal microarray testing (CMA) is utilised in prenatal diagnosis to detect chromosomal abnormalities not visible by full, conventional karyotyping. We present our prospective cohort of women undergoing fetal microarray and karyotyping for an abnormal prenatal ultrasound scan (USS). This cohort is presented in the context of a systematic review and meta-analysis of the literature (until December 2012) which defines overall detection rates by microarray over karyotyping.

Systematic review methods: MEDLINE (1970–June 2012), EMBASE (1980–June 2012), Cinhal (1982–June 2012) were searched electronically. Selected studies had >5 cases and microarray testing was performed prenatally in addition to karyotyping. The search yielded 559 citations. Full manuscripts were retrieved for 85 and 24 primary studies were included in the systematic review.

Cohort Methods A prospective cohort study of 243 women undergoing microarray testing alongside karyotyping when a structural abnormality was detected on prenatal USS.

Results When clinical indication for testing was abnormal fetal USS our cohort study noted a 4.1% increase in detection rate; lower than the rate of 10.1% (95% CI 8.0–12.7%) by meta-analysis. When any clinical indication for prenatal microarray was meta-analysed the detection rate over karyotyping was 5.6% (95%CI 3.0–10.6%) and the variant of unknown significance (VOUS) rate was 1.4% (95%CI 0.5–3.7%).