Conclusion Chromosomal microarray is useful prenatally particularly for an abnormal fetal USS. Prospective counselling should include the approximate VOUSS rate (1.4% rising to 2.1% for abnormal USS). It is likely that microarray testing will replace karyotyping in high risk pregnancies (such as abnormal USS).

BACKGROUND

Background Multiple pregnancies are associated with a higher risk of spontaneous preterm birth (sPTB). Whilst fetal fibronectin (fFN) and cervical length (CL) measurement can predict sPTB in singleton pregnancies (Kurtzman et al, 2009), their value for twin pregnancies is unknown.

Methods Prospective blinded secondary analysis of longitudinal samples of cervicovaginal fluid fFN concentration (nanograms per milliliter) using a bedside 10 qfFN analyzer (HOLOGIC, USA), and transvaginal ultrasound CL of 98 consecutive women with multiple pregnancies attending a Preterm Surveillance Clinic at St. Thomas’ Hospital from 18 weeks gestation (Oct 2010-Jan 2012). qfFN was assigned 4 ranges; <10, 10–50, 50–200, >200 ng/ml to detect spontaneous delivery before 30, 34 and 37 weeks. qfFN was blinded to clinicians using an embedded code in the analyzer.

RESULTS The rate of sPTB (<37 weeks) rose with increasing qfFN from 17.5% (<10 ng/ml) to 61.5% (>200 ng/ml) and the negative prediction value for sPTB <30 weeks at <10 ng/ml was 98%. 4/15 (30%) of women with qfFN > 200 ng/ml delivered <30 weeks gestation. Using combined CL/qfFN testing, the positive prediction value of a qfFN value >200 ng/ml and CL < 25 mm was 87.5% for SPTB <37 weeks.

Conclusion This is the first report of 10 qfFN in twins, demonstrating that it adds predictive value to the qualitative results (negative cut-off at 50 ng/ml). High levels, even in early pregnancy, are associated with preterm delivery. Using cervical length and qfFN, management can be targeted to this group; e.g. antenatal maternal steroids.

In pregnancies complicated by IUGR there is a significant decrease in platelet function compared to healthy pregnant controls. Several agonists were assessed at incremental concentrations to characterise the response to multiple receptors. The findings were compared to healthy controls matched for gestational age with normal fetal weight. A total of 24 pregnancies complicated with IUGR and 36 healthy controls were recruited. Platelet reactivity in response to the agonists Arachidonic acid, Adenosine-diphosphate, Epinephrine and Thrombin-receptor activating protein was significantly reduced in the IUGR cohort. There was a nonsignificant trend to decreased reactivity in response to collagen (Table 1).

In pregnancies complicated by IUGR there is a significant decrease in platelet function compared to healthy pregnant controls. This may reveal valuable insights into the pathophysiology of the disease, and may represent an inadequate growth factor response in IUGR. Further evaluation of the role of platelets may aid in the development of future interventions for IUGR.