## 2.1 INCIDENCE, CAUSES AND OUTCOMES OF SEVERE MATERNAL SEPSIS MORBIDITY IN THE UK

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**Background** The incidence of severe genital-tract sepsis has increased in the UK and is now the leading cause of direct maternal death. Underlying this trend is a larger number of severe morbidity cases. The aim of this study was to describe, on a national level, the incidence, causes and outcomes of severe maternal sepsis morbidity in the UK.

**Methods** A national population-based study was undertaken using the UK Obstetric Surveillance System (UKOSS) between June 2011 and May 2012.

**Results** 378 women with severe sepsis were identified; an estimated incidence of 5.0 per 10,000 maternities (95%CI 4.6–5.7). Septic shock was diagnosed in 17.5% (N = 66) of women. Sources of infection were: intrauterine (N = 109; 39.9%), urinary-tract (N = 72; 26.4%), wound (N = 35; 12.8%), and respiratory-tract infection (N = 20; 7.3%). Laboratory-confirmed causative organisms were *E. coli* (31.3%), group A streptococcus (13.9%), group B streptococcus (13.4%), *Staphylococcus aureus* (10.4%) and polymicrobial growth (9.6%). Causative organisms differed significantly according to diagnosis of septic shock and mode of delivery (P = 0.002; P < 0.001); group A streptococcus was predominant amongst women with septic shock (30.8%) and spontaneous vaginal deliveries (33.3%), while *E. coli* was predominant amongst women without septic shock (32.6%), operative vaginal deliveries (36.0%) and caesarean sections (37.1%). Of all severely septic women, 73.0% (N = 276) required critical care and five women died.

**Conclusions** For every death from maternal sepsis, there are more than 75 women with severe sepsis morbidity. The pattern of infective organisms appears different amongst women who suffer septic shock. Further work is needed to investigate the risk factors associated with sepsis.

## 2.2 PLASMA PLACENTAL GROWTH FACTOR (PLGF) IN THE DIAGNOSIS OF WOMEN WITH PRE-ECLAMPSIA REQUIRING DELIVERY WITHIN 14 DAYS: THE PELICAN STUDY

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**Introduction** Evidence exists to suggest that the symptoms of pre-eclampsia are mediated by an imbalance of circulating angiogenic factors of placental origin; reduced concentrations of placental growth factor (PlGF) have been correlated with disease severity.

**Methods** A prospective, observational, cohort study was undertaken in seven UK maternity units. Women presenting 20 + 0 to 40 + 6 weeks gestation with suspected pre-eclampsia had serum PlGF measurement. ISSHP definitions of hypertensive disease were assigned, blinded to PlGF values. Analysis of the enrolment sample was conducted to evaluate diagnostic accuracy for pre-eclampsia requiring delivery within 14 days for very low PlGF (<12 pg/ml) and low PlGF (>12 pg/ml < 5$^{th}$ centile) using PlGF high (>5$^{th}$ centile) as referent.

**Results** Diagnosis of pre-eclampsia requiring delivery within 14 days using 5$^{th}$ centile as threshold.

**Conclusion** In women presenting <35 weeks' gestation with suspected pre-eclampsia, low PlGF level rules in women requiring delivery within 14 days and high PlGF rules out preterm delivery. Test performance falls off in women presenting over 35 weeks' gestational. PlGF can assist diagnosis and identify women requiring increased care.

**Abstract 2.2 Table**

| | < 35+0<br>N = 287 | 35+0 to 36+6<br>N = 137 | ≥37+0<br>N = 201 |
|---|---|---|---|
| Sensitivity | 0.95 (0.89–0.99)<br>79/83 | 0.71 (0.59–0.82)<br>47/66 | 0.59 (0.48–0.70)<br>49/83 |
| Specificity | 0.56 (0.49–0.63)<br>114/204 | 0.65 (0.53–0.76)<br>46/71 | 0.77 (0.69–0.84)<br>91/118 |
| Positive Predictive Value | 0.47 (0.39–0.55)<br>79/169 | 0.65 (0.53–0.76)<br>47/72 | 0.65 (0.53–0.75)<br>49/76 |
| Negative Predictive Value | 0.97 (0.92–0.99)<br>114/118 | 0.71 (0.58–0.81)<br>46/65 | 0.73 (0.64–0.80)<br>91/125 |
| Positive Likelihood ratio | 2.2 (1.8–2.5) | 2.0 (1.4–2.9) | 2.6 (1.8–3.8) |
| Negative Likelihood ratio | 0.09 (0.03–0.23) | 0.4 (0.3–0.7) | 0.5 (0.4–0.7) |

## 2.3 CIRCULATING MICROPARTICLES AND ARTERIAL STIFFNESS INDEX – A MEASURE OF VASCULAR ENDOTHELIAL DYSFUNCTION IN PREGNANCY AND SYSTEMIC LUPUS ERYTHEMATOSUS

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**Introduction** Microparticles (MPs) are circulatory vesicles with pro-thrombotic and inflammatory characteristics. Systemic lupus erythematosus (SLE) is an autoimmune condition with increased pregnancy morbidity. We investigated the role of MPs in pregnancy and SLE by longitudinal assessment, and correlated these with arterial stiffness index (SI), as a marker of vascular dysfunction.

**Methods** Plasma was obtained from pregnant (n = 20) and non-pregnant healthy women (n = 19), and pregnant (n = 15) and non-pregnant SLE patients (n = 30). MPs were quantified by flow cytometry. Arterial SI was measured by digital Pulse Contour Analysis (dPCA) (Micro Medical Ltd).

**Results** Platelet and endothelial MPs were significantly higher in non-pregnant women with SLE compared to healthy women ($p < 0.05$). In healthy pregnancy, platelet MPs declined at 12 wks gestation from non-pregnant controls ($p \leq 0.01$), but thereafter returned. This pattern was not observed in SLE pregnancy, which remained higher throughout gestation. Outside pregnancy, a positive correlation was recorded in SLE patients between SI and platelet, endothelial and total MPs ($p \leq 0.05$, $p \leq 0.05$ and $p = 0.05$; respectively). This relationship was lost in the SLE pregnant group. Placental MPs were unchanged in SLE and healthy pregnancies.

**Conclusions** SLE is associated with increased circulating MPs. These MPs may contribute or reflect vascular dysfunction as determined by arterial SI. A loss of this association implies alternative vascular and MP regulation in pregnancy. A lack of decline in platelet MPs in early pregnancy may predispose SLE patients to pregnancy complications, but the mechanism remains unclear.

## 3.1 PREDICTION OF FETAL COMPROMISE: THE USE OF FETAL DOPPLER ASSESSMENT IN NORMAL PREGNANCIES PRIOR TO LABOUR

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**Introduction** Up to 63% of cases of intra-partum hypoxia occur in pregnancies with no antenatal risk factors. Identification before labour of these antenatally normal fetuses at risk of intra-partum hypoxia would enable a more targeted approach to intra-partum care.