OBSTETRIC CASE REPORT

Breastfeeding and placental abruption

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Case report
A 32-year-old woman in her fifth pregnancy presented at 28 weeks’ gestation with a significant, painless, antepartum haemorrhage (APH). She reported intermittent uterine tightenings and backache over the previous week. Routine ultrasound at 20 weeks’ gestation had shown the placenta to be clear of the cervical os and she had no antenatal problems of note. She was a non-smoker and had been consistently normotensive throughout pregnancy. Her first pregnancy had ended in an emergency caesarean section for pre-eclampsia. This was followed by a normal vaginal delivery at term, a first trimester miscarriage and a tubal ectopic pregnancy. She had been breastfeeding her 2-year-old child during the pregnancy. On admission, she was normotensive and described good fetal movements. Her abdomen was soft and non-tender and a cardiotocograph (CTG) was normal.

Vaginal bleeding settled within 2 hours of admission and the patient remained in hospital for 3 days for observation and to complete a course of antenatal steroids to promote fetal lung maturity. Just before leaving the hospital she was directed to the hospital ultrasound department for an ultrasound assessment of fetal biometry to act as a baseline from which to assess subsequent growth. Whilst waiting for her scan, her sister arrived to transport her home and brought in the patient’s 2-year-old child whom the patient promptly breastfed. Within 5 minutes, she developed back and lower abdominal pain and removed the child from the breast. Ultrasound, performed some 5 minutes later, showed normal growth for gestation, normal liquor volume but evidence of a large placental haemorrhage (Figure 1). During the ultrasound, the sonographer noted a period of fetal bradycardia of 90 beats per minute. The patient was immediately returned to the antenatal ward. CTG initially revealed a baseline of 140 with absent variability but soon a bradycardia developed. An emergency lower segment caesarean section was performed and a female child weighing 1280 g was delivered in extremely poor condition with no heart beat (cord arterial pH 6.66, venous pH 6.77). A huge retroplacental clot was removed and the patient required a 4 unit blood transfusion. The baby was resuscitated but developed Grade 3 hypoxic ischaemic encephalopathy. Over the next week she developed acute tubular necrosis, followed by multi-system failure and eventual death on day 14.

Discussion
To our knowledge this possible association between breastfeeding in pregnancy and placental abruption has not been reported before. Breastfeeding in pregnancy is generally acknowledged to be safe but many infants wean themselves at around 5 months’ gestation as the volume of milk produced decreases markedly and the taste changes. Breastfeeding, nipple and areolar stimulation cause a reflex release of oxytocin from the posterior pituitary which not only promotes milk ejection, but also causes uterine contractions. This is the basis for using nipple stimulation as a contraction stress test of fetal well-being (Figure 2). Plasma oxytocin concentrations increase markedly during nipple stimulation in the second and third trimesters of pregnancy, although the magnitude of such an increase is diminished in comparison with that obtained in non-pregnant women or postpartum breastfeeding women (Amico and Finley, 1986). This is likely to be due to an indirect inhibitory effect of high concentrations of oestrogen and progesterone during pregnancy. The response of the uterus to oxytocin in pregnant women who have been breastfeeding long term is less clear, but there is evidence

Figure 1. Retroplacental haemorrhage following breastfeeding.

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that such long-term nipple stimulation is associated with greater oxytocin release than those who commence breastfeeding in pregnancy (Leake and Nemes, 1984). Uterine hyperstimulation can occur following nipple stimulation during pregnancy and some authorities advise stimulation be restricted to one side only (Huddleston et al., 1984).

We could find no literature on the effects of breastfeeding during pregnancy on uterine activity. However, as breastfeeding produces greater oxytocin release and more uterine activity than nipple stimulation in postpartum studies (Chua et al., 1994) then it is reasonable to assume this would also be true during pregnancy. Only one case of abruption following nipple stimulation has been reported (Taylor and Green, 1987) in a chronic paranoid schizophrenic who was known to have an abnormal contraction stress test. She surreptitiously manually stimulated both her nipples whilst undergoing a non-stress test and alerted midwifery staff when sudden vaginal bleeding commenced. Eighty per cent of the placenta was separated from its site of implantation at caesarean section.

It is likely that breastfeeding and its effect on uterine activity was a factor in the initial abruption our patient suffered; it certainly appears to have precipitated the final and unfortunately fatal bleed. This case highlights an unusual but possibly important risk related to breastfeeding in pregnancy. We do not suggest that all pregnant women should deprive their infants of a valuable source of nutrition, however we do suggest that those with a previous pregnancy complication predisposing them to APH (e.g. hypertension or pre-eclampsia), those with a previous APH in the index pregnancy or those with fetal growth retardation, should be strongly encouraged to stop breastfeeding.

References