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Impaired renal function in the mother may result in a high false positive rate in Down Syndrome screening

L. M. Byrd & P. Baker

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presenting symptoms. About 11% of cases may be asymptomatic (Jensen 1992).

We present a case of asymptomatic uterine torsion and highlight how a change in placental location on ultrasound scan during pregnancy should alert the obstetrician to the possibility of this rare event.

Case report

A 38-year-old woman presented in her third pregnancy with dichorionic diamniotic twins following *in vitro* fertilisation (IVF). Both previous pregnancies had followed IVF with the first ending in miscarriage at 9 weeks and the second in a ventouse delivery at term. The antenatal period had been uneventful in the last pregnancy. In her present pregnancy she had an uncomplicated antenatal period and ultrasound scans at 12, 20, 28, 32 and 36 weeks showed normally growing fetuses and placentae to be high anterior. However, at 38 weeks, a repeat scan showed a change in placental position which was low posterior and lying across the internal os. Following this scan, she was admitted to the antenatal ward for elective caesarean section at 39 weeks. The day after her admission she spontaneously ruptured the membranes and an emergency caesarean section was performed under general anaesthesia. Intraoperative findings showed the uterus to be completely dextro-rotated through 180° with engorged veins of the broad ligament stretched over the lower segment. The left ovary and tube were stretched across the anterior surface of the uterus. The uterus was successfully de-rotated and a lower segment caesarean section was performed. Both the babies were delivered in good condition. The placentae were noted to be posterior extending to just above the internal os. The mother recovered uneventfully and both mother and babies were discharged on the fifth postoperative day.

Discussion

Aetiology of torsion during pregnancy can be varied. About 20% are idiopathic and torsion may be found as an isolated phenomenon. A total of 30% of torsions were associated with fibroids; 15% with uterine anomalies; 6.5% with ovarian cysts; 8.4% with pelvic adhesions and 2.8% with abnormalities of spine and pelvis (Nesbitt and Corner 1956). Apart from these, variation in size, anatomy, position and mobility of bladder and rectum may also cause torsion of gravid uterus. Other less common associated factors seen were abnormal uterine suspension and fetal position.

It is thought that certain activating factors result in torsion, since torsion is not frequently seen despite local associated pathology. Robinson and Duvall have suggested that irregular body movements, positions and postures may precipitate torsion. Irregular contractions of the abdominal muscles, fetal movements and even uterine contractions may facilitate torsion in the presence of predisposing factors (Nesbitt and Corner 1956).

An index of suspicion could lead to correct diagnosis. Antenatally, a recent change in placental location on ultrasound scan is an important finding, which could raise the suspicion of uterine torsion and lead to a correct diagnosis (Kremer and Van Dongen 1989).

Finding of uterine torsion is usually unexpected at laparotomy or caesarean section. Preoperative differential diagnosis includes ectopic pregnancy, degeneration of fibroids, torsion of a pelvic tumour, rudimentary horn pregnancy, abruptio placentae, obstructed labour, placenta previa, rupture uterus and abnormal fetal presentation.

Once the correct diagnosis is made, management is relatively straightforward. Delivery is either by posterior hysterotomy (Kim et al. 2001) followed by re-positioning of the uterus or by re-positioning the uterus first before proceeding with lower segment caesarean section. Correct assessment of the degree of torsion is important for planning the incision. Failure to do so may increase the risk of injury to the uterine vessel and possibly the ureter. The best method of assessing the degree of torsion is by using the round ligaments as 'landmarks'.

Uterine torsion is very difficult to diagnose clinically and is mainly an intraoperative finding. A suspicion of torsion should arise when there has been a recent change in placental location from anterior to posterior or right to left. Management should ideally be to reposition the uterus before proceeding with caesarean section, as this prevents the associated complications of a posterior hysterotomy.

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Correspondence: B. Dandawate, Flat 3, 91 Longfleet Road, Poole, Dorset BH15 2HP, UK. E-mail: beenasrajan@yahoo.co.uk

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Impaired renal function in the mother may result in a high false positive rate in Down Syndrome screening

L. M. BYRD & P. BAKER

Department of Obstetrics and Gynaecology, St Mary's Hospital, Manchester, UK

Case report

A 35-year-old woman booked at 7^{+6} weeks' gestation. She was diagnosed with chronic renal insufficiency secondary to chronic pyelonephritis 4 years ago and she developed hypertension 2 years later treated with Nefedipine LA 20 mg/daily.

On booking, her blood pressure was 134/92 mmHg, and a 24-h urine collection confirmed proteinuria (4.5 g). Serum creatinine

was 120 μmol/l. Labetalol (20 mg b.d.) was started. The options for Down's screening were discussed and she opted for serum screening; this gave a risk of 1 in 90. After discussion she decided not to have an amniocentesis.

An anomaly scan at 20 weeks' gestation showed no gross abnormality. The pregnancy proceeded without event. Serial growth scans indicated good fetal growth. Serum creatinine remained stable (range 115–127 μmol/l), proteinuria persisted.

Labour was induced at 38 weeks' gestation. An abnormal cardiotocograph (CTG) necessitated an emergency caesarean section. A male infant weighing 2,320 g was delivered in good condition. There were no features of Downs' syndrome, and subsequent karyotype confirmed a normal XY genotype.

Discussion

The 1 in 90 risk of Down's syndrome obtained from serum screening raises some important issues with regard to such screening in women with renal impairment.

Chao et al. (2002) reported a false positive rate of greater than the 5% usually quoted for Down's syndrome screening among 15 pregnant women undergoing haemodialysis. They speculated that this was due to an overproduction of human chorionic gonadotropin (hCG) by the placenta, in conjunction with impaired excretion of hCG during haemodialysis. It was also suggested that this false positive Down's serum screen was indicative of adverse pregnancy outcome (particularly intrauterine growth retardation, IUGR); the raised serum hCG was linked to hypoxia-induced trophoblastic tissue formation.

In a case control study of 24 patients (12 in each group), a similarly high false positive rate for serum screening was seen in women who had undergone renal transplantation, compared with controls. Direct correlation was observed between multiples of the mean (MoM) of beta hCG and serum concentrations of creatinine ($r = 0.5746$, $p < 0.01$) (Cararach et al. 1997). Serum hCG of 3.0 MoM was found in those with a mean serum creatinine level of 130.8 ± 32.7 μmol/l, while serum hCG of 1.12 MoM was found in those with a serum creatinine of 60.1 ± 8.8 μmol/l.

A third study recruited 14 patients who had undergone renal transplantation and were now pregnant. Direct correlation was again shown between serum free beta HCG and serum creatinine (Karidas et al. 2002), with an increased false positive rate.

In view of these findings all women with known renal disease, and/or hypertension, should have bloods taken for creatinine, at booking. The use of serum biochemical markers to screen for Down's syndrome, even in the presence of modest renal insufficiency (creatinine >110 μmol/l in this case) can be misleading. In order to avoid causing anxiety and unnecessary invasive diagnostic tests (amniocentesis/chorionic villus sampling) which carry a risk of miscarriage, such tests should perhaps be avoided in this group of women. Non-invasive screening methods such nuchal translucency, would be a preferable alternative.

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Correspondence: L. M. Byrd, 9 Orchard Green, Alderley Edge, Cheshire SK9 7DT, UK. E-mail: tahghighibyrd@aol.com

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Spontaneous rupture of caesarean scar at 16 weeks

S. M. MAHENDRAN, P. K. JOTHILAKSHMI, & M. R. STEEL

Department of Obstetrics and Gynaecology, Victoria Hospital, Blackpool, UK

Case report

A 40-year-old woman in her eighth pregnancy was admitted to hospital at 16 weeks' gestation, with lower abdominal pain and vaginal bleeding of 7 hours duration.

Previously she had a full term normal pregnancy and normal delivery, followed by a pre-term labour at 24 weeks and then a molar pregnancy. She then had two uncomplicated term pregnancies and vaginal deliveries, followed by an elective lower segment caesarean section for placenta praevia.

She underwent a myomectomy to remove a 10 cm fundal fibroid, which had caused menorrhagia, dysmenorrhoea and pressure symptoms on the bladder. During the myomectomy the uterine cavity was entered. She then became pregnant again and was delivered by elective lower segment caesarean section at 37 weeks.

In the index pregnancy a dating scan carried out at 12 weeks' gestation confirmed the expected date of delivery.

On admission to hospital her vital signs were within the normal range. Due to obesity (her body mass index was 37), abdominal examination was difficult. There was some tenderness in the lower abdomen. Vaginal examination revealed clots in the vagina. The cervix was uneffaced, central and 2 cm dilated. The initial blood test revealed a haemoglobin concentration of 12.3 g/dl. An ultrasound scan carried out on the ward demonstrated a non-viable fetus in the uterine cavity. A clinical diagnosis of inevitable miscarriage was made. Shortly after admission, the patient began to experience more abdominal pain with a sudden and heavy increase in vaginal bleeding including large clots. By this time, the estimated blood loss had reached 800 ml. The abdominal tenderness had increased and the patient became tachycardic and hypotensive. A repeat vaginal examination showed no change in the cervix. In view of the heavy vaginal bleeding, the patient was taken for an urgent hysterotomy.

The laparotomy was carried out through the previous suprapubic transverse scar. On opening the abdomen, a significant