Case report

A 35-year old P 0+3 woman presented to the Acute Gynaecology Unit (AGU) at Nepean Hospital at 5+3 weeks' gestation for an early pregnancy location scan as she had previously been diagnosed with a right interstitial pregnancy in 2007. Her past obstetric history included a missed miscarriage at 7 weeks' gestation in 2007, an interstitial pregnancy managed expectantly in 2007 and a recent empty sac miscarriage managed expectantly. Past gynaecological history included two laparoscopic surgeries for endometriosis. She had no other risk factors for ectopic pregnancy.

Transvaginal ultrasound (TVS) at 5+3 weeks gestation demonstrated a pregnancy of unknown location (PUL), i.e. no signs of an intra- or extra-uterine pregnancy. The endometrial thickness (ET) measured 2.6 mm; the right ovary was enlarged measuring 63 × 46 mm with a bilocular cyst, each locule measuring 28 × 36 mm and 33 × 42 mm, respectively; the left ovary was normal in size and appearance. There was no free fluid in the pouch of Douglas. Serum hCG at presentation and at 48 hours were 199 IU/l and 250 IU/l respectively, i.e. hCG ratio (hCG 48 hrs/hCG 0 hrs) was 1.25. This equated to a rise of serum hCG of 25% in 48 hrs.

She was rescanned the following week to ascertain the location of the pregnancy. Her serum hCG levels continued to rise sub-optimally measuring 328 IU/l. Repeat TVS demonstrated an empty uterus with an ET of 3.5 mm and a persistent right ovarian cyst that was now unilocular and haemorrhagic, measuring 22 × 23 × 22 mm. There was no obvious tubal ectopic pregnancy. The left ovary was normal in size, measuring 30 × 15 mm; however this contained a cystic structure with a double hyperechoic ring measuring 9.4 × 7.0 mm. This represented the gestational sac and within this was a yolk sac measuring 4.5 × 5.1 mm. The colour Doppler score was 1, i.e. no vascularity was demonstrated. There was no haemoperitoneum. A diagnosis of left ovarian ectopic pregnancy was confirmed. She was eligible for conservative management. In accordance with the unit's protocol, we arranged the pre-treatment hCG ratio. If the hCG ratio at 48 hours was < 1, i.e. the serum hCG levels were falling, then she was to be managed expectantly; if the hCG ratio at 48 hours was > 1, i.e. the serum hCG levels were increasing, then she was to be managed medically with methotrexate (MTX) 50 mg/m². Serum hCG measurements at 0 hours and 48 hours were 328 IU/l and 341 IU/l respectively, i.e. the hCG ratio was >1. She was therefore given 75 mg MTX intramuscularly. The serum hCG fall was 33% (292 IU/l to 196 IU/l) between days 4 and 7, i.e. > 15%. One weekly serum hCG levels were arranged thereafter until the hCG levels were < 5 IU/l.

Discussion

Ovarian pregnancy is an extremely rare type of ectopic pregnancy, occurring only in 1 in 25,000 to 40,000 pregnancies which accounts for 0.5-3% of all ectopic pregnancies [1]. The incidence of interstitial pregnancy is around the same. The diagnosis of ovarian ectopic pregnancy can be a difficult one, but with the help of high-resolution transvaginal ultrasound, specific ultrasonographic criteria do exist. These include an empty uterus, the presence of an ovarian cystic mass lesion within the ovary itself with or without internal echoes or a characteristic double hyperechoic ring surrounding a visible yolk sac.

In women with an ectopic pregnancy who are clinically stable, calculation of the pre-treatment hCG ratio were increasing, then she was to be managed medically with methotrexate (MTX) 50 mg/m². Serum hCG measurements at 0 hours and 48 hours were 328 IU/l and 341 IU/l respectively, i.e. the hCG ratio was >1. She was therefore given 75 mg MTX intramuscularly. The serum hCG fall was 33% (292 IU/l to 196 IU/l) between days 4 and 7, i.e. > 15%. One weekly serum hCG levels were arranged thereafter until the hCG levels were < 5 IU/l.

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(hCG 48 h/hCG 0 h), allows evaluation of the trophoblast activity or 'trophoblastic load' (2-4). An hCG ratio <1 which equates to falling serum hCG levels at 48 hours, suggests that the ectopic trophoblast is resolving spontaneously and it may be possible to avoid MTX administration in this sub-group. Women with an hCG ratio >1 which equates to increasing serum hCG levels at 48 hours, indicating still active trophoblasts should be targeted for MTX. This approach does not compromise the outcome of these women and it has the potential to reduce the blind administration of cytotoxic drug MTX at presentation in an ectopic pregnancy which would have resolved spontaneously anyway [5].

In conclusion, this case highlights the importance of the pre-treatment hCG ratio in decision making regarding the appropriateness of MTX for women with non-tubal ectopic pregnancy.

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Pseudohypoaldosteronism type 1 in an infant

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Ceylon Medical Journal 2011; 56: 71-72

Introduction

Pseudohypoaldosteronism (PHA) type 1 is a rare but serious abnormality characterised by mineralocorticoid resistance causing hyperkalaemia and hyponatraemia [1]. There are two clinically distinguishable entities of PHA type 1, an autosomal recessive severe variant with multiple target organ involvement and an autosomal dominant less severe variant with isolated renal involvement. Autosomal recessive PHA type 1 is due to defective epithelial amiloride sensitive sodium channels. We report an infant with autosomal recessive PHA type 1 associated with choledolithiasis and hypocalcaemia who was successfully treated with 6% saline, G solution and calcium polystyrene sulphonate resins.

Case report

A six-week old baby boy was referred from a local hospital where he had presented at 4 weeks of age with vomiting, poor feeding and lethargy of two weeks duration. He was the 2nd child of consanguineous parents. Maternal polyhydramnios had been noted antenatally. He had been collapsed on admission to the local hospital with severe dehydration. Examination had shown a fair complexioned child with a weight of 2.83 kg which was 450g lower than the birth weight. Examination of the systems was unremarkable with normal male external genitalia. His initial serum electrolytes were: Na+ 97 mmol/l, K+ 9.7 mmol/l and Cl- 84 mmol/l. Blood glucose was 7.3 mmol/l, pH 7.43 and bicarbonate 20.6 mmol/l. CRP was negative, blood cultures sterile and 2D echocardiogram was normal. A tentative diagnosis of salt-losing congenital adrenal hyperplasia had been made and treatment commenced with hydrocortisone and fludrocortisone. Response to therapy was poor after 2 weeks of treatment and as the 17 hydroxy progesterone done on the 28th day of life was normal at 9.8 ng/ml (29.4nmol/l) he was transferred for further management.

On admission to our ward at 45 days of age he was dehydrated with a weight of 2.85 kg and length and head circumference of 54 cm and 37 cm, which were on the 25th and 10th percentiles respectively. His blood pressure was 70/40 Hgmm. We optimised his treatment with hydrocortisone and fludrocortisone and further investigations were done. Polyuria was observed at 7-10 ml/kg/hour. His serum electrolytes on several occasions showed a persistent hyponatraemia ranging from 111 mmol/l to 121 mmol/l and hyperkalaemia ranging from 7.0

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