Table I. Outcomes of pregnancies with multiple myeloma reported in the literature

Age (years)	Ethnic origin	Gestation at the time of diagnosis	Presentation	Outcome	Reference
42	Puerto Rican	Not pregnant	Headaches and lumps on forehead	Live baby	Rosner et al. 1968
35	Afro-Caribbean	Not pregnant	Bone pain	Live baby	Kosova and Schwartz 1966
21	Afro-Caribbean	Not pregnant	Bone pain	Live baby	Lergier et al. 1974
33	Black	20/40	Anaemia and dizziness	Live baby	Caudle et al. 1990
32	Black	3rd postpartum day	Lower extremity weakness, leg weakness	Live baby	Malee 1990
41	Hispanic	Not pregnant	Anaemia	Live baby	Sakata et al. 1995
34	White	6/40	Threatened miscarriage	Live baby	Maglione et al. 2003
N/A	N/A	6/40	Threatened miscarriage	Live baby	
41	N/A	2nd trimester	Pathological fracture at 2nd trimester	Live baby	

start her on high dose melphalan and stem cell re-infusion in the near future.

Discussion

The median age of diagnosis of multiple myeloma is 65 years, with only 2–4% of cases aged under 40 and is thus extremely rare in women of child-bearing age (Malee 1990). It is twice as common in men as women, and more common in blacks than whites. Maternal cancer complicates only 0.02–0.1% of all pregnancies (Maglione et al. 2003). Only 12 cases have been reported so far in the literature. The most common presentation of multiple myeloma is bone pain in those that are diagnosed in pregnancy. Our patient presented with excessive vomiting and is the first case reported to be presenting in this fashion. Most of the reported cases, after counselling, decided to carry on with their pregnancies (Table I).

This report is the only reported case where termination of pregnancy was carried out as the mother felt she could not cope with being pregnant with the disease and would rather embark on treatment straight away despite the offer of support during pregnancy. She was counselled based on reports in the literature and their successful outcome. The longest reported fetal follow-up was 6 years, with no evidence of disease in the child (Rosner et al. 1968).

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Pre-eclampsia and hyponatraemia

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Case report

A 38-year-old primigravida, booked at 8 weeks' gestation with a blood pressure (BP) of 110/60 mmHg. She was a low-risk primigravida with no significant medical problems. She had a glucose tolerance test at 28 weeks because of a family history of diabetes and this was normal. A growth scan was performed at 30 weeks, as the fundal height was greater than expected for the gestational age and revealed an average size baby with normal liquor volume.

Pre-eclampsia was diagnosed at 34 weeks' gestation due to hypertension and significant proteinuria of 0.6 g/24 h. She was managed conservatively as her BP stabilised at 140–150/90–95 mmHg. Her haematological and biochemical parameters relevant to pre-eclampsia remained satisfactory. Polyhydramnios with an amniotic fluid index (AFI) of 26.0 was detected by an ultrasound scan at 35 weeks, when fetal growth was satisfactory. Blood tests for TORCH and Parvovirus were negative. However, the blood tests performed for follow-up of pre-eclampsia revealed hyponatraemia with a serum level of 128 mmol/l. Her previous serum sodium

value had been satisfactory (133 mmol/l). Serum sodium fell further to 123 mmol/l, 6 h later. Reassuringly, the rest of her serum biochemistry remained normal. (bicarbonate 20 mmol/l; potassium 4.3 mmol/l; urea 2.7 mmol/l; creatinine 66.0 μmol/l; urate 276 μmol/l; glucose 6.4 mmol/l; and liver function tests were normal). The patient's serum osmolality was reduced at 261 mmol/l and urine osmolality was raised to 279 mmol/l. She was asymptomatic with no pedal oedema. She was reviewed by the physicians and had further investigations to elucidate the nature of hyponatraemia. Her lipid and thyroid profiles were within normal limits. Urinary sodium excretion was increased at 65 mmol/l.

The patient was managed conservatively by fluid restriction and the sodium levels increased to 128 mmol/l. Induction was planned at 37 weeks for worsening pre-eclampsia. Her serum sodium was 130 mmol/l and the rest of her serum biochemistry had remained essentially unchanged. However, she had an emergency caesarean for non-reassuring cardiotocography following the first dose of Prostin. A female infant, weight 3.545 kg was delivered with good Apgar scores and normal cord gases.

She had an uneventful postnatal course and serum sodium levels returned to normal (137 mmol/l) within 48 h.

Discussion

The normal re-setting of the osmostat of pregnancy reduces the serum osmolality by 6–10 mmol/l (Hayslett et al. 1998). This also results in a fall in serum sodium levels and a serum sodium value of 130 mmol/l is acceptable in pregnancy. This case report shows hyponatraemia-complicating pre-eclampsia in a patient with increased urine osmolality and sodium excretion and with decreased plasma osmolality.

We believe that in this case there was hypo-osmolar, normovolaemic hyponatraemia (serum osmolality <275 mmol/l). A high urinary osmolality of 279 mmol/l (>200 mmol/l) reflected impairment in water excretion and this along with a high urinary sodium of more than 30 mmol/l, helped to differentiate this patient from those associated with hypovolaemia. There was no significant medical history of thyroid, cardiac, liver, adrenal or renal failure. Nephrotic syndrome was excluded as a result of the mild proteinuria (0.6 g/24 h).

A literature search revealed a few case reports of hyponatraemia associated with pre-eclampsia with or without nephrotic syndrome. To our knowledge, this is the ninth case report of hyponatraemia complicating pre-eclampsia. Dilutional hyponatraemia in women with pre-eclampsia has been described with or without nephrotic syndrome, with normal or nearly normal renal function (Hayslett et al. 1998; Magriples et al. 2001; Goodlin and Mostello 1987). Severe hyponatraemia in pre-eclampsia could also lead to neonatal hyponatraemia (Burrell and de Swiet 2004). A similar case was described by Ravid et al. (2005). The hyponatraemia in a patient with pre-eclampsia occurred in the absence of nephrotic syndrome and it resolved following delivery.

The mechanism of hyponatraemia in pre-eclampsia is not fully understood. Various mechanisms have been postulated and they include inappropriate secretion of antidiuretic hormone (ADH) and raised renal sensitivity to normal arginine vasopressin levels (Sutton et al. 1993; Hayslett et al. 1998; Knepper 1997).

Clinical features of hyponatraemia include headache, lethargy, muscle cramps, confusion, convulsion and coma. Their occurrence depends on the absolute sodium concentration and its rate of fall. Such convulsions may be difficult to differentiate from eclampsia. Death can occur in up to 50% of patients in whom the serum sodium concentration falls below 120 mmol/l within a period of 24 h (Narins 1986). In this case, maternal hyponatraemia improved with fluid restriction and resolved spontaneously within 48 h following delivery, with no residual effects.

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Cardiac arrest associated with uterine inversion during caesarean section

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Case report

A 30-year-old woman, para 1 (vaginal delivery) with a history of Behçet's disease went into spontaneous labour at term, 20 h after rupture of the membranes. At 3 cm dilation, the liquor was blood-stained and the cardiotocograph abnormal; a decision was made to perform lower segment caesarean section (LSCS). Her observations were normal and preoperative haemoglobin was 9.7 g/dl.